YANKASA SHEEP FED WHOLE OR CRUSHED MAIZE GRAIN AND HAY (DIGITARIA SMUTSII) AND RESULTANT LACTIC ACID PRODUCTION

BY

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A DISSERTATION SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES AHMADU BELLO UNIVERSITY ZARIA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER OF SCIENCE DEGREE (M.Sc.) IN FOOD ANIMAL MEDICINE

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JANUARY, 2020

DECLRATION

I declare that the work in this dissertation entitled 'YANKASA SHEEP FED WHOLE OR CRUSHED MAIZE GRAIN AND HAY **(**DIGITARIA SMUTSII) AND RESULTANT LACTIC ACID PRODUCTION' has been carried out by me in the Department of Veterinary Medicine under the supervision of Professor L. B. Tekdek and Professor S. O. Okaiyeto. The information derived from the literature has been duly acknowledged. No part of this dissertation was previously presented for another degree or diploma at this or any institution.

Hassan Kanti MADU _____ Signature _____ Date

CERTIFICATION

This dissertation '**YANKASA SHEEP FED WHOLE OR CRUSHED MAIZE GRAIN AND HAY (DIGITARIA SMUTSII) AND RESULTANT LACTIC ACID PRODUCTION'** by Hassan Kanti MADU meets the regulations governing the award of the degree of Master of Science of Ahnadu Bello University, Zaria and is approved for its contribution to knowledge and literary presentation.

Prof. L. B. Tekdek Chairman Supervisory Committee	(Signature)	Date
Prof. S. O. Okaiyeto Member Supervisory Committee	(Signature)	Date
Prof. P. A. Abdu Head of Department, Department of Veterinary Medicine Ahmadu Bello University, Zaria	(Signature)	Date
Prof. Sani A. Abdullahi Dean, School of Postgraduate Studies, Ahmadu Bello University, Zaria	(Signature)	Date

DEDICATION

This dissertation is dedicated to my parents, my wives and my children Mustapha, Maryam and Sa’adatu for their understanding and support.

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ABSTRACT

Rumen lactic acidosis is a metabolic disease condition that is caused by ingestion of excessive quantities of highly fermentable carbohydrate feed. This study was carried out to evaluate the responses of Yankasa sheep fed whole or crushed maize grain and *Digitaria smutsii* hay. The study was conducted using 15 sheep(6 male) and (9 female) distributed randomly into three experimental groups (A, B and C) of five sheep each. The sheep were acclimatized for three weeks. At week 1 the feeding trial Group A sheep were fed with 50% of 4% body weight of whole maize grain and 50% of 4% body weight *Digitaria smutsii* hay; Group B with 50% of 4% body weight of crushed maize grain and 50% of 4% body weight *Digitaria smutsii* hay and Group C(control) were fed with *Digitaria smutsii* hay only all for 24 hours. At week 2, Group A were fed with 75% of 4% body weight whole maize grainand 25% of 4% body weight *Digitaria smutsii* hay; Group B fed with 75% of 4% body weight crushed maize grainand 25% of 4% body weight *Digitaria smutsii* hay Group C(control) were fed with *Digitaria smutsii* hay only all for 24 hours. At week 3, Group A was fed with 100% of 4% body weight whole maize grainand 0% of 4% body weight *Digitaria smutsii* hay and group B fed with 100% of 4% body weight crushed maize grain and 0% of 4% body weight *Digitaria smutsii* hay all for 24 h. Group C(control) were fed with *Digitaria smutsii* hay only for 24 hours each week and all the groups were given water *ad libitum.* Vital parameters were taken 6 hourly and rumen fluid was collected every 2 h for 24 h through rumen catheter for 3 consecutive weeks and assessesfor pH, acetic acid, lactic acid and rumen bacterial count. Blood samples were collected 6 hourly through the jugular vein into EDTA container and evaluated for blood pH and haematological parameters (PCV,HGB, RBC, WBC). There was significant increase ($p<0.05$) in respiratory rate (21.0 ± 0.95 to 23.0±0.55) and pulse (79.8 ± 2.92 to 84.2±2.71) from 12 h in both group A and B throughoutthe three

weeks of the study.Rectal T^0C were obtained 6 hourly.There were slight decreased (38.74 ± 0.24 to 38.08 ±0.08 and 39.12 ± 0.20 to 38.12±0.09) in rectal temperature from 6 h after introducing the experimental feed in both groups A and B. Rumen pH were significantly decreased (6.72 ± 0.02 to 5.59±0.15) from 4 h after introducing the experimental feed in group B and (6.82 ± 0.03 to 5.96±0.07) at 6 h in group A respectively. Blood pH was significantly decreased from 6 h (7.34±0.02 to 7.04 ± 0.02) after introducing the experimental feed in both group A and B.The observed clinical signs were loss of appetite in some animals, depression, diarrhoea, bruxism (teeth grinding) salivation. Death was observed 6 days post commencement of feeding trial.Acetic acid was significantly increased ($p<0.05$) in both group A and B for the three weeks of the study. Lactic acid increased significantly ($p<0.05$) from 8 h (44.96 ± 2.61to 56.90±1.23) after introducing the experimental diet at both groups A and B and continued to increase through weeks 2 and three, then decreased from 10 h (40.48 ± 2.46 to 35.86±0.56) in group A animals and 8 h (42.34 ± 1.40 to 35.00±0.57) in group B animals and increased at 20 h (35.86±0.56 to 52.94±1.27) in groups A and 18 h (35.00±0.57 to 44.34±4.89) in groups B to 24 h respectively. There were an increase in the values of rumen microbial count (4.37 ± 0.05 to 4.58 ± 0.10); also there was increase in the values of packed cell volume (35.20 ± 3.15 to 39.69 ± 3.87)The haemoglobin, red blood cell and white blood cell obtained in this study were within the reference values of the species. Histopathology changes observed in this study were sloughing of the rumen mucosa. It was concluded that sub-acute ruminal acidosis was obtained. We recommend that high levels of maize grain either whole or crushed should not be given to sheep at 75% of 4% body weight as feed for whatever reason because it may expose the animals to acidosis and death.

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LIST OF ABREVIATIONS

%	Percentage
/	Minutes
@	At
<	Less than
>	Greater than
µl	Microlitre
^{0}	Degrees
^{0}C	Degrees Centigrade
A	Ash
A.O.A.C	Association of Official Analytical Chemists
AA	Acetic Acid
ABUECAUC	Ethical Committee on Animal Use and Care of Ahmadu Bello University, Zaria
ANOVA	Analysis of Variance
CF	Crude Fibre
cm	Centimetre
CO_2	Carbon dioxide
Conc.	Concentration
CP	Crude protein
DFM	Direct-fed microbials
dl	Decilitre

DM	Dry Matter
Dr.	Doctor
e.g.	Example
EDTA	Ethylene Diamine Tetra Acetate
EE	Ether extract
FAO	Food and Agricultural Organization
Fig	Figure.
g/dl	Gram per decilitre
GIT	Gastrointestinal tract
h	Hour
HGB	Haemoglobin
i.u	International Unit
Kcal	Kilo-Caloric
L	Litre
LA	Lactic Acid
LPS	lipopolysaccharide
M	Mole
ME	Metabolisable energy
mg	Milligrams
mg/L	Milligrams per litre
ml	Millilitre
mM	Millimole

MMT	Million Metric Tons
N	North
NDF	Neutral Detergent fibre
ODH	Oxalyldehydrzide
OIE	Office of International des Epizootic
PCV	Packed Cell Volume
RAGFAR	Reference Advisory Group on Fermentative Acidosis of Ruminants
RBC	Red Blood Cell
SARA	Sub-Acute Ruminant Acidosis
SCFA	Short-Chain Fatty Acids
SEM	Standard Error of Mean
SPSS	Statistical Package for Scientist
TP	Total Protein
UK	United Kindom
USA	United States of America
VFA	Volatile Fatty Acid
via	Through
WBC	White Blood Cell

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background of the Study

Rumen lactic acidosis is a metabolic disease disorder that is caused by ingestion of excessive quantities of highly fermentable carbohydrate feed. These feeds are rapidly fermented in the rumen producing large amount of lactic acid overwhelming the buffering capacity of the rumen and resultanting in rumen acidosis, rumen stasis, metabolic acidosis, dehydration, recumbency, hypovolemic shock and death (Anderson, 1992; Wendy, 1992). Morbidity varies from 10-50% depending on type and preparation of the fed grain. The case fatality rate may be up to 90% in untreated cases and between 30-40% in the treated (Radostist *et al.,* 2009). In domesticated ruminants, lactic acidosis may occur as a consequence of ingestion of large amount of grain, especially when the rumen microbe population is poorly adapted to deal with grain (Pugh, 2002). Ruminant acidosis occurs in dairy (Penner *et al.,* 2009) and beef cattle (Steele *et al.,* 2012). However, the most common form of ruminal acidosis in dairy cattle is thought to be sub-acute ruminal acidosis, whereas, acute lactic acidosis is thought to be the primary form in beef cattle fed high concentrate diets (Schwaiger *et al.,* 2013). Sub-acute ruminal acidosis is caused by a rapid rate of Volatile Fatty Acid (VFA) production while, for acute acidosis is as a result of activity of various rumen microorganisms resulting in accumulation of various VFA (acetic, propionic and butyric acids) and lactic acid which are partially dissociated (Kahn, 2005). The resulting production of large quantities of VFA and lactic acid decrease rumen pH and simultaneously weakening the buffering capacity of saliva in the rumen, and reduces the efficiency of rumen flora and fermentation (Kim *et al.,* 2013).

Sheep (*Ovis aries*) typically consume a diet high in fiber consisting of fresh forage or hay, with very little energy concentrate feed. Energy concentrates contain a high density of nutrients, usually low in crude fiber (less than 18% of dry matter) and high in total digestible nutrients (FAO, 2010). Energy concentrate feed provides a source of easily digestible carbohydrates, but if fed in excess can lead to ruminal acidosis (Kleen *et al.*, 2003; Nordlund, 2003; Castillo *et al.*, 2006; Rustomo *et al.*, 2006; Nagaraja, and Titgemeyer 2007;).The rumen constitutes an effective animal-microbe mutualism system from which both partners derive benefit (Krause *et al.*, 2003). High-producing beef and dairy cattle use highly fermentable diets to increase growth rates and milk production; they predispose cattle to digestive disorders such as ruminal acidosis (Khafipour *et al.*, 2009).

High fermentable diets are rapidly converted to organic acids [i.e., short-chain fatty acids (SCFA) and lactic acid] within the rumen. The resulting releases of protons can constitute a challenge to the ruminal ecosystem and animal health. Health disturbances, result from acidogenic diets, are classified as sub-acute and acute acidosis best on the degree of ruminal pH depression (Aschenbach *et al., 2011*). Although increase acid production is a nutritionally desired effect of increased concentrate feeding, the accumulation of protons in the rumen is not. Consequently, mechanisms of proton removal and their quantitative importance are of major interest. Saliva buffers (i.e., bicarbonate, phosphate) have long been identified as important mechanisms for ruminal proton removal. An even larger proportion of protons appear to be removed from the rumen by SCFA absorption across the ruminal epithelium, making efficiency of SCFA absorption a key determinant for the individual susceptibility to sub-acute ruminal acidosis (Aschenbach *et al., 2011*).

After the consumption of a high grain diet, non-structural carbohydrates will activate the rumen by physiological process, promoting their fermentation by amylolytic bacteria,

producing pyruvate and finally volatile fatty acids (VFA), dissociating, and producing a drop in ruminal pH. This drop implies that many gram negative (−ve) bacteria will disappear, including lactate-consuming bacteria, like *Megasphaera elsdenii* and *Selenomonas ruminantium* (which convert lactate to pyruvate), because they are sensitive to pH. Conversely, there is an increase in the population of some gram positive (+ve) bacteria, especially *Streptococcus bovis,* known as a lactate-producing bacteria; this property contributes further to the decline in ruminal pH, growing only bacterial pH resistance, like *Lactobacilli spp.*, great lactate producer, especially for D-lactate. Acid will be undissociated, crossing the ruminal wall to the bloodstream and provoking a metabolic acidosis (Joaquin *et al.,* 2014).

1.2 Statement of Research Problem

In livestock production, ration is formulated to meet the body demand for maintenance, growth and reproduction with the assumption that the animal will maintain a healthy state under favourable environmental conditions. Account is not taken of the fact that any disturbances change the equilibrium and hence calls for more nutrient demand. Similarly, research on diseases place emphasis on disease process without adequate attention given to the role that nutrition would have in the cause, treatment and prevention.

The economic importance of sheep in a developing country cannot be over-emphasized. Sheep with their small body size, high productive capacity and rapid growth rates are ideally suited for production by resource-poor smallholders. In sub-saharan Africa, sheep provide almost 30% of the meat consumed and around 16% of the milk produced, and about 50% of the total domestically produced meat in Nigeria (Ayantunde *et al.*, 2008).

Ruminants inproduction settings face disease challenges similar to other livestock which is influenced by many factors, including immune status, level of stress, pathogen load, environment, nutritional background, and feeding management, as well as the level of

execution of animal husbandry practices. They are primarily susceptible to infections and metabolic diseases (Joaquin *et al.,* 2014).

Lactic acidosis among domestic ruminants has been well known as a management disease for many years.Clinically, is characterized by anorexia, depression, abdominal distension, weakness and inactivity. The disease is commonly encountered due to unintended ingestion of large quantities of cereal grains or their flour kept for human consumption (Mohammed-Nour *et al.,* 1998).

Ruminal acidosis is increasingly recognised as a significant disorder of ruminants. This condition increases the morbidity and mortality of stock, markedly reduces weight gains in the feedlot, complicates drought feeding strategies for sheep and cattle, and is increasingly recognised in pastoral and confined dairying. It may be the most significant health disorder of ruminants fed on high-quality pastures and grain (RAGFAR, 2007).

Over 10% of households in humid/sub humid tropical Africa keep sheep. Sheep thus supplement cattle in providing meat as well as income to large population. Commonly, women usually keep these grain preparations carelessly and sheep accidentally get access to them, and excessively consume it resulting to ruminal acidosis and death (Joaquin *et al.,* 2014).

In many dairy operations, the challenge is sub-clinical acidosis. The major clinical manifestation of sub-clinical acidosis is reduced and/or cyclic feed intake, decreased efficiency of milk production, poor body condition despite adequate energy intake, unexplained diarrhoea, episodes of laminitis and death (James, 1995). Animals are usually affected to varying degrees, from mild indigestion to severe poisoning. Acute cases show staggering, appear blind and drunk‘ and go down after 10 to 48 hours. Death can occur 12 to 72 hours after the onset of signs (Joaquin *et al.,* 2014).

A major challenge regarding ruminant nutrition is lack of information on how much of whole or crushed maize grains sheep could consumed that could cause metabolic acidosis but achieving its purpose of market weight. Carbohydrate is a major feed component of sheep ration has its attending disorder notably acidosis Lack of quick intervention when a ruminant gets access to readily fermentable carbohydrate results in death (James, 1995).

This research was aimed at determining the level at which it is desirable to feed sheep with whole or crushed maize and *Digitaria smutsii* hay for increased performance without causing metabolic acidosis.

1.3 Justification of the Study

In Nigeria, there is dearth of information on how much grain could be given to sheep without causing metabolic acidosis. Small ruminants are kept by farmers and in many households in Nigeria and provide the populace with a very good source of protein and other by small ruminant products such as manure, hide, horn, and hoof for glue and fertilizer. Notably, is the economic benefits derived from sheep as source of income, commonly described as the small farmer holders “bank” that is quite handy, moderate and convenient, similar to the money cards of today (FAO, 2010).

Ruminal adaption is critical in determining the amount of carbohydrate that will induce acidosis (Nagaraja and Town, 1990). A critical challenge for ruminant fermers is to identify occurrence of both sub-clinical acidosis and laminitis and make appropriate adjustments in feeding and husbandry management practices (James, 1995). Fulton *et al.* (1979);Nocek, (1987); Nagaraja and Town, (1990); and Nocek, (1992) reported that the amount of feed necessary to induce ruminal acidosis depends on the type of carbohydrate processing,

adaptation period, nutritional status of the animal, and frequency of which the carbohydrate is fed.

This study was therefore aimed at determining the amount of maize that may cause metabolic acidosis in sheep. The outcome of this study could be used by farmers and household (small ruminant keepers) on the appropriate adjustments in feed and feeding for increased performance and avoidance of metabolic acidosis.

1.4 Aim of the Study

To determine lactic acid production in sheep fed whole or crushed maize grains in addition to *Digitaria smutsii* hay.

1.5 Objectives of the Study:

The objectives of the study were to determine the:

i. the amount of whole or crushed maize grain fed to sheep in addition to *Digitaria smutsii* hay that could not cause metabolic acidosis but increase performance (weight gain)
ii. clinical manifestation in sheep fed with whole or crushed maize grain and *Digitaria smutsii* hay.
iii. the level of acetic and lactic acids in sheep fed with whole or crushed maize grain and *Digitaria smutsii* hay.
iv. PCV, RBC and WBC counts of sheep fed with whole or crushed maize grain and *Digitaria smutsii* hay.
v. Gross and histopathology of sheep fed with whole or crushed maize grain, and *Digitaria smutsii* hay.

1.6 Research Questions

1. What level of whole or crushed maize grain and *Digitaria smutsii* hay fed to sheep could increase weight gain without causing metabolic acidosis?
2. What are theclinical manifestations in sheep fed with whole or crushed maize grain in addition to *Digitaria smutsii* hay?
3. What are the levels of whole or crushed maize grain and *Digitaria smutsii* fed to sheep that could produce increased levels of acetic acid and lactic acid?
4. What effect could the feeding whole or crushed maize grains and *Digitaria smutsii* hay have on PCV, RBC and WBC count of sheep?
5. What are the Gross and histopathological findings in sheep fed withwhole or crushed maize grain in addition to *Digitaria smutsii* hay?

CHAPTER TWO

2.0 Literature review

2.1 Acidosis

Acidosis is a pathological condition associated with the accumulation of acid or depletion of alkaline reserves in blood and body tissues, and characterised by increased hydrogen ion concentrations (Blood and Studdert, 1988). Ruminal acidosis is a nutritional disorder of ruminant which receives a lot of attention by the feedlot industry even though it occurs with relatively low frequency. Ruminal acidosis in feedlot cattle is a common metabolic disorder of digestive origin with significant economic and welfare implications. The main risk factors are high grain, low roughage diets because of their high rate and extent of degradation by rumen microbes (González*et al* 2012).

Ruminal acidosis is frequently defined as a decrease in the ruminal pH. But the question is whether this condition is a disease or not. So, many researchers recognized that acidosis is not one disease, but rather a continuum of degrees of ruminal acidity, because nonphysiological accumulation of organic acids and consequent reduction in pH below the normal have a significant impact on microbial activity, rumen function, and animal productivity and health (Nagaraja and Lechtenberg, 2007).

Accordingly, it may be better to define ruminal acidosis as a fermentation disorder in the rumen characterized by a lower than normal ruminal pH, but reflecting an imbalance between microbial production, microbial utilization, and ruminal absorption of volatile fatty acids (VFA) (Castillo*et al.,* 2012). Some 30% to 50% of the acid in the rumen is neutralized by

salivary buffers or bound to ammonia generated from urea entering across the ruminal wall. A smaller quantity passes on into the lower gastrointestinal tract (González*et al.*, 2012 and Nocek, 1997). However, even the most conservative estimates leave a significant proportion of about 30–50% of the acid that is ruminally produced and that has to be absorbed by the ruminal wall, and one of the most important reasons for the appearance of ruminal acidosis would be a decrease in the absorptive capacity of the rumen which is thus unable to maintain a stable pH (Joaquín, 2014).

Absorption of VFA, by removing unionized acid and by the exchange of ionized VFA for bicarbonate during the absorption process, aids in maintaining pH near neutrality. Consequently, a reduced rate of VFA absorption causes ruminal pH to drop for two reasons: ruminal VFA accumulate and bicarbonate input from the blood stream is decreased (Owens*et al.,* 1998).

Ruminants are adapted to digest and metabolise predominantly forage diets. The ruminal microflora of ruminants entering a feedlot consists mainly of cellulolytic gram negative bacteria with protozoa. Fermentation occurs under anaerobic conditions. As a consequence sugars are metabolised predominantly to volatile fatty acids.Sheep (*Ovis aries*) typically consume a diet high in fibre consisting of fresh forage or hay, with very little energy concentrate feed. Energy concentrates contain a high density of nutrients, usually low in crude fibre (less than 18% of dry matter) and high in total digestible nutrients (FAO, 2010). Energy concentrate feed provides a source of easily digestible carbohydrates, but if fed in excess can lead to ruminal acidosis (Kleen*et al.,* 2003;Nordlund, 2003;Castillo*et al.,* 2006;Rustomo*et al.,* 2006;Nagaraja and Titgemeyer, 2007). Mastication of grass hay stimulates the production of copious amounts of saliva, which act to buffer the ruminal pH. Normal ruminal pH in sheep is between 6.4 and 6.8. Ruminal pH values less than 5.5 or

greater than 7.0 are considered abnormal; ruminal pH of less than 5.5 is defined as subacute rumen acidosis (House*et al.,* 1992; Kleen*et al.,* 2003; Owens*et al.,* 1998;Beauchemin and Yang, 2005).

Grains are often used in the late summer and autumn months as supplements to fill feed shortages in dryland situations as well as to put condition on ewes pre-tupping. However, just like kids with ice cream and given the opportunity, sheep will often gorge themselves if they are introduced suddenly to grain feeding. This is especially true if they are hungry before the grain is introduced i.e. the introduction of grain to a flock in drought situations where little feed is available (Fact sheet, 2013).

2.1.1 Pathogenesis of Acidosis

Cattle and sheep have a large fore-stomach, the rumen, which contains a stable population of microorganisms. These microorganisms derive energy mainly by fermenting the carbohydrates which the host animal ingests. The events leading to acidosis occur when the animal's diet is suddenly changed from forage to concentrate (high in starch or other rapidly fermentable carbohydrates), or when it is fed excessive amounts of such concentrates (McSweeney and Mackie, 1997; Galyean, 2001). Introduction of starch into the rumen, or a sudden increase in the starch supply, leads to rapid fermentation and increased production of volatile fatty acids (VFA). Furthermore glucose, normally found in extremely low concentrations in the rumen, is liberated from starch or other rapidly fermented carbohydrates, resulting in increased ruminal glucose concentrations. This has negative consequences including growth of organisms such as *Streptococcus bovis* and other lactic acid producing organisms, and increased ruminal osmolality, which further increases ruminal acidity by inhibiting VFA absorption from the rumen (Galyean, 2001). As the rate of VFA production exceeds their rate of removal, rumen pH may fall below 6.0. *Streptococcus*

*bovis*and other lactic acid producing bacteria (especially the *Lactobacilli species*) are more tolerant to these acidic (low pH) conditions and they start to replace the other normal rumen microorganisms to become the major components of the microbial population in the rumen (Dawson*et al.,* 1997). At the same time organisms such as *Megasphaera elsdenii*, which would normally prevent lactate accumulation by metabolizing lactate to VFA's, are inhibited. Lactic acid is a 10 times stronger acid than the VFA's and pH continues to fall further until even the resilient *S. bovis* can no longer grow and *Lactobacilli* take over, further fermenting the starch to produce even more lactic acid a self-perpetuating cycle of lactate production leading to ruminal pH values below 5.0. This whole process may be very rapid and overgrowth by lactic acid bacteria can occur within 24 hours (Dawson*et al.,* 1997). Lactic acid, which is normally found at very low concentrations in the rumen, can increase to around 100 mM in severe cases of acidosis (McSweeney and Mackie, 1997).

Under the above conditions the ciliate protozoa population, which normally has a stabilizing effect on ruminal fermentation, declines because the ruminal pH is below their growth optimum (McSweeney and Mackie, 1997). These protozoa have an important role in regulating the production of lactic acid and VFA's in the rumen. They ingest starch, soluble sugars and bacteria, thereby reducing the rate of fermentation in the rumen. They also appear to be involved in the metabolism of lactic acid. The absence of a stable and active population of ruminal protozoa is therefor likely to increase the severity of lactic acidosis (McSweeney and Mackie, 1997).

Lactic acid production increases osmotic pressure within the rumen so that fluid is drawn into the rumen from the circulatory system as well as other tissues. The rumen pH drops resulting in rumen stasis. The D- and L- lactic acids are converted to sodium lactate. In the rumen they contribute to hyper tonicity. D-lactate, because of its slower rate of metabolism, is absorbed

into the circulation and contributes to the depression of blood pH. There is some absorption of lactate from the omasum andabomasums and some as a result of continuing fermentation in the small intestine. An osmotic gradient is established within the intestinal tract, drawing fluid into the rumen, contributing to the profuse diarrhoea (Henning, 2004).

Ruminal epithelial cells are not protected by mucus, so they are vulnerable to chemical damage by acids. Low ruminal pH leads to rumenitis, erosion and ulceration of the ruminal epithelium. Once the ruminal epithelium is inflamed, bacteria may colonise the papillae and leak into the portal circulation. *Fusobacterium necrophorum*, a gram negative obligate anaerobic bacterium and a component of normal rumen microflora, is the primary etiologic agent of ulceration of the ruminal epithelium (Henning, 2004). Two biovars have been implicated. Biovar A (*F. necrophorum necrophorum*), the more virulent, is the most predominant biovar in the rumen microflora and is isolated usually in pure culture, from most cases of liver abscessation. Biovar B (*F. necrophorum fundiliforme*) is commonly isolated from micro-abscesses in the rumen wall and is less commonly isolated from liver abscesses, in which it is always found in mixed culture with Biovar A, or other bacterial species. *Arcanobacterium pyogenes*, Streptococci, Staphylococci and Bactroides (Henning, 2004).

These bacteria may cause liver abscesses, which may eventually lead to peritonitis around the site of infection. Most liver abscesses are occult lesions that regress to a sterile abscess. Untoward sequelae include peritonitis after abscess rupture into the peritoneal cavity (Henning, 2004). If the ruminal bacteria go through the liver undetected (or if bacteria from the liver infections are released into the circulation) this may result in the caudal vena cava syndrome. They may colonise the lungs, heart valves, kidneys or joints, resulting in pneumonia, endocarditis, pyelonephritis or arthritis. The infection of the lungs (chronic suppurative pneumonia) as a result of the septic emboli, may lead to the invasion of

pulmonary blood vessels, causing them to rupture into the airways. This leads to hemoptysis, epistaxis and per acute deaths due to massive pulmonary haemorrhage (Henning, 2004).

Acidosis is categorized as acute or subacute primarily on the basis of presence or absence of various symptoms (Nagaraja*et al.,* 1998).

2.1.2 Acute acidosis

An acute acidosis problem in ruminants is the result of excessive consumption of fermentable carbohydrates which causes a non-physiological reduction in pH and the production of a toxic factor(s). The low ruminal pH is the result of the production of large quantities of volatile fatty acids as well as other acids (such as lactic, which has a pH of 3.7) and the weaker buffering power of concentrates compared with that of forages (in the pH range of six to four in the rumen). It is most likely to occur if glucose accumulates in conjunction with ruminal pH reductions to 5.0 or less. Acidosis problems should occur in the absence of ruminal glucose accumulation if ruminal pH is sufficiently low and if other readily fermentable carbohydrates which aciduric bacteria can utilize are available in excess. Wheat seems more prone to cause acidosis than other farm grains (Slyter, 1976). Sheep with acute ruminal lactic acidosis included a disturbed general condition characterised by anorexia, apathy, teeth grinding and muscle twitching, ruminal stasis, the excretion of soupy or watery faeces, rumenitis, dehydration, laminitis and liver abscesses. The ruminal fluid of affected animals was milky, had a sour odour and a low pH (McSweeney and Mackie, 1997). The abovementioned changes in rumen microbial population, as well as a reduction or complete absence of ciliated protozoa will also be evident(Nagaraja*et al.,* 1998).

Acute acidosis is easier to detect, and if diagnosed early, can be treated directly, or measures taken to prevent further episodes, subacute acidosis is probably the most prevalent form, and more difficult to detect and treat (Nagaraja*et al.,* 1998).

2.1.3 Subacute acidosis

Subacute acidosis on the other hand decreased and/or erratic feed intake may be the only sign, and this may be difficult to observe for individuals in group-fed cattle. It should be clear that ruminal acidosis cannot merely be defined as a condition of low ruminal pH but that it is best described as a syndrome related to a fermentative disorder of the rumen (Hall, 2002). Another important point is that subacute acidosis does not involve lactate accumulation in the rumen (Goad*et al.,* 1998), and that the term "lactic acidosis", if used correctly, can only refer to "acute acidosis". From the foregoing it is also evident that, although a distinction is often made, subacute and acute acidosis are not two distinct "diseases" but that subacute acidosis can easily develop into acute acidosis if the appropriate circumstances deteriorate (Henning, 2004).

2.2 Clinical Picture of Acidosis

The onset of the clinical signs associated with ruminal acidosis depends on the clinical form, varying from sudden death in peracute course to depression in subacute way(Joaquín, 2014).

Direct action of VFA is one of the most important mechanisms to consider, since chemical receptors in the epithelium send a feedback signal to the brain to reduce ruminal motility. When VFA contacts with them, signal will be sent to the central nervous system, promoting ruminal atony (González*et al.*, 2012)

Another mechanism to promote the rumen hypomotility is related to the increase in the osmolality in the ruminal content, produced by the accumulation of organic acids and glucose

increasing the osmotic pressure inside the rumen, which implies a water flux from the bloodstream across the rumen wall, sometimes producing a hydrorumen. As a consequence of hydrorumen, animal will show a decrease in packed cellular volume, with haemoconcentration and sometimes polyuria, with the animal feeling dehydrated (Kleen *et al.,* 2003). Taking into account an abnormal composition of the ruminal juice, animal may show diarrhea, which will complicate the hydroelectrolytic balance of the animal. Considering that the structure and consistency of the faeces depend on rumination, activity of the ruminal flora and ruminal passage, the animal will show some changes in colour, odour, pH, and consistency, and even whole cereal grains may be present. The impaired ruminal function in terms of rumination, bacterial breakdown, and passage leads to the alteration in faecal aspects (Kleen *et al.,* 2003;Kleen, 2004).

The third mechanism involved in this hypomotility is the different vasoactive substances, such as histamine, tyramine, and tryptamine, which are produced in the rumen by decarboxylation of histidine, tyrosine, and tryptophan, respectively and also Bacterial endotoxins which have been related to the decrease in rumen motility although the exact mechanism remains unclear (Kleen *et al.,* 2003;Kleen, 2004).

The growth of ruminal epithelium has been shown to be directly linked to the nonstructural carbohydrates presence in the tissue. Propionic and butyric acid are promoting the growth of the ruminal papillae, thus providing a higher absorption from the rumen by the mucosa, but, in a low ruminal pH, with excessive amount of VFA, these will lead to a parakeratosis of the ruminal epithelium, and resultant rumenitis, characterised by the presence of microabscesses within the ruminal mucosa, with *Fusobacterium necrophorum* and *Arcanobacterium pyogenes,* colonizing the liver tissue and spreading to other organs like kidneys, heart, and lungs (Norlund*et al.*, 1995; Nagaraja, 2000) and promoting the parakeratosis-rumenitis liver

abscesses complex (Nagaraja, 2000) and One important complication of this is that, as a consequence of the ruminal mucosae destruction, many anaerobic bacteria will be able to cross the ruminal wall, incorporating with the bloodstream and favouring infections like pneumonia, pyelonephritis, and typical endocarditis (Joaquín, 2014).

Another symptom is that the animal would develop polioencephalomalacia, produced by a B1 vitamin (thiamine) deficit. The bacteria in the rumen normally create this vitamin, so ruminants do not normally need it in feed. So, thiamine inadequacy can be caused by decreased production by rumen microbes or factors that interfere with the action of thiamine, for example, plant thiaminases or thiamine analogs. which can be produced by gut bacteria or ingested as preformed plant products and can either destroy thiamine or form antimetabolites that interfere with thiamine function. Thiaminase I, produced by *Bacillus thiaminolyticus* and *Clostridium sporogenes*, and thiaminase II, produced by *Bacillus aneurinolyticus*, catalyze the cleavage of thiamine. The latter microorganism proliferates under conditions of high grain intake with resultant Neurologic symptoms including depression, anorexia, blindness, convulsions, incoordination and opisthotonos in standing position, and even animals show a typical star grazing stand(Abeysekara*et al.*, 2007). It is important to point out that many of the neurologic signs are not promoted by thiamine deficit, because the serum D-lactic acid increase allows it to cross the blood-brain barrier by monocarboxylate protons transporters. The majority of neurological disturbances (i.e., ataxia and depressed menace, palpebral, and tactile reflexes) are related to D-lactate accumulation in cerebrospinal fluids rather than in blood (Abeysekara*et al.*, 2007).

One clinical sign regularly mentioned to be associated with ruminal acidosis is laminitis (Kleen *et al.*, 2003), or pododermatitis aseptica diffusa, which is an aseptic inflammation of the dermal layers inside the foot. Nutritional management has been identified as a key

component in the development of laminitis, particularly the feeding of increased fermentable carbohydrate, which results in an acidotic state. It is suspected that there are vasoactive substances entering the bloodstream from the rumen, leading to damage in the corium. The initial problem is thought to be metabolic in nature like a low ruminal pH, which allows a chain of pathological mechanisms to take place, eventually leading to ischemia of the distal limb and a clinically detectable form of laminitis, manifesting by blood imbibition of the sole during acute phases of the disease and classical picture of hoof deformation as the disease becomes chronic (Kleen *et al.,* 2003). Histamine lipopolysaccharide endotoxin (LPS) (Nocek, 1997)and lactate are biological active agents suspected to interact in this complex (Kleen, 2004), although it is true that the most important histamine producer, *Allisonella histaminiformans*, increases at alkaline pH, although it is able to grow at ruminal pH around 4.5. This fact implies that, probably, the main reason for the relationship between acidosis and laminitis will be not only an increase in the histamine production, but also a decrease in the histamine destruction, because, at low pH, there is a decreased diamine oxidase activity, promoting a histamine increase net flux from rumen to bloodstream (Kleen *et al.,* 2003). The role of tyramine and tryptamine, other vasoactive substances related to vascular episodes in the corium of the hoof and produced from tyrosine and tryptophan, respectively, remains unclear at this moment in the pathogenesis of the process (Kleen *et al.,* 2004). Bacterial endotoxins present in ruminal fluid also have been named as a possibly causative agent in the bovine laminitis complex. In an acidotic environment, the ruminal flora changes to a mainly gram-positive pattern. It has been shown that there is a detectable increase in endotoxins in the rumen, probably derived from the breakdown of the gram-negative bacteria (Kleen *et al.*, 2003), that damage the capillaries of the lamellae in the foot and cause hemorrhage, inflammation, and lameness (Nocek, 1997), albeit it has been demonstrated that grain-induced Subacute ruminal acidosis(SARA) increased free LPS in the rumen but not in

peripheral blood, which disagrees with the hypothesis that LPS damages the capillaries of the hoof (Gozho*et al.,* 2007).

Finally, as a consequence of the metabolic acidosis, animal could show symptoms like hyperventilation and signs derived from compensatory hyperkalemia. Note that hyperkalemia could develop from itself ventricular fibrillation or cardiac arrest, producing the death of the animal in some circumstances (Joaquín, 2014).

2.3 Diagnosis

In addition to a detailed clinical examination, sheep may require ancillary laboratory tests and/or additional diagnostic procedures before a definitive diagnosis may be reached.The recommended protocol for sub-acute ruminal acidosis diagnosis is the collection of ruminal fluid by rumenocentesis (Garrett *et al.,* 1999; Nordlund *et al.,* 1995) and Rumenocentesis is a practicable diagnostic procedure for routine determination of ruminal pH and diagnosis of sub-acute ruminal acidosis in dairy sheep.

is recommended by Enemark *et al.,* (2002) as a better field test in comparison to oro-ruminal probe for measurement of rumen pH.

2.4 Ruminal acidosis and the relationship with feed intake

Animals exhibit acute acidosis as an overt illness following consumption of readily fermentable carbohydrates in sufficient amounts to threaten life (Nagaraja and Titgemeyer, 2007). With sub-acute acidosis, feed intake and performance might be reduced but animals

do not appear sick (Owens *et al.*, 1998). However,the economic impact caused by the effects of sub-clinical acidosis on animal performance could be greater than those of acute acidosis (Britton and Stock, 1989). Ruminal fluid pH of 5.6 or lower is considered the benchmark for sub-acute ruminal acidosis whereas a pH below 5.0 is considered the benchmark for acute acidosis (Owens *et al.*, 1998, Krause and Oetzel, 2006 and Nagaraja and Titgemeyer, 2007). In acute acidosis, ruminal fluid pH reaches such low levels because of the accumulation of lactic acid as a result of increased production while its utilization by ruminal microbes is reduced because lactic acid is not fermented any more (Nagaraja and Titgemeyer, 2007). Subsequent absorption of organic acids into the bloodstream might overwhelm the bicarbonate buffering system, the excretion rate of acids, and the capacity of tissues and organs to metabolize acids resulting in systemic acidosis (Brown *et al.*, 2000). In sub-clinical acidosis, the reason for pH dropping below 5.6 is the accumulation of volatile fatty acids. Although lactic acid is produced during sub-clinical acidosis, it does not accumulate because lactate-fermenting bacteria remain active (Goad *et al.*, 1998) and rapidly metabolize it to volatile fatty acids. Ruminal fluid pH does not always explain the typical signs and symptoms of ruminal acidosis although it is the most commonly used indicator (Huber, 1976). Both a reduction in feed intake and variation in feed intake among days have been used as indexes of subclinical acidosis based on the concept that an increased variability from day to day is associated with feeding acidogenic diets (Britton and Stock, 1989, Stock *et al.*, 1995 and Bevans *et al.*, 2005). However, the reduction in feed intake in acidotic sheep is inconsistent and may depend on the extent and convergence of multiple factors associated with low ruminal pH and the control mechanisms of feed intake. Many theories for the reduction of dry matter intake during sub-clinical acidosis have been hypothesized: low ruminal fluid pH, high concentration of fermentation products (volatile fatty acid, VFA), high osmolality, inflammatory (acute-phase) responses, reduced rumen motility, and reduced fibre digestion.

These changes might be detected by sensors in the rumen wall, or within the body after the changes become systemic through the blood and sensed by the brain or different organs such as the liver (Forbes and Barrio, 1992 and Allen *et al.,* 2005). Also, low pH of the rumen fluid may reduce fibre digestion which reduces rate of passage and may eventually reduce feed intake (Plaizier *et al*., 2009). However, this theory is less likely in feedlot cattle because of the low fibre content of their rations (González*et al* 2012).

2.5 Acid–base balance in the rumen

The process of eating and the fate of ingested feed in ruminants have a great influence on the acid–base balance of rumen fluid. After being chewed in the mouth, the ingested feed goes to the reticulorumen where fermentation by microbes results in the production of organic acids. The amount of organic acids produced at a given point in time after ingestion depends on the amount of feed ingested (meal size), and the rate and extent of degradation of feed by the rumen microbes. After dissociation, these organic acids liberate protons which may decrease ruminal fluid pH. However, this reduction in pH depends on the amount of saliva added to the feed while eating (which neutralizes the acids) and the existing buffering capacity of the ruminal fluid (resistance to pH change). Feed is chewed in the mouth to reduce particle size, moisten it and form the bolus for easy swallowing. The length of time spent chewing per unit of feed depends on the type of feed, eating rate and determines feed ensalivation (mL of saliva added per gram of feed). The greater the eating rate, the lower the amount of saliva per unit of feed is added. Eating rate is influenced by the proportion of forage, particle length and initial moisture content of feedstuffs (Bailey, 1961 and Beauchemin *et al.,* 2008) but also by other factors such as hunger, feeding management (Carter *et al.,* 1990), competition for feed or social pressure from pen-mates (Olofsson, 1999) and even the presence of predators (Illius and Fitzgibbon, 1994). Ruminants also regurgitate the feed from the rumen back to the mouth

to chew it again and further reduce particle size until particles are small enough to pass from the rumen to the lower digestive tract (Allen, 1997).

When grazing, sheep tend to select the more digestible parts of the plants than cattle. Sheep also have less powerful jaws and need a longer time chewing their cud and they grind their food particles finer than cattle. Sheep spend a shorter time eating than cattle, but a longer time ruminating. On pasture sheep spend half the time eating compared to cattle, but almost four times longer chewing. It is unclear whether the pathophysiology of acidosis differs substantially between sheep and cattle, but it generally appears to be similar (RAGFAR, 2007). Saliva production is very important for rumen function because it has high buffering capacity as a result of high concentration of bicarbonate (125mEq/mL) and phosphate (20 mEq/mL of phosphate; Bailey and Balch, 1961). Saliva production during eating, ruminating and resting contributes to neutralizing around 0.30 of the acids produced daily in dairy cows (Erdman, 1988, Allen, 1997 and Russell, 2002). However, this figure should be lower in feedlot cattle because of shorter chewing time and consequently, lower saliva production per day. In addition to neutralization by saliva, acids produced during fermentation are eliminated from the rumen by absorption through the ruminal epithelium, which eliminates around 0.50 of the total acids produced daily (Allen, 1997 and Russell, 2002). Moreover, liquid and solids passing out from the reticulorumen wash out approximately 0.15–0.20 of the protons produced daily from which 0.10 pass associated to phosphates (Allen, 1997). In addition to chewing time and meal size, the distribution of daily feed intake throughout the day and meal frequency also play an important role on ruminal acid–base balance. Greater meal frequency and wider distribution of daily intake throughout the day lead to a better synchronization in time between bouts of acid production and saliva production (particularly if rumination occurs between meals), and absorption and passage of organic acids from the rumen.

Furthermore, time periods between feeding events allow timely metabolization of acids (e.g. oxidation in the liver) and elimination through the urine of protons absorbed from the rumen (González*et al* 2012).

2.6 Factors affecting feeding behaviour and ruminal fluid pH

Factors affecting feeding behavior, and consequently ruminal fluid pH, can be divided into those related to feed characteristics, feeding or animal management, and the environment (González*et al* 2012).

2.6.1 Feed related factors affecting feeding behavior and rumen function

From a practical point of view, two strategies related to feed formulation are useful to prevent ruminal acidosis. The first is related to diet formulation including the choice of grain and forage in terms of type and proportion in the diet. The second is the use of feed additives such as buffers, natural plant extracts, ionophores, yeasts, direct-fed microbials, organic acids or polyclonal antibodies against *Streptococcus bovis* (González*et al.*, 2012).

2.6.2 Amount and type of grain

Feedlot diets are commonly based on high-concentrate diets where cereal grains are the main ingredient and the content of non-structural carbohydrates is high. Structural carbohydrates have slow rumen degradation rates as opposed to the fast rate of starch, sugars and pectins. Acidosis is most prevalent following ingestion of large amounts of starch present in cereal grains. The contribution of starch to the acidogenic power of a diet depends on the starch content and its rate of degradation in the rumen. According to starch content, grains are ranked in decreasing order as follows: wheat (0.78), corn and sorghum (0.72), and barley and oats (0.58), whereas NDF content follows the opposite pattern (Huntington *et al.*, 2006). There is a strong negative correlation ($r = -0.97$) between ruminal fluid pH and digestible

starch content of a diet (Sauvant *et al.,* 1999). The rate of cleavage of starch to glucose is related to the ruminal degradation rate and varies with grain source, grain processing, and starch type (Owens *et al.,* 1997 and Huntington *et al.,* 2006). In general, metabolizable energy from grains increases with both ruminal degradation and total tract digestion. Ruminal degradation rate and extent depends on the chemical and physical structure of starch granules such as size, shape and embedment within the protein matrix and both concentration of the soluble fraction and the fractional degradation rate of starch have a great influence on ruminal degradability, both being greater for rapidly degrading starch feedstuffs (Offner *et al.,* 2003). Several processing methods are known to increase ruminal degradability of starch such as reduction of particle size, thermal treatment (e.g. steam flaking) and shear forces (e.g. extrusion). Based on ruminal starch degradation rate, grains are ranked from fastest to slowest as follows: oats, wheat, barley, high moisture corn, steam flaked corn, dry rolled corn, dry whole corn, and dry whole milo (Herrera-Saldana *et al.,* 1990, Huntington, 1997, Offner *et al.,* 2003 and Huntington *et al.,* 2006). The choice of grain type and processing is therefore one of the most important considerations to prevent ruminal acidosis. It is desirable to increase the extent of grain digestion without increasing the risk for ruminal acidosis and causing a reduction of DMI (Owens *et al.,* 1997). New grain varieties or hybrids are therefore becoming available in the market with characteristics such as harder or more vitreous endosperm (e.g. flint vs. dent or floury corn) resulting in a slower rate and extent of ruminal digestion and therefore, higher ruminal fluid pH (Taylor and Allen, 2005a and Jaeger *et al.,* 2006).

The acidogenic effect of a diet increases with the proportion of concentrate and rate and extent of ruminal degradability of the ingredients (Fulton *et al.,* 1979 and Murphy *et al.,* 1994). However, other factors not related to the rate of acid production in the rumen also play

an important role in the acid–base balance. First, diets with lower proportions of forages and smaller particle sizes have greater density allowing faster eating rate and therefore lower feed ensalivation (Beauchemin *et al.,* 2008), and result in larger meals of both total and ruminally degradable DM (Dado and Allen, 1995 and Tolkamp *et al.,* 2002). Similarly, eating rate is faster, time spent eating is shorter and feed ensalivation lower for whole wheat compared to whole corn (Beauchemin *et al.,* 1994), and time spent eating is shorter for vitreous compared with floury corn (Taylor and Allen, 2005b). Second, time spent ruminating per day and per unit of intake is also reduced more in diets with higher concentrate proportion (Faleiro *et al.,* 2011). Presentation of the diet is also an important factor affecting feeding behavior and ruminal fluid pH. For instance, feeding mixed rations, as opposed to ingredients fed in separate containers, promotes better synchronization in time between acid production and neutralization and elimination. Mixed rations avoid the ingestion of large amounts of concentrates shortly after feed delivery which is typical of feeding separate ingredients, and reduces daily eating rate (DeVries and von Keyserlingk, 2009) which is expected to increase feed ensalivation. However, feedlot cattle fed a mixed ration with 100 g/kg of corn silage DM showed similar ruminal pH to those fed free choice of the ingredients allowed for ad libitum consumption (Moya *et al.,* 2011). However, animals fed free choice of ingredients consumed a greater proportion of corn silage (200 g/kg of DM) and had longer and fewer meals per day with more frequent visits per meal compared those fed the diet containing 100 g/kg of corn silage DM. Third, adaptations to acidogenic diets have to occur at several levels of the animal: rumen microbial populations, epithelium of the digestive tract, metabolism, and behavior. Adaptations in the rumen epithelium help to improve the absorption and metabolism of organic acids whereas other metabolic or physiologic adaptations of the animal help to eliminate acids by oxidation (e.g. in the liver), synthesis of larger molecules, and neutralization and excretion of protons (Aschenbach *et al.,* 2011 and Penner *et al.,* 2011).

However, excessive acid production may also lead to parakeratosis and hyperkeratosis which may consequently reduce the absorption of acids (Dirksen *et al.,* 1985). Adaptation of the rumen microbial populations includes maintaining active lactate-fermenting and reducing lactate-producing bacteria (Nagaraja and Titgemeyer, 2007). Behavioral adaptations to cope with acidotic diets are also very important although research in this area is scarce. For instance, animals consuming a high-forage diet may be adapted to eat a large meal after feed delivery in the morning as they can deal with the low acid production (González*et al* 2012).

However, the acidogeniceffect of such meals increases with the proportion of concentrate and ruminal degradability. Therefore, animals need to change their feeding behavior toward smaller, more frequent meals to avoid the 'harmful' effect of large meals from more fermentable diets. The animal needs first to understand the post-ingestive consequences of diets that are more fermentable (such as unpleasant sensations due to damage in the rumen tissue or high osmolality) to develop the ability to self-regulate and avoid a 'lethal meal' (González*et al* 2012).

2.7 Maize

Maize or corn (*Zea mays*) is an important annual cereal crop of the world belonging to family Poaceae. Zea is an ancient Greek word which means "sustaining life" and Mays is a word from Taino language meaning "life giver." The word "maize" is from the Spanish connotation "maiz" which is the best way of describing the plant. Various other synonyms like zea, silk maize, makka, barajovar, etc. are used to recognize the plant (Kumar & Jhariya, 2013).

It is considered as a staple food in many parts of the world. It is the third leading crop of the world after rice and wheat (Sandhu and Malhi, 2007). The world production of maize was

967 million metric tons (MMT) and in India its production was 23 MMT in 2013–14 (India maize summit, 2014).

Due to its highest yield potential among the cereals it is known globally as queen of cereals. The largest producer of maize is United States of America (USA) contributing about 35% of the total world maize production (Milind and Isha, 2013). It is known as mother grain of Americans and it is the driver of the US economy. In India, the major maize growing states are Uttar Pradesh, Bihar, Rajasthan, Madhya Pradesh, Punjab, Haryana, Maharashtra, Andhra Pradesh, Himachal Pradesh, West Bengal, Karnataka, and Jammu and Kashmir, jointly accounting for over 95% of the national maize production (Milind and Isha, 2013).

Maize is used by agricultural bodies and research institutes such as the FAO. However, in commodities trading, corn consistently refers to maize and no other grains (Utah, 2016).

2.7.1 Nutritional value of Maize

Maize kernel is an edible and nutritive part of the plant. It also contains vitamins C, E, K, B1 (thiamine), B2 (niacin), B3 (riboflavin), B5 (pantothenic acid), B6 (pyridoxine), folic acid, selenium, N-p-coumaryl tryptamine, and N-ferrulyl tryptamine and Potassium which is major nutrient present and is of good significance because an average human diet is deficient in potassium (Kumar and Jhariya, 2013).

Maize germ contains about 45–50% of oil that is used in cooking and for salad is obtained from wet milling process (Orthoefer *et al.,* 2003).

The oil contains 14% saturated fatty acids, 30% monounsaturated fatty acids, and 56% polyunsaturated fatty acids. The refined maize oil contains linoleic acid (54–60%), oleic acid (25–31%), palmitic acid (11–13%), stearic acid (2–3%) and linolenic acid (1%) and maize

silk contains various constituents essential for our diet such as maizenic acid, fixed oils, resin, sugar, mucilage, salt, and fibers (Kumar & Jhariya, 2013).

In a 100-gram serving, maize kernels provide 86 calories and are good sources (10-19% of the Daily Value) of the B vitamins, thiamin, niacin, pantothenic acid (B5) and folate; in moderate amounts, they also supply dietary fiber and the essential minerals, magnesium and phosphorus whereas other nutrients are in low amounts. Maize has been the subject of genetic engineering research to improve levels of carotenoids, such as provitamin A, beta-carotene (Vallabhaneni and Wurtzel, 2009).

2.8 Digitaria

Digitaria is a genus of plants in the grass family native to tropical and warm temperate regions. Common names include crabgrass, finger-grass, and fonio. They are slender monocotyledonous annual and perennial lawn, pasture, and forage plants; some are often considered lawn pests. Digitus is the Latin word for "finger", and they are distinguished by the long, finger-like inflorescences they produce (Gilani, *et al.*2003a)

Though some Digitaria species are weeds, others have uses, especially as food. The seeds, most notably those of fonio, can be toasted and ground into flour, which can be used to make porridge or fermented to make beer. Fonio has been widely used as a staple crop in parts of Africa. It also has decent nutrient qualities as forage for cattle (Gilani, *et al.*2003b).

Digitari smutsii or Eggplant (*Solanum melongena*) is a commonly cultivated nightshade vegetable herb/forb in most ecologic zones of Nigeria. It is a very important vegetable crop cultivated commercially mainly by small scale farmers in most parts of the country all year round (Ozobia *et al.* 2013). The Funtua green striped variety is the most commonly cultivated in Zaria (11o 4’ 0” North, 7o 42’ 0” East) environ (All things are plants 2014).

2.9 Prevention and control of Acidosis

Individual animal can be treated successfully, although the chances of success depend on the severity of the case (RAGFAR, 2007), based on controlling changes associated with systemic acidosis and dehydration (fluid therapy should be applied but lactate enrichment fluids, such as Ringers lactate should be avoided), and trying to correct complications, trying to restabilize ruminal functions (Joaquin *et al.,* 2014).

In the herd, the most important thing to do is to anticipate the ruminal acidosis, and in order to do that the Reference Advisory Group on Fermentative Acidosis of Ruminants (RAGFAR, 2007) have proposed some indirect indicators of ruminal acidosis in feedlot ruminant which include (i)decline in pen feed consumption of more than 10% for two or more consecutive days, causing a weight loss,(ii)a pen incidence of bubbly scours of more than 3% on any given pen inspection,(iii)evidence of laminitis in any Bos taurus cattle and more than 3% of Bos indicus cattle,(iv)a decrease in chewing activity (less than 50% of the calf rest time), due to a decrease in neutral detergent fiber (Joaquin *et al.,* 2014).

But prevention is the most important tool to avoid acidosis appearing. In order to do that, the ruminal pH was kept in physiologic ranges, by increasing the neutral detergent fiber and decreasing concentrate intake and, in a second place, trying to keep ruminal microbiota, which will allow controlling the fermentative process. There are three strategies for the prevention of the high-concentrate syndrome (Calsamiglia*et al.,* 2012): (1) proper diet balancing and feeding management, (2) control of ruminal pH, and (3) control of the fermentation process.

2.9.1 Feeding management

Feeding management is an important consideration in animal production because it may affect production costs and animal performance. According to González *et al* 2012, Feeding behaviour is particularly sensitive to feeding management and may therefore disrupt or help to maintain the acid–base balance in the rumen. For example, feeding practices that reduce meal size and eating rate, increase meal frequency, or result in more uniform feed intake throughout the day are desirable because the risk of ruminal acidosis is reduced. Feeding regimes that act in harmony with feeding behaviour and allow the maintenance of ruminal acid–base balance are desirable whereas those that disrupt the feeding pattern should be avoided.

2.9.2 Feeding frequency

Increasing the frequency of feed delivery during the day might decrease acidosis problems by favoring more stable acid production throughout the day and improving the synchronization with saliva production, and absorption and passage of acids from the rumen (Soto-Navarro *et al.,* 2000). To achieve this, feeding management should promote greater meal frequency or more even distribution of feed intake, reduce meal size particularly of large meals typically occurring after the delivery, and promote rumination at times of the day with low ruminal fluid pH (Soto-Navarro *et al.,* 2000). Several studies have reported that more frequent feeding leads to more stable ruminal pH with fewer daily fluctuations (Soto-Navarro *et al.,* 2000). In theory, more frequent feed delivery is more important for diets with medium to high rates of degradation, during adaptation to more concentrate diets, and poor feeding management including large day-to-day variation in the proportion of forage of the diet or in the amount of feed delivered (Grant and Albright, 1995). Cattle being adapted to higher concentrate diets exhibited greater feed intake when the frequency of feeding increased from 1 to 4 times per day which may be attributed to a reduction of acidosis (Tremere *et al.,* 1968). Multiple

feedings may also reduce the magnitude of feeding errors and abnormal feeding patterns, or promote the self-regulation of short-term feed intake of cattle (Pritchard and Bruns, 2003). In addition, more frequent feeding may reduce social competition for feed and aggression after delivery (DeVries *et al.*, 2005).

In a study carried out by Robles *et al.* (2007) where there was increase in frequency of feeding from 1 to 4 times per day in beef cattle fed concentrate and straw to allow ad libitum consumption, there was no effect on intake, meal frequency or size, mean ruminal pH or fermentation profile as daily averages. However, feed intake and chewing patterns were drastically changed by treatments. Eating rate and the amount of feed consumed (31%) during the 2 h after the first morning feeding decreased linearly with feeding frequency, which may indicate lower motivation for feeding. Twice daily feeding showed two similar peaks of feed intake after each feed delivery and delayed the second peak of feeding typically observed around sunset in heifers fed once daily. This led to 2 equal peaks in ruminal fluid pH before each feeding with the second one being 0.85 pH units greater at 12 h post-feeding compared with the once daily feeding. The second peak in ruminal pH was the result, in part, of a build-up of buffering capacity in the rumen from saliva production as animals spent more time ruminating during the 2 h previous to the second feed delivery while waiting for the feed. Ruminal fluid pH of heifers fed four times per day fell steadily from the first morning feeding until 12 hours post-feeding. Thus, frequency of feed delivery changes both feed intake and rumination patterns throughout the day, which promotes a better synchronization in time between acid production and neutralization by saliva or elimination by absorption and passage from the rumen (González *et al* 2012).

2.9.3 Consistency of feeding

‘Planned’ periods of time of feed deprivation each day can lead to, animals accidentally becoming hungry as a consequence of mistakes in the amount of feed delivered or poor bunk management such as delays in feed delivery. Furthermore, it is known that subordinate animals within a group have limited access to feed, particularly after feed delivery, because socially dominant animals have priority of access (DeVries *et al.,* 2004), resulting in ruminant ingesting large quantities of feed during a short time period once feed becomes available. However, experimental simulations of these situations do not support this hypothesis. González *et al.* (2009) delayed the time of feed delivery (with regard to the routine time) to some experimental animals while others in adjacent stalls received the feed as per the normal routine. Delaying feed delivery time was not detrimental to ruminal fluid pH because a stress response (increased cortisol) was triggered which led to reduced concentrate intake, eating rate, and size of first meal, and increased straw intake. Withholding feed or delaying feed delivery with respect to the routine time has also been used as a model to induce ruminal acidosis (Nagaraja and Titgemeyer, 2007). However, evidence suggests that ruminal acidosis might not be induced by short periods of delays in feed delivery time of individually housed animals. Erickson *et al.* (2003) used a model of acidosis challenge consisting of feeding 1.25 of the previous day's intake at 4 h later than the routine feeding time. The authors did not succeed in inducing ruminal acidosis and concluded that their intake challenge may not have been severe enough to cause acidosis symptoms or effects on the animal. Interestingly, meal size (2.8 vs. 3.5 ± 0.5 kg/meal) and eating rate (0.17 vs. 0.32 ± 0.17/h) were apparently reduced during the challenge compared with the pre-challenge phase, respectively, when animals were fed under a clean bunk management program. These results are in agreement with the report by González *et al.* (2009). Day-to-day variations in the amount of feed offered have also been considered as a risk factor of ruminal acidosis (Schwartzkopf-Genswein *et al.,* 2003 and Schwartzkopf-Genswein *et al.,* 2004). However,

some studies testing this hypothesis do not support this theory (Soto-Navarro *et al.,* 2000) or obtained contradictory results among trials (Cooper *et al.,* 1999). It is likely that some discrepancies are due to changes induced in feeding behavior and consequently from feeding management and feed intake.

2.9.4 Feeding Management Strategies

Feeding management includes changes to diet composition, increasing fiber content, and applying feed additives (González*et al.,* 2012)

Applying diet changes includes giving the animals a proper balanced diet, increasing the chewing activity, and increasing particle size and length of the component, which will in turn increase salivation production and ruminal pH (Galyean and Defoor*et al.,* 2003). Also, the kind of cereal was controled and avoid using, at the same time, cereals with fast rate of fermentation, thus also avoiding producing a quick drop in the ruminal pH (Castillo*et al.,* 2012). Therefore, it will be important to take into account not only the grain composition, but also the fermentation rate and grain processing. Rate and extent of starch digestion in the rumen are determined by intricate interrelations among several factors, including source of dietary starch, diet composition, amount of feed consumed per unit time, mechanical alterations (grain processing, chewing), chemical alterations (degree of hydration, gelatinization), and degree of adaptation of ruminal microbiota to the diet. However, almost all the adversities associated with feeding high-grain diets are the result of excessively rapid fermentation of starch. It follows that most feed additives, feed treatments, and management techniques designed to ameliorate these adversities focus on ways to slow the fermentation rate or neutralize the acids produced. Similarly, the main goal of research on grain-processing techniques has been to increase digestibility of grain starch yet to avoid too much of a good thing by making starch too readily available for microbial attack (Castillo*et al.,* 2012).

2.9.5 Supplementation with Ruminal Buffers

Another measure to prevent and control acidosis is to incorporate buffers, like sodium and potassium bicarbonate, or alkalinizing agents (sodium and potassium carbonate, magnesium oxide) in the diet, with different objectives, because buffers will be able to neutralize ruminal pH changes; while the alkalinizing agent will increase the ruminal pH. These agents have direct effect on rumen fluid pH through chemical changes in the rumen as they neutralize acidity through H+ sequestration and increase buffering capacity of ruminal fluid (Calsamiglia *et al.,* 2012). However some experiences have suggested that the potential benefits of controlling ruminal pH with buffers and alkalizers are limited, and they cannot prevent ruminal acidosis alone and this is consistent with the hypothesis by Calsamiglia *et al.,* (2012) that part of the effects observed is pH-independent and should be resolved using alternative feeding strategies

2.9.6 Organic Acids

In the list of feed additives authorized by EU legislation, organic acids fall in the technological group, and their use is currently allowed in all the livestock species. They may be considered safe substances because they produce no detectable abnormal residues in meat (Joaquin *et al.,* 2014). Organic acids that have been evaluated as feed additives are malic acid, fumaric acid, and aspartic acid. Malic acid and fumaric acid are four-carbon dicarboxylic acids found in biological tissues (e.g., plants) as intermediates of the citric acid cycle and are intermediates in the succinate-propionate pathway of ruminal bacteria, such as *Selenomonas ruminantium*, the main gram-negative ruminal bacteria that can account for more than 50% of the total viable bacteria within the rumen (Castillo *et al.,* 2004). Organic acid malate is main characteristics are: (1) stimulation of lactate utilization; (2) increase in

ruminal pH, concentrations of propionate, and total volatile fatty acids; (3) increased digestibility of dry matter (DM) and organic matter (OM); neutral detergent fibre (NDF) and hemicellulose; (4) decreased methane production; and (5) decrease in ruminal lactate concentration (Castillo*et al.,* 2012). However these properties show controversial results in different in vitro and in-vivo studies. The addition of the acid form to the ration could contribute to reducing buffer blood bases, attributable to the decreased rumen pH, in line with in vitro results (Martin and Streeter, 1995).

2.9.7 Plant Products

It is worth noting that plant bioactivities are still an underexplored area of research and in many cases, though biological activity has been observed, but the natural phytochemicals responsible for the activity have not been identified (Joaquin *et al.,* 2014).

In ruminant health, the focus has been on bioactive effect of plants on ruminal flora rather than on specific pathogenic bacteria. This is perhaps understandable, since many of the desirable effects of antibiotics used as growth stimulants act through modification of the ruminal microbe population (Rochfort*et al.,* 2008).

In relation to the plant products as feed additives, phytochemical can be classified considering different aspects. So, attending to biological derivation, formulation, chemical description, and purity, phytobiotics comprise a very wide range of substances, and four subclasses in animal feeding may be categorized into (1) herbs (product from flowering, nonwoody, and nonpersistent plants), (2) botanicals (entire or processed parts of a plant, e.g., root, leaves, and bark), (3) essential oils (hydrodistilled extracts of volatile plant compounds), and (4) oleoresins (extracts based on nonaqueous solvents) (Hashemi and Davoodi, 2011).Careful selection and combination of these additives may allow the manipulation of

rumen microbial fermentation. However, their efficacy requires determination of potential ruminal adaptation in long-term in vivo feeding conditions. Thus, results based on short-term in vitro fermentation studies with several plant extracts should be interpreted with caution (Joaquin *et al.*, 2014).

2.9.8 Probiotics

Probiotics, also known as Direct-fed microbials (DFM), are live, naturally occurring bacterial supplements that have been used to improve digestive function of livestock (Joaquin *et al.*, 2014).

Feeding bacterial DFM is based on the concept of providing positive postrumen effects on the animal by improving the population of beneficial gut microflora, being able to alter rumen fermentation in order to reduce the risk of ruminal acidosis (Mc Daniel, 2009). The main objective is to stimulate the growing of *Megasphaera elsdenii* (a gram-negative and large coccus which is probably the most important ruminal organism with regard to lactic acid fermentation and, therefore, has a central role in the prevention of ruminal lactic acid accumulation in grain-adapted animals). Bacteriae (*M. elsdenii* YE34 and *Butyrivibrio fibrisolvens* YE44) could be used to reduce the risk of ruminal acidosis. Lactate-consuming bacteria have also been proposed as DFM and have been used successfully to decrease concentrations of lactate and maintain ruminal pH. *Megasphaera elsdenii* may utilize lactate and prevent drastic pH drops caused by accumulation of lactate in the rumen when fed a highly fermentable diet (Seo*et al.*, 2010;Campbell, 2010). Also, Mc Daniel (2009) pointed out that the use of *M. elsdenii* as a probiotic can reduce lactate accumulation. Inoculating cattle with *M. elsdenii* could be effective in bolstering populations of lactic-utilizers bacteria. Dosing cattle with *M. elsdenii* prior to the introduction of a concentrate diet may successfully

prevent the accumulation of lactic acid and resultant acidosis. According to Desnoyers*et al*. (2009)Yeast (*Saccharomyces cerevisiae*, dried or live-active dry-) and fungi (*Aspergillus oryzae*) are potential alternatives to bacterial microbials, with different mode of action. In general, they facilitate beneficial changes in the rumen motility by their stimulation of growth of rumen protozoa In relation to yeasts, and especially for *S. cerevisiae*, the main effects on rumen fermentation be are stat: (1) increase in rumen pH (+0.03 on average), (2) increase in rumen volatile fatty acid concentration (+2.17 mM on average), with no influence on acetate-to-propionate ratio, (3) decrease in rumen lactic acid concentration (−0.9 mM on average), and (4) increase in total-tract organic matter digestibility (+0.8% on average) (Desnoyers*et al*., 2009). Research has indicated an increase in rumen pH or decreased pH depression when yeast culture is included in ruminant diets (Castillo*et al*., 2012). However, other studies have found no changes with it. In fact, several reports have shown that dietary composition influences the extent of pH alteration by yeast culture and that ingredients utilized to maintain pH could mask yeast culture's effects (Castillo*et al*., 2012).

Fungal DFM have been extensively used in ruminants for improving performance and normalizing rumen fermentation, increasing the ruminal bacterial activity and preventing the lactic acid production ((Mc Daniel, 2009; Seo*et al.,* 2010; Campbell, 2010; Desnoyers*et al*., 2009).

2.9.9 Immunization

Immunization against ruminal acidosis has been proposed as an alternative to avoid this process. Vaccination against *Streptococcus bovis* and *Lactobacillus spp.* was successful in maintaining greater rumen pH and decreasing L-lactate concentration (Gill*et al*., 2000). Similarly, preparations of polyclonal antibodies against *S. bovis* or *Fusobacterium*

necrophorum were successfully applied to calves, reducing rumen concentrations of target bacteria and increasing pH in steers fed high-grain diets (Calsamiglia *et al.,* 2012)

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Ethical Approval

Ethical approval was obtained from the Ethical Committee on Animal Use and Care of Ahmadu Bello University Zaria (ABUECAUC), with Approval No. ABUCAUC/2016/002

3.2 Location of Study

The study was carried out in the small ruminants pen of the Department of Veterinary Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University Zaria located within the sub humid zone of Nigeria, latitude 11^{o} 12'N, longitude 7^{o} 33' E and an altitude of 610 m above sea level (GPS, 2017).

3.3 Experimental Animals

3.3.1 Source of the Experimental Animals

Fifteen (15) Yankasa sheep (X males and Y females) were purchased from Division of Agricultural Colleges (DAC) Livestock Animal Farm, Ahmadu Bello University Zaria and transported to the Department of Veterinary Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria.

3.3.2 Housing of Experimental Animals

The sheep were kept in pens in the Department of Veterinary Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University Zaria. The pens were well ventilated

3.4 Acclimatization

The sheep were acclimatized after arrival for a period of three (3) weeks during which they were clinically observed. During this period, faecal and blood samples were obtained from all the sheep to check for gastrointestinal tract (GIT) and blood parasites at the Parasitology and Helminthology Laboratories, Department of Parasitology and Entomology Ahmadu Bello University Zaria and prophylactically dewormed with albendazole at the dose rate of 10 mg/kg orally once; treated against coccidiosis with sulfadimidine injectable at the dose rate of

100 mg/kg I.M for 3 days. An acaricide (Amitrax) was reconstituted at 1 m l/ 1litre and sprayed on the bodies of the sheep against ectoparacites, whilst Long Acting Oxytetracycline (T.L.A.) 20% was administered to all the sheep at the dose rate of 20 mg/kg i.m against any bacterial infections. During the 3rd week of the acclimatization, second faecal and blood samples were collected to again screen for GIT and blood parasites

3.5 Preparation of Experimental Feed

A hundred kilogram of white maize was obtained from Samaru-Zaria. Fifty kilogram (50 kg) of the maize was then crushed using Hammer Milling Machine with an average mesh size of 2 mm, and the remaining 50kg was left as whole maize grain. Thirty (30) boles of *Digitaria smutsii* hay were obtained from the Division of Agric. Colleges (DAC) Livestock Animal Farm, Ahmadu Bello University Zaria. The chemical composition (Proximate Analysis) of maize grain and *Digitaria smutsii* hay were analysed according to the method described by the Association of Official Analytical Chemists (A.O.A.C., 2005) (Table 3.2).

3.6 Admistration of Feed to theExperimental sheep

Before cannulation of the sheep they were fed with *Digitaria smutsii* hay without concentrate.

3.7 Rumen Cannulation of the Experimental Sheep

In the third week of acclimatization, the sheep were prepared for cannulation as follows:

i. Left paralumbar fossa was liberally shaved.

ii. 10mls of2 % Lignocaine was infiltrated around the surgical (shaved) site

iii. One (1) cm of vertical incision was made on the site cutting through the skin, the muscle fibres and into the peritoneal.

iv. A Catheter of size 22 FR (Foley Balloon Catheter-100% Silicone Coate, Hospibrrand®, China) was inserted into each sheep via the surgical site and appositional sutures were placed using nylon size 1 to anchor it into the peritoneal cavity (Plate 1).

3.8 Experimental Grouping of sheep fed whole or crushed maize and hay

The fifteen (15) cannulated sheep were divided ramdomly into three (3) groups (A.B.C) of five (5) sheep each (Table 3.1).

3.9 Experimental Administration of Feed to Cannulated Sheep: 24 hours (1 day) grain feeding and 6 days hay feeding.

The experimental sheep were fed with whole or crushed maize grain at 4% of their body weight for 24 hours and *Digitaria smutsii* hay were fed to them for 6 days weekly. In the first week of feeding Group A sheep were fed with 50% of whole maize grain and 50% of *Digitaria smutsii* hay Group B sheep were fed with 50% of crushed maize grain and 50% of *Digitaria smutsii* hay and group C sheep were fed with *Digitaria smutsii* hay only. In week 2, Group A sheep were fed with 75% of whole maize grain and 25% of *Digitaria smutsii* hay, Group B sheep were fed with 75% of crushed maize grain and 25% of *Digitaria smutsii* hay and group C sheep were fed with *Digitaria smutsii* hay only. In week 3, Group A sheep were fed with 100% of whole maize grain and 0% of *Digitaria smutsii* hay, Group B sheep were fed with 100% of crushed maize grain and 0% of *Digitaria smutsii* hay and group C sheep were fed with *Digitaria smutsii* hay only. CleanTap water was also provided *ad-libitum*to each feeding group.

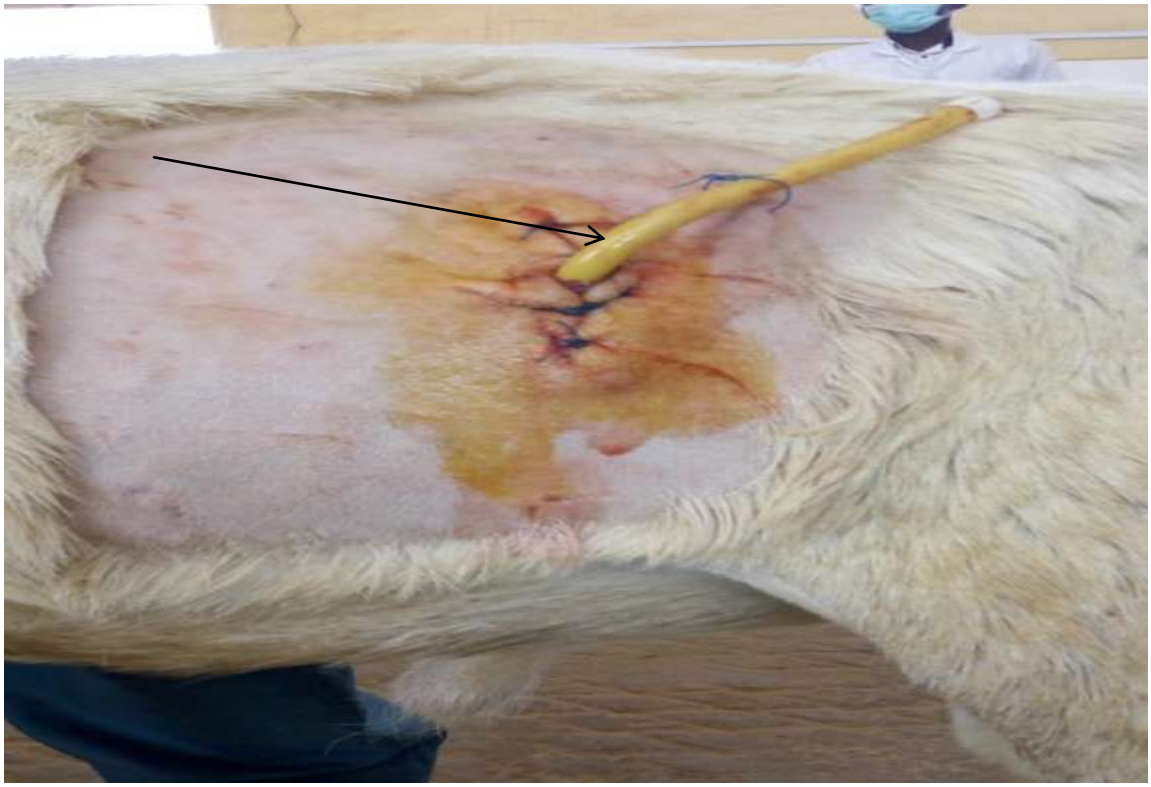

Plate I: Rumen cannula (arrowed) fixed into the sheep rumen at the surgically prepared area of the left paralumbar fossa

Table: 3.1 Average body weights of experimental sheep in Group A.B. and C. at the commencement of feeding trials

GROUP	NO. OF SHEEP	AVERAGE (MEAN) WEIGHT (Kg)	4% OF BODY AS FEED (Kg)
A	5	19.1 ± 1.0	3.82
B	5	18.6 ± 0.5	3.72
C	5	18.2 ± 0.6	3.64

Table: 3.2 Chemical compositions of maize grain and *Digitaria smutsii*hay

Parameters	***Digitaria smutsii hay* (%)**	**Maize grain (%)**
Dry matter (%)	93.10	92.22
Crude protein (%)	8.06	8.06
Crude fibre (%)	41.28	5.44
Ether extract (%)	8.39	-
Oil (%)	-	1.68
Ash (%)	9.95	0.82
Acid detergent fibre (%)	50.59	-
Neutral detergent fibre (%)	63.42	-
Nitrogen free extract (%)	-	84.00
Hemicellulose (%)	-	12.82
Metabolisable energy (M.E in MJ/KgDM)	-	10.52

N.B: *M.E value of feed ingredients and experimental feed is calculated using formula developed by Alderman and Cottrill (1985) as follow:

$ME\ (MJ/KgDM) = 11.78 + 0.00654CP + (0.000665EE)^2 – 0.0118A$

Where DM = Dry matter; CP = Crude protein; EE = Ether extract; CF = Crude Fibre; A= Ash

Table: 3.3. The feeding of whole or crushed maize and *Digitaria smutsiii* hay to the experimental groups in weeks1, 2 and 3

Graded Feed levels	Group	Treatment 1	Duration in hours (day)	Treatment 2	Duration (days)
50%	A	50% of 4% body weight as Whole maize grain(1)of*Digitaria smutsii* hay	24	50% of 4% body weight	6 days
	B	50% of 4% body weight as Crushed maize grain (1) of *Digitaria smutsii* hay	24	50% of 4% body weight	6 days
	C	4% body weight as *Digitaria smutsii* hayonly	all through for 1 week (7days)		
75%	A	75% of 4% body weight as Whole maize grain(1)of*Digitaria smutsii* hay	24	25% of 4% body weight	6 days
	B	75% of 4% body weight as Crushed maize grain (1) of *Digitaria smutsii* hay	24	25% of 4% body weight	6 days
	C	4% body weightas*Digitaria smutsii* hay only	all through for 1 week(7days)		
100%	A	100% of 4% body weight as Whole maize grain(1)of*Digitaria smutsii* hay	24	0% of 4% body weight	6 days
	B	100% of 4% body weight as Crushed maize grain (1) of *Digitaria smutsii* hay	24	0% of 4% body weight	6 days
	C	4% body weight as *Digitaria smutsii* hay only	all through for 1 week (7days)		

3.10 Measurement of Vital Parameters (Respiratory rate, Pulse rate and body/rectal Temperature)

Vital parameters were recorded 6 hourly during the experimental trials. Respiratory rates were obtained by counting the number of respiratory flank movements for one minute, as cycles per minute each experimental animal was restrained in a non-stressful way for measurements of pulse rate and rectal temperature. The pulse rate were obtained at the femoral artery, on the medial aspect of the thigh, as beats per minute and rectal temperatures were recorded using a standard digital thermometer, inserted about 3.5 cm into the rectum until alarm sound was heard indicating the stability of reading, usually lasting 1 to 2 minutes (Radostits *et al.,* 2010)

3.11 Collection of Rumen Fluid Sample

Ten (10mls) of rumen fluid were aspirated using a sterile 20 mls disposable syringe 2 hourly at 0, 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24 hours through the inserted rumen catheter after the introduction of whole and crushed maize grain and hay accordingly. Each rumen fluid aspirated (10mls) was emptied into a 50 mls beaker and pH determined and later transferred into 10 mls plastic bottle container and kept at 4^{o}C until needed. Acetic acids and lactic acid levels were determined from the rumen fluid; this procedure was repeated for all the cannulated treated animals giving a total of 13 samples per animal, 65 samples per group and 195 samples per week.

3.11.1 Evaluation of Rumen Fluid pH

The ten (10 mls) of collected rumen fluid was poured in to a beaker and pH meter (pH-lep®, Hanna Instruments USA) probe was inserted deep into the sample immediately after collection before exposure to air to avoid increases in pH. The pH values were read and recorded as the mean value as described by Ullah *et al.* (2013)**.**

3.12 Blood Sample Collection

Five (5 ml) of blood samples were collected 6 hourly and aseptically from the jugular vein of each sheep using 10 ml syringe into Ethylene Diamine Tetra Acetate (EDTA) containing bottle for haematological and blood pH analysis.

3.12.1 Determination of blood pH

The five (5) ml of blood collected was poured in to a beaker and pH meter (pH-lep®, Hanna Instruments USA) probe was inserted deep into the sample immediately after collection. The pH values were read and recorded as the mean value (Radostits *et al.*, 2009).

3.13.2 Determination of Haemogram

The collected blood sample was taken to the Clinical Pathology Laboratory in the Department of Veterinary Pathology and the haemogram was evaluated using standard procedure as described by Schalm *et al.* (1975).

3.13 Determination of Acetic Acid level by a Spectrophotometric Method

The spectrophotometric procedure is based on the Montgomery method (Montgomery*et al*., 1962). In this study the procedure was modified as follows: aqueous sample (0.5 mL) was taken into a dry test-tube, and then after the addition of 1.5 mL ethylene glycol reagent and 0.2 mL of 19.5N sulphuric acid were added and the mixture was heated for 3 min in a boiling bath and thecontent of the test-tube wasimmediately cooled in cold bath. After cooling, 0.5 mL of 10% hydroxylamine hydrochloride solution, 2 mL of 4.5 N NaOH solution and 10 mL of 10% ferric chloride solution was added. The absorbance was measured at 495 nm by spectrophotometer. The standard solutions of acetic acid (1200 mg/L CH3COOH) wasdiluted

with deionized water to obtain appropriate concentrations at the range of 10 to 1200 mg/L. Deionized water was obtained in-house by treating tap water with a carbon filter, reversed osmosis, a mixed bed of ion exchangers and a 0.45 μm filter as reported by Siedlecka *et al.* (2008).

The effect of the alkalinity and phosphate ions on the spectrophotometric response for the acetic acid was also investigated.

The application of ion exchange (Amberlite IRA 410) for VFA separation has been also investigated. The different volume of feed solution was subjected to the top of the column and flow down. VFAs were eluted using different volume of saturated solution of NaCl at the flow rate 0.4 mL/min. The analytes were collected and determined by spectrophotometric method as reported by Siedlecka *et al.* (2008).

3.14 Determination of Lactic Acid level by a Spectrophotometric Method

All chemicals used were of analytical quality.

Oxidizing reagent: A solution was prepared containing ceric ammonium nitrate (0.033 M), nitric acid (0.333 M), ammonium sulphate (0.170 M) perchloric acid (0.800 M) and copper sulfate (0.533 mM)

Alkaline reagent: A solution was prepared containing trisodium citrate (9.200 M), disodium tetraborate (0.115 M), disodium hydrogen phosphate (0.232 M) and sodium hydroxide (0.174 M).

Standard procedure: oxidizing reagent (1.5 ml) was added to the lactate sample (0.1 to 1.0 ml) in a 15 ml centrifuge tube. A lactate standard and black (distilled water were treated similarly. After an oxidation period (15 min) at room temperature, 0.6 M nitrate solution (0.2 ml) was added followed by alkalinizing reagent (5 ml) and 5mM oxalyldehydrzide (ODH) solution (1ml). The samples were mixed thoroughly and centrifuged (100 g, 1min). After colour development (30 min) at room temperature, the absorbance of the supernatant solution

were measured (610nm) against the blank in a Spectronic 1001 spectrophotometer with a peristaltic pump accessory (Bauch and Lomb U.S.A.). Concentrations were calculated from the lactate standard value (David and Johan, 1991).

3.15 Determination of rumen microbial plate count

The rumen microbial count was determined using the method described by Cheong (2016). The total aerobic colony plate count was carried out on the homogenized rumen samples by taking 0.1 ml into 9.9 ml normal saline diluent using a sterile pipette. Then 0.1 ml was taken from 10^3 dilutions to nutrient ager and then spread to cover the entire surface using glass bent rod, and thereafter, incubated at 37^oC for 24 hours. The colonies were identified and then counted using colony counter machine after the incubation and expressed as colony forming units per ml (CFU/ml) of each of the samples analysed.

3.16 Data Analysis

Data obtained were presented in forms of tables and figures, data generated from vital parameters, rumen fluid, blood pH and haemogram were expressed as means (± Standard Error of Mean, SEM). Group means were analysed with One-way Analysis of Variance (ANOVA), using Statistical Package for Social Scientists (SSPS, version 20.0, IBM, USA 2011). Values of $P < 0.05$ were considered statistically significant.

CHAPTER FOUR

4.0 RESULTS

4.1 Vital Parameters

4.1.1 Respiratory rate

Mean Respiratory rate slightly increased from 20.60 ± 0.40 to 22.4 ± 0.68 at 12^{th} hour after the introduction of the experimental feed in group A (fed whole maize grain) and from 21.0 ± 0.95 to 23.0 ± 0.55 in group B (fed crushed maize grain) and significantly increased ($p< 0.05$) from 22.4 ± 0.68 to 25.0 ± 0.89 at 24^{th} hour in group A and from 23.0 ± 0.55 to 25.8 ± 0.73 in group B in the first weekof the experimental period. The respiratory rate slightly increased from 21.00 ± 0.00 at 12^{th} hour in group B and significantly increases ($p< 0.05$) from 21.20 ± 0.20 to 24.80 ± 0.80 at 24^{th} hour in group A and from 21.00 ± 0.00 to 25.20 ± 0.80 in group B in the second weekof the experimental period. However, there were no-significant increases in the respiratory rate in both group A and B in the third week of the experimental period (Figure 4.1, 4.2, 4.3,appendices I, II and III).

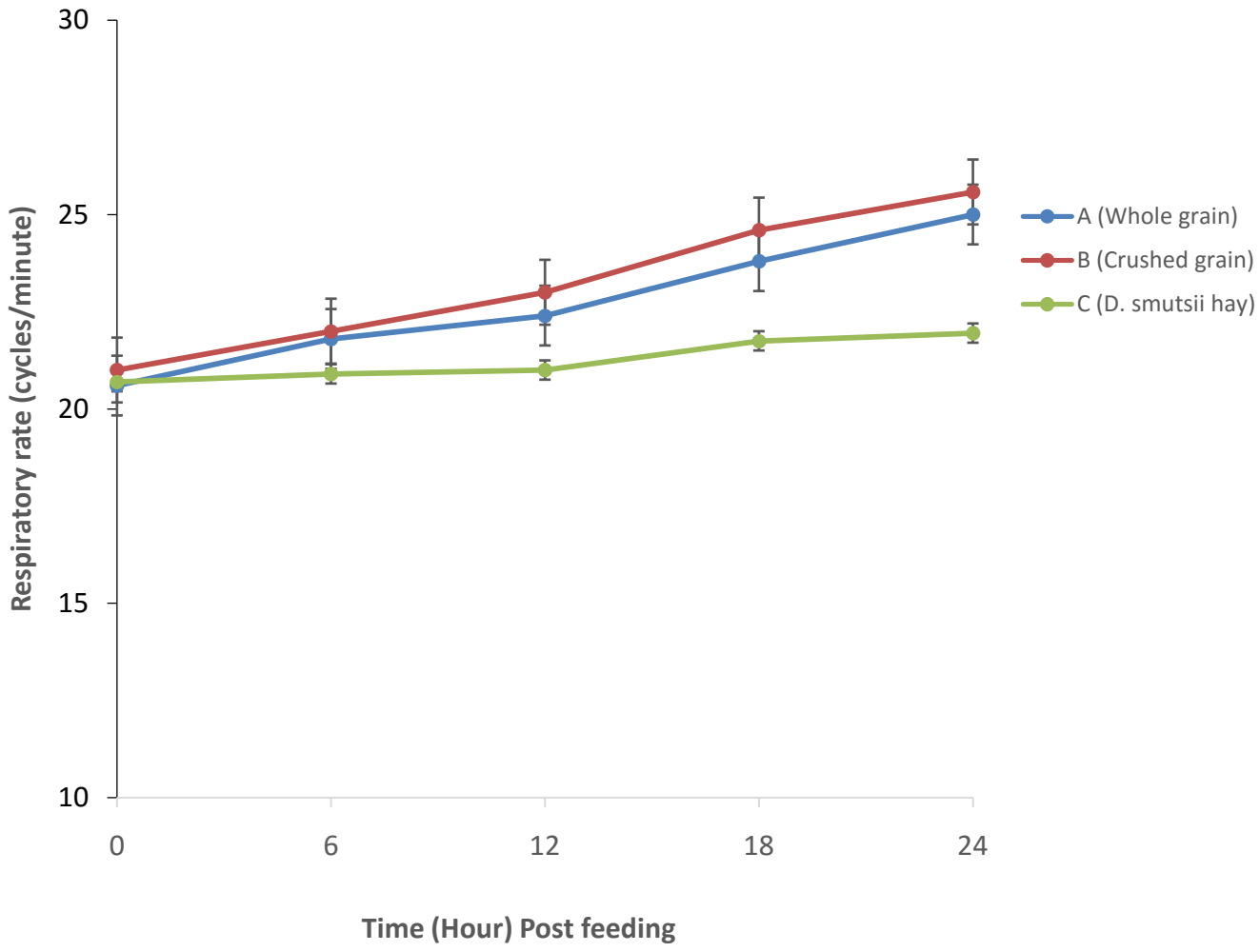

Figure 4.1: Effect of feeding whole or crushed maize grain on Mean values of respiratory rate (cycle/minute) of Yankasa sheep for 24 hrs in the first week of feeding trial

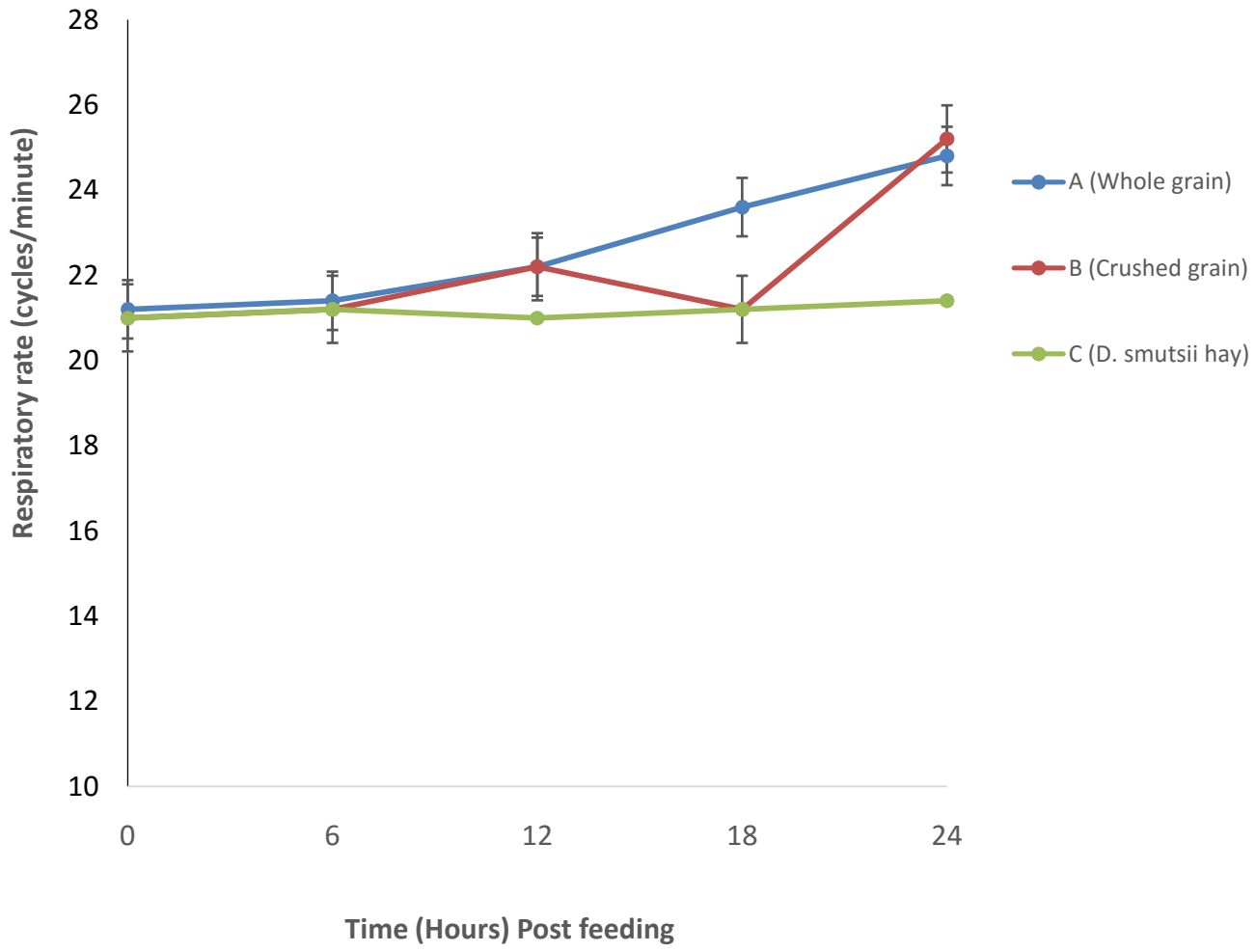

Figure 4.2: Effect of feeding whole or crushed maize grain on Mean values of respiratory rate (cycle/minute) of Yankasa sheep for 24 hrs in the second week of feeding trial

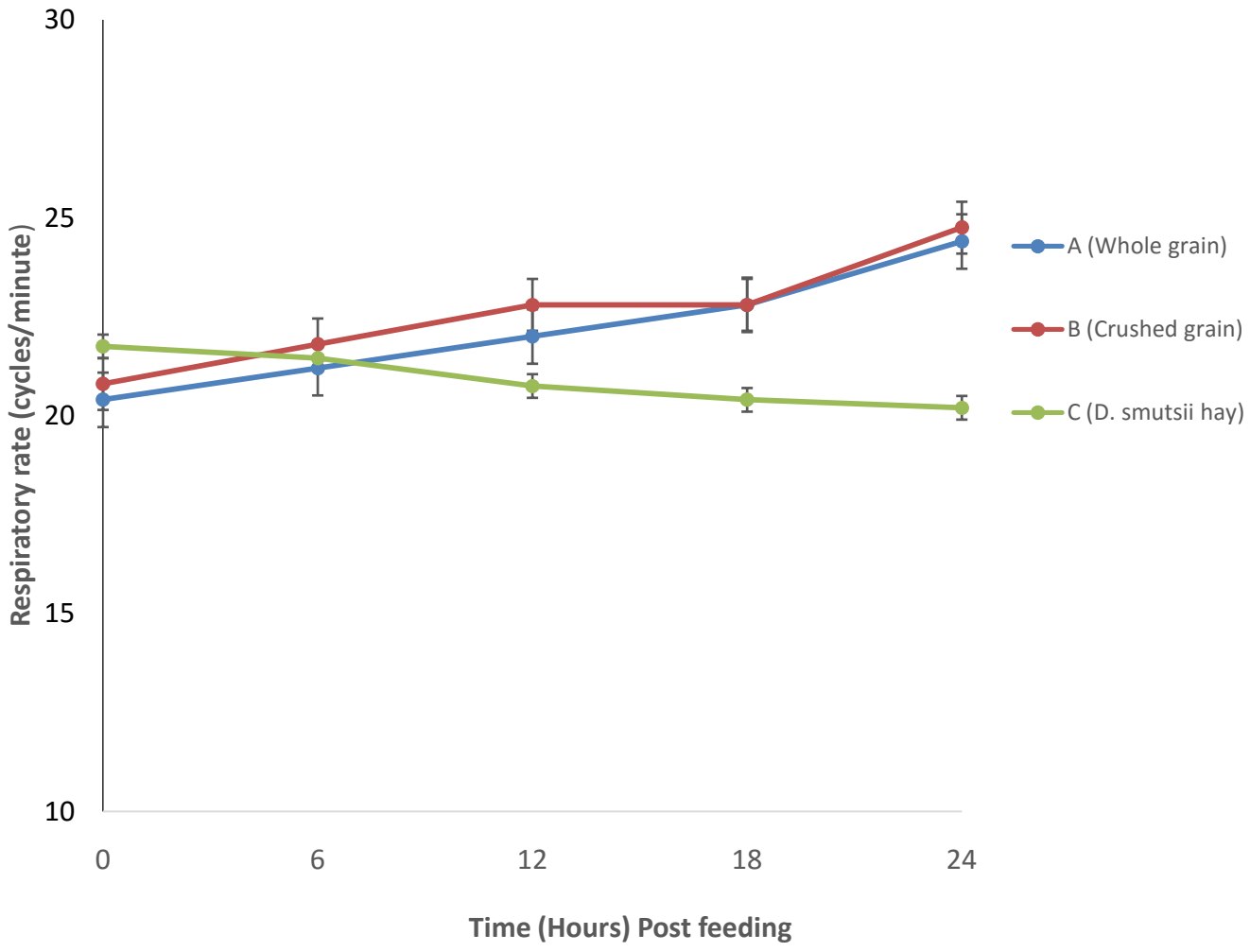

Figure 4.3: Effect of feeding whole or crushed maize grain on Mean values of respiratory rate (cycle/minute) of Yankasa sheep for 24 hrs in the third week of feeding trial

4.1.2 Pulse rate

Pulse rate slightly increased from 76.0 ± 1.04 to 80.8 ± 0.97 at 12th hour after the introduction of the experimental feed in group A (fed whole maize grain) and increased significantly ($p < 0.05$) from 79.8 ± 2.92 to 87.8 ± 2.58 at 24thhours post feeding in in group B at the first week but there were no–significant increase in both groups A and B in the second and third week of the experimental period (Figure 4.4, 4.5, 4.6, appendices I, II and III)

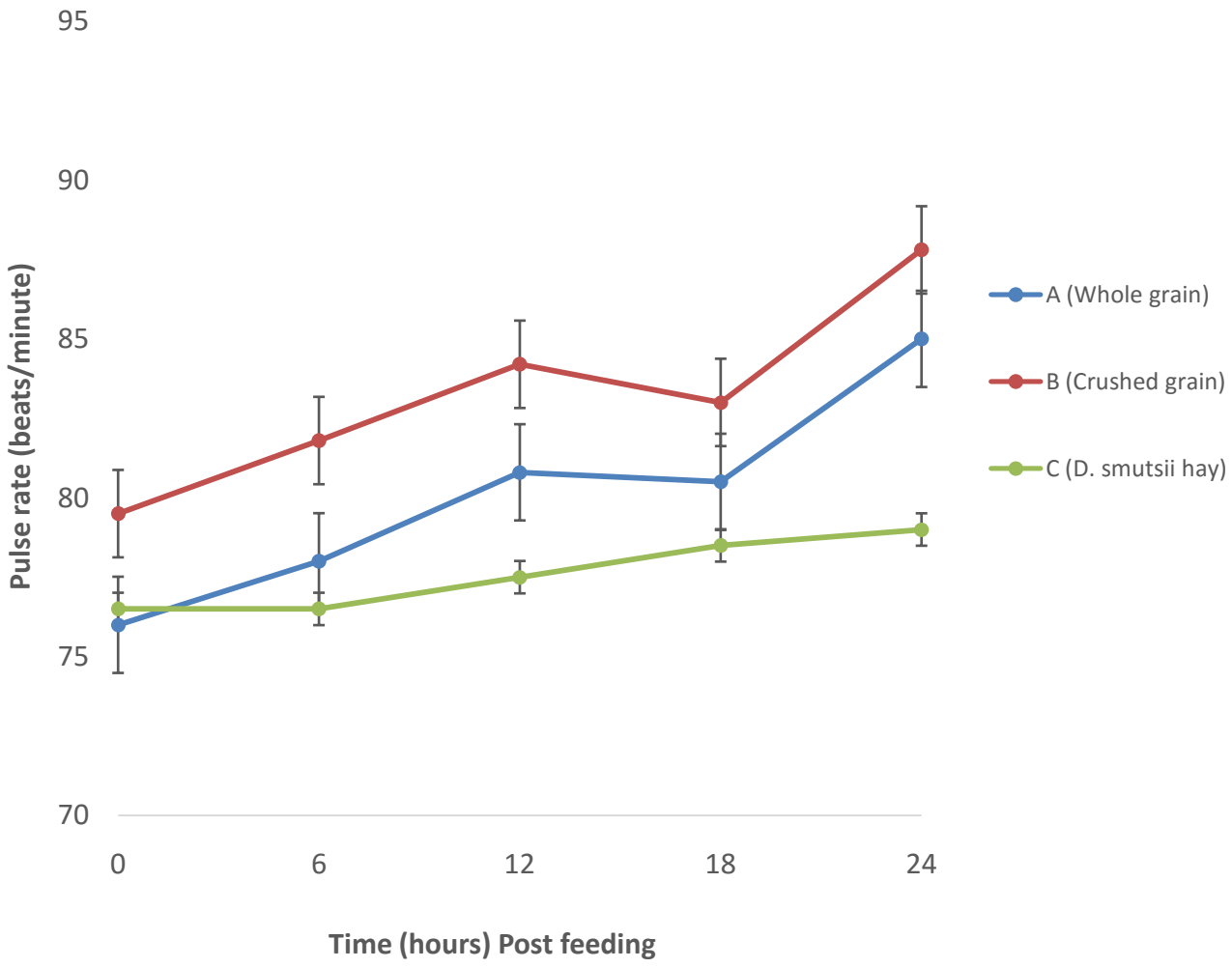

Figure 4.4: Effect of feeding whole or crushed maize grain on Mean values of pulse rate (beats/minute) of Yankasa sheep for 24 hrs in the first week of feeding trial.

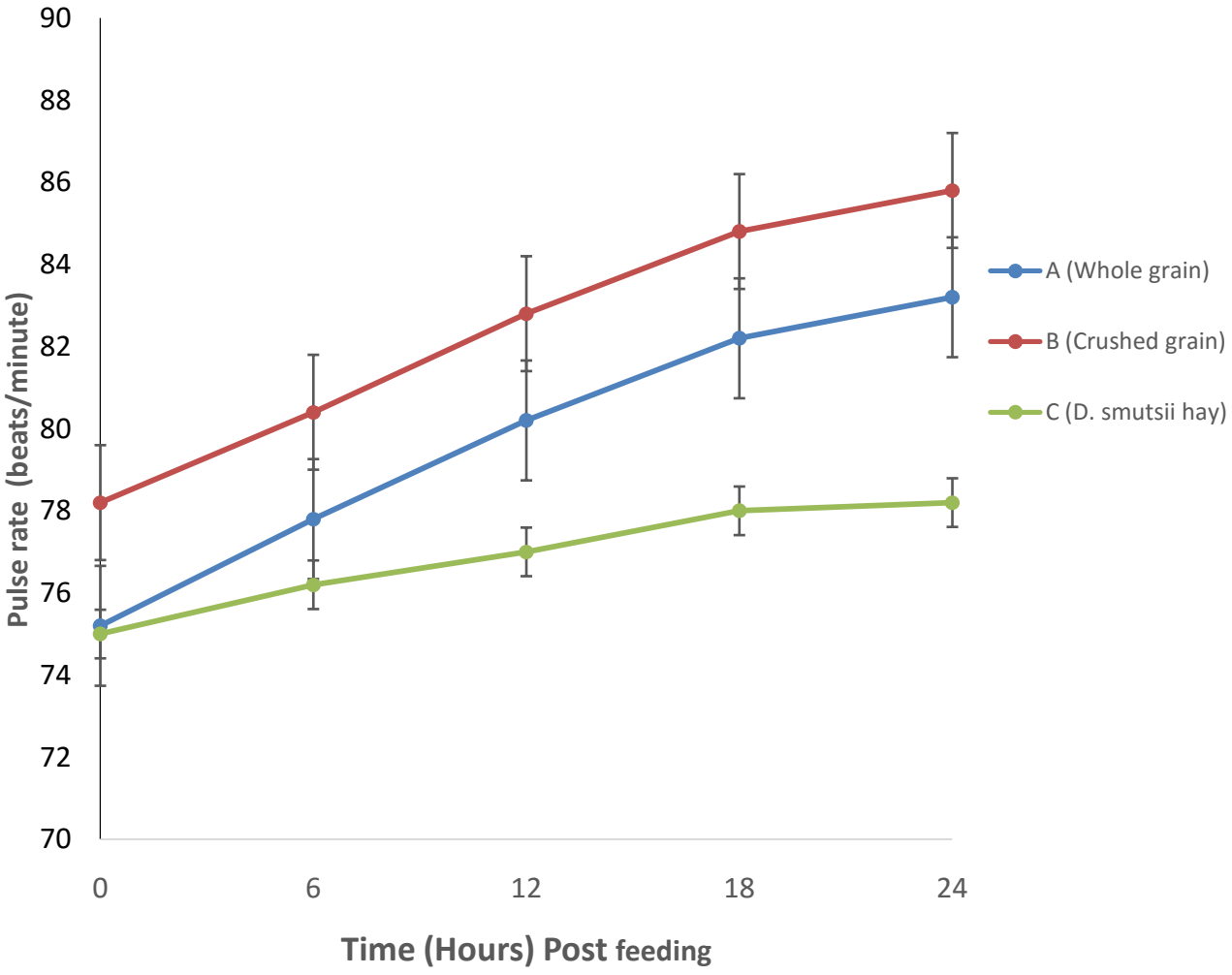

Figure 4.5: Effect of feeding whole or crushed maize grain on Mean values of pulse rate (beats/minute) of Yankasa sheep for 24 hrs in the second week of feeding trial.

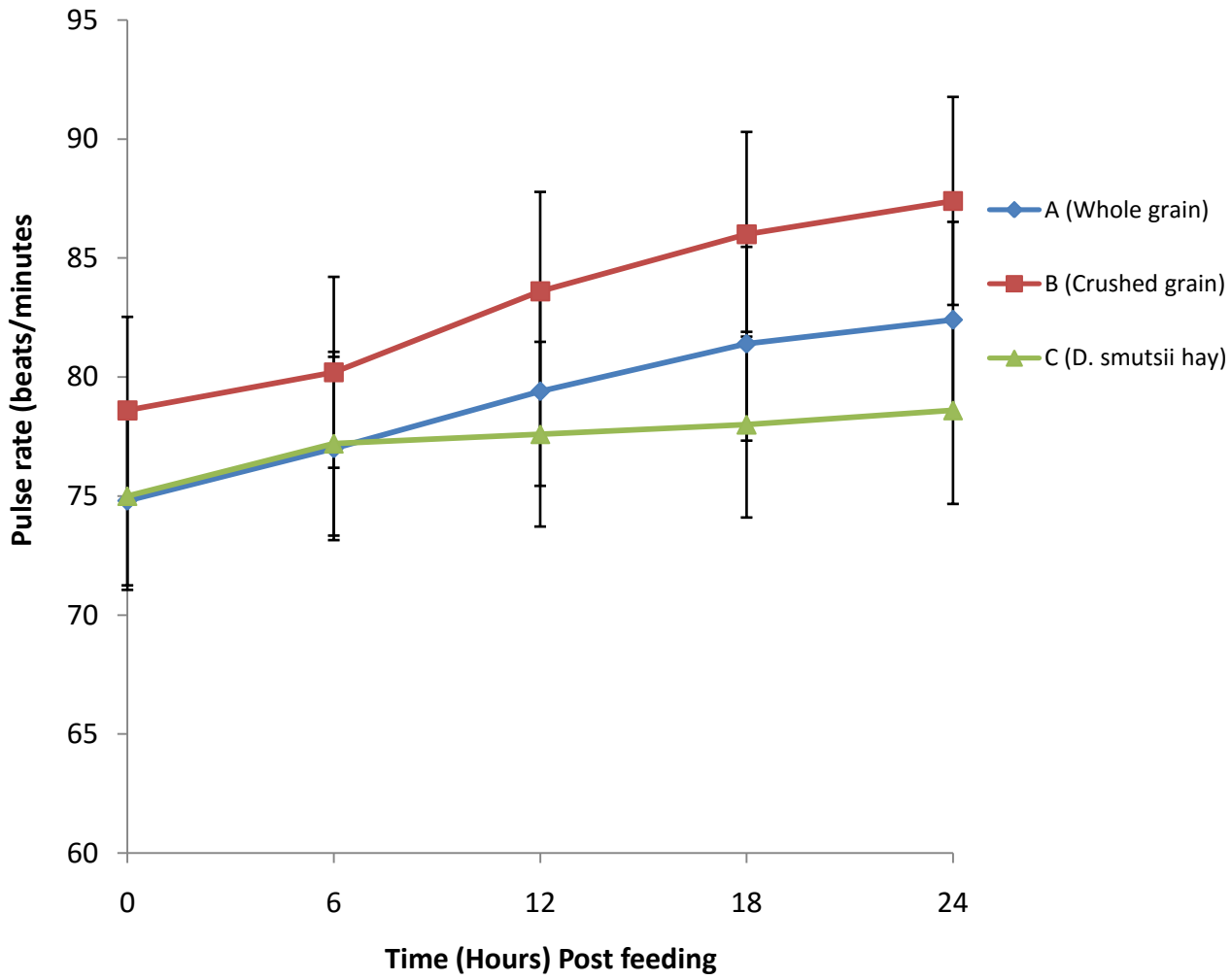

Figure 4.6: Effect of feeding whole or crushed maize grain on Mean values of pulse rate (beats/minute) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.1.3 Rectal temperature

The mean rectal temperature increased non- significantly ($p > 0.05$) from 39.04 ± 0.20 to 39.70 ± 0.20 with the introduction of grain preparations at 6^{th} hours of the experimental period in group A at the first and from 39.22 ± 0.20 to 40.30 ± 0.12 in group B at the third weeks of the experimental period and decreased significantly ($p < 0.05$) from 39.12 ± 0.20 to 36.98 ± 1.72 at the 12^{th} hours in group B (fed crushed maize) at the second weeks of the experimental period (Figure 4.7,4.8,4.9, appendices I, II and III)

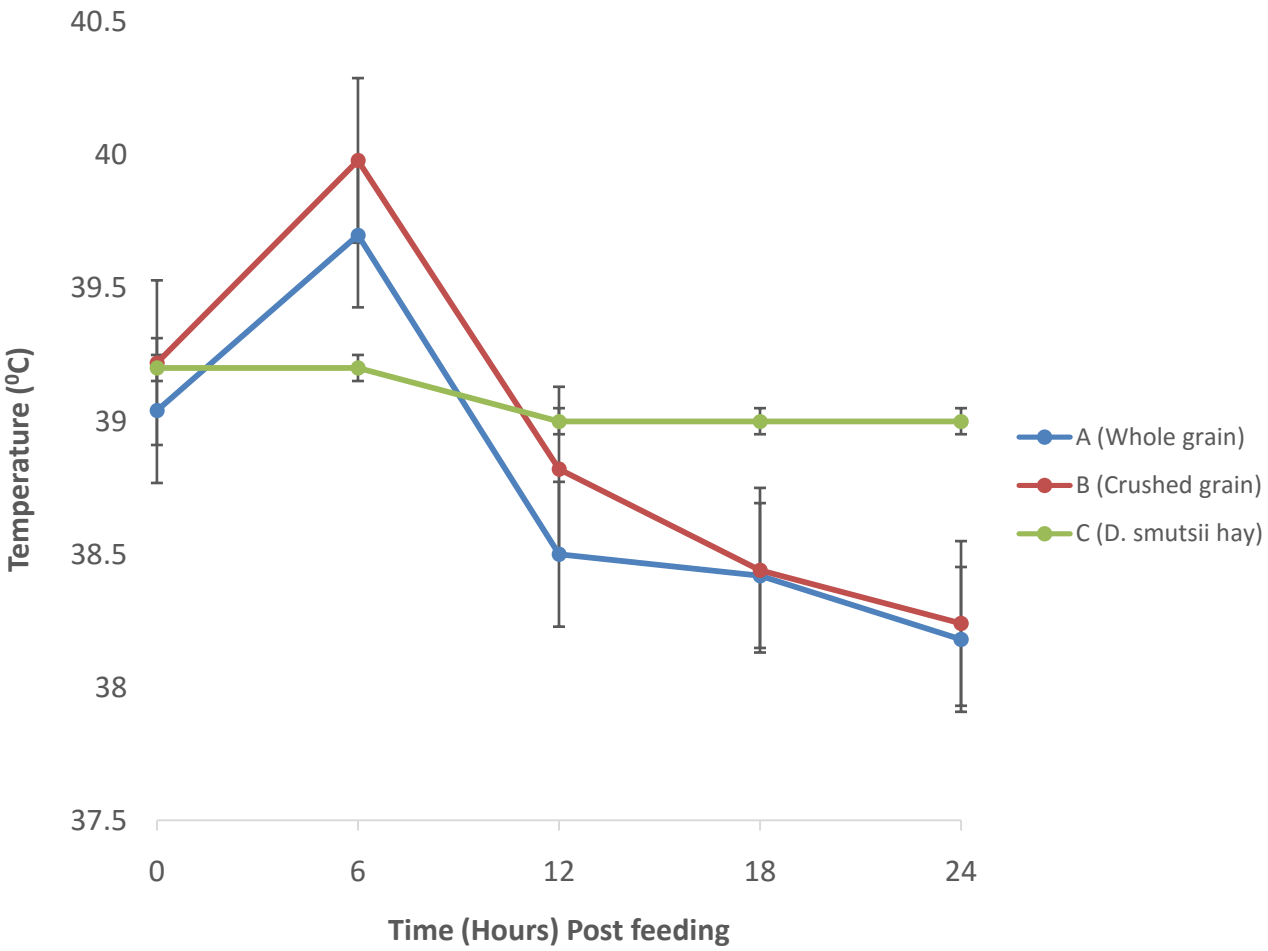

Figure 4.7: Effect of feeding whole or crushed maize grain on rectal temperature (^{0}C) of Yankasa sheep for 24 hrs in the first week of feeding trial

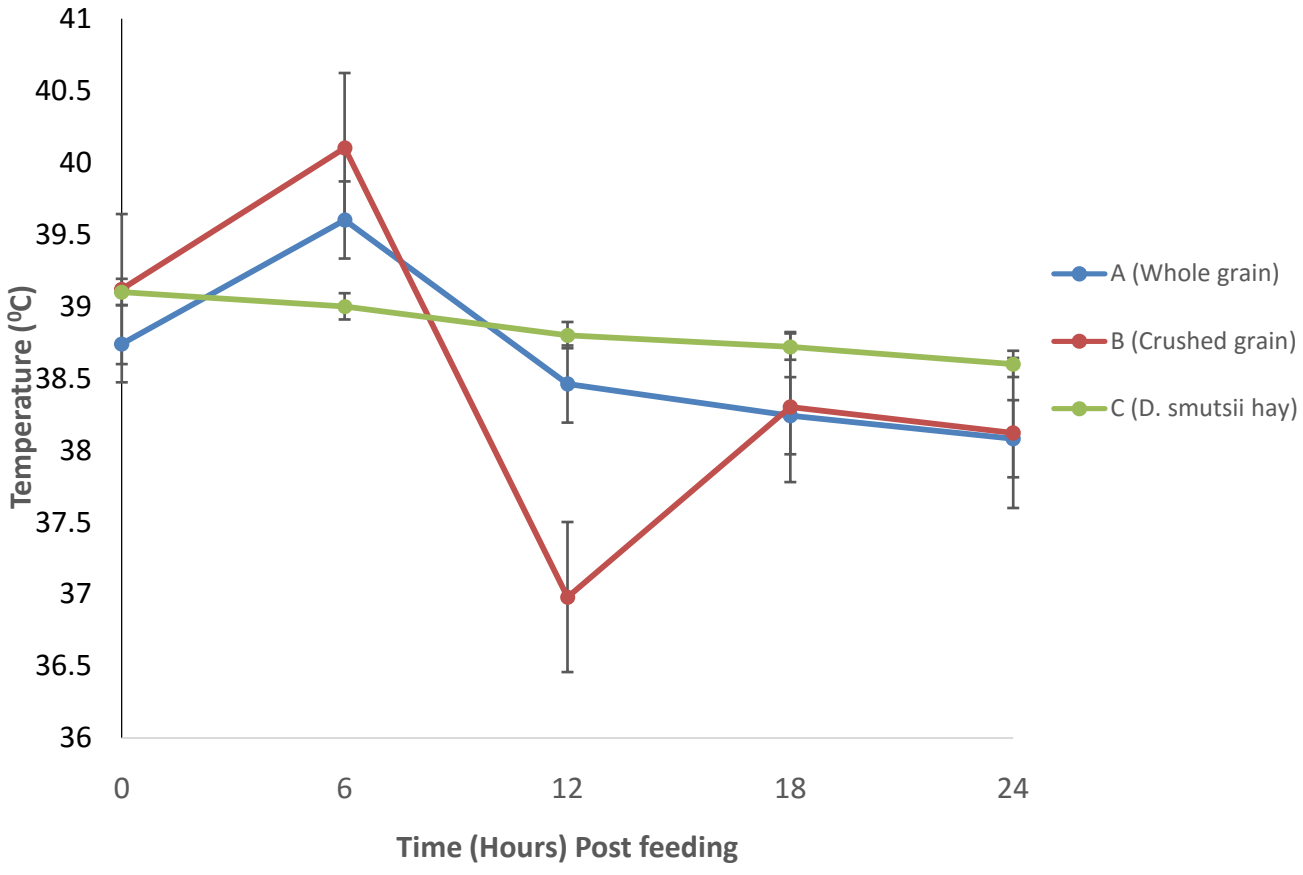

Figure 4.8: Effect of feeding whole or crushed maize grain on Mean values of rectal temperature (^{0}C) of Yankasa sheep for 24 hrs in the second week of feeding trial

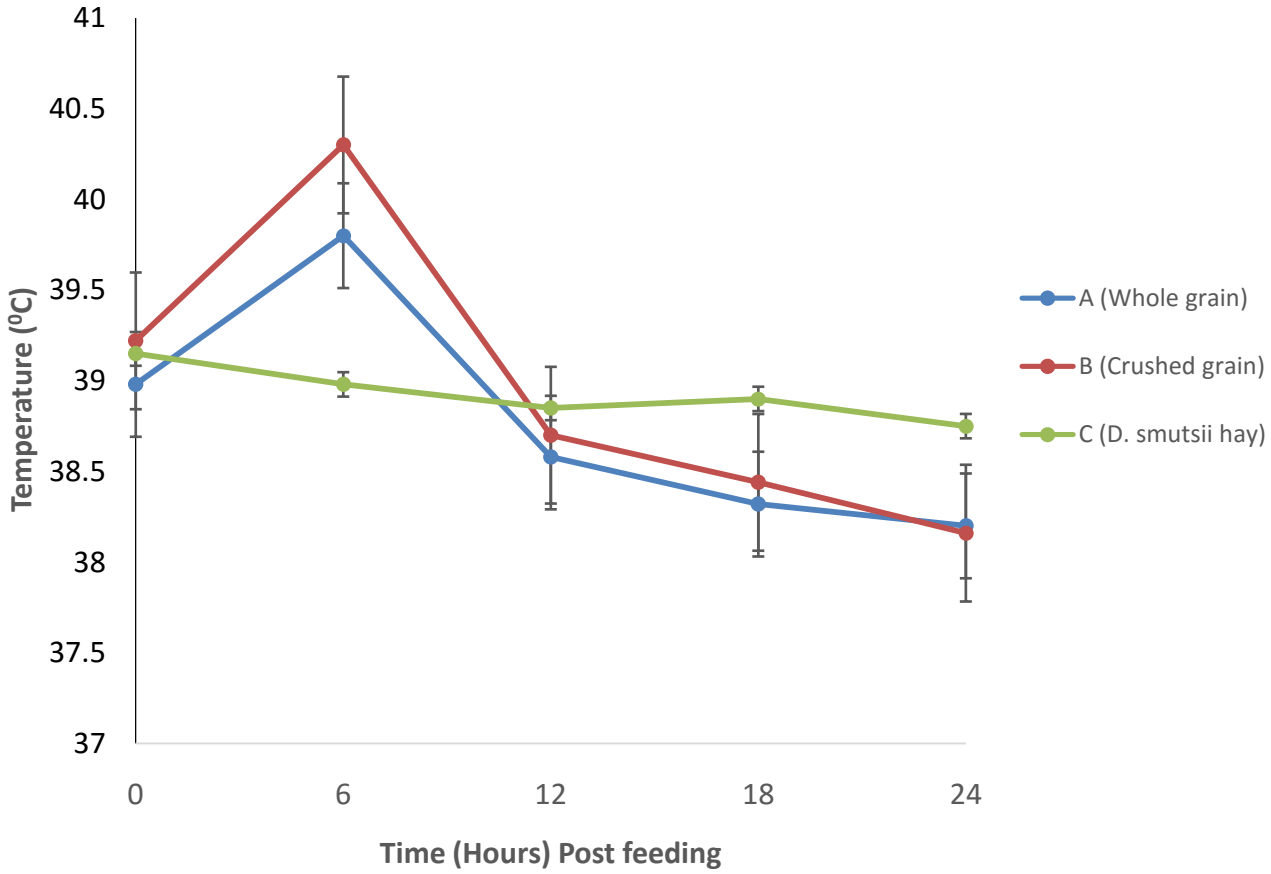

Figure 4.9: Effect of feeding whole or crushed maize grain on Mean values of rectal temperature (^{0}C) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.2 Clinical signs observed during the experiment

The clinical signs exhibited by the experimental sheep were noted. Loss of appetite in some animals, depression, diarrhoea, bruxism (teeth grinding) salivation and finally the animals started dying 6 days after the last sample collection of the experiment.

4.3 Rumen pH

The rumen pH decreased no-significantly ($p > 0.05$) in group A (fed whole maize grain) from 6^{th} to 10^{th} hour then significantly decreased from 5.50 ± 0.03 at 12^{th} hour to 5.38 ± 0.02 at 24^{th} hour of the experimental period. Rumen pH decreased significantly ($p<0.05$) in group B (fed crushed maize grain) from 5.59 ± 0.15 at 4^{rd} hour to 5.28 ± 0.07 at 24^{th} hour in the first week of the experimental period. Rumen pH decreased non-significantly ($p > 0.05$) in group A (fed whole maize grain) from 8^{th} and 10^{th} hours then significantly decreased ($p<0.05$) from 5.66 ± 0.02 at 12^{th} hour to 5.12 ± 0.12 at 24^{th} hour of the experimental period. There were no-significantly decreased ($p>0.05$) of rumen pH in group B (fed crushed maize grain) from $6^{rd}$to

10^{th} hour, but significantly decreased ($p<0.05$) from 5.46 ± 0.04 at 12^{th}hour to 5.38 ± 0.10 at 18^{th} hour and begin to increased slightly from 5.60 ± 0.08 at 20^{th} hour to 5.60 ± 0.11at 24^{th} hour in the second week of the experimental period. Rumen pH decreased non-significantly ($p> 0.05$) in group A (fed whole maize grain) from 16^{th} to 20^{th} hour then significantly decreased from 5.50 ± 0.05 at 22^{nd} hour to 5.42 ± 0.05 at 24^{rd} hour of the experimental period. There weresignificantlydecreased ($p<0.05$) in rumen pH in group B (fed crushed maize grain) from 14^{rd} to 24^{rd} hour in the third week of the experimental period (Figure 4.10, 4.11, 4.12 and appendix IV)

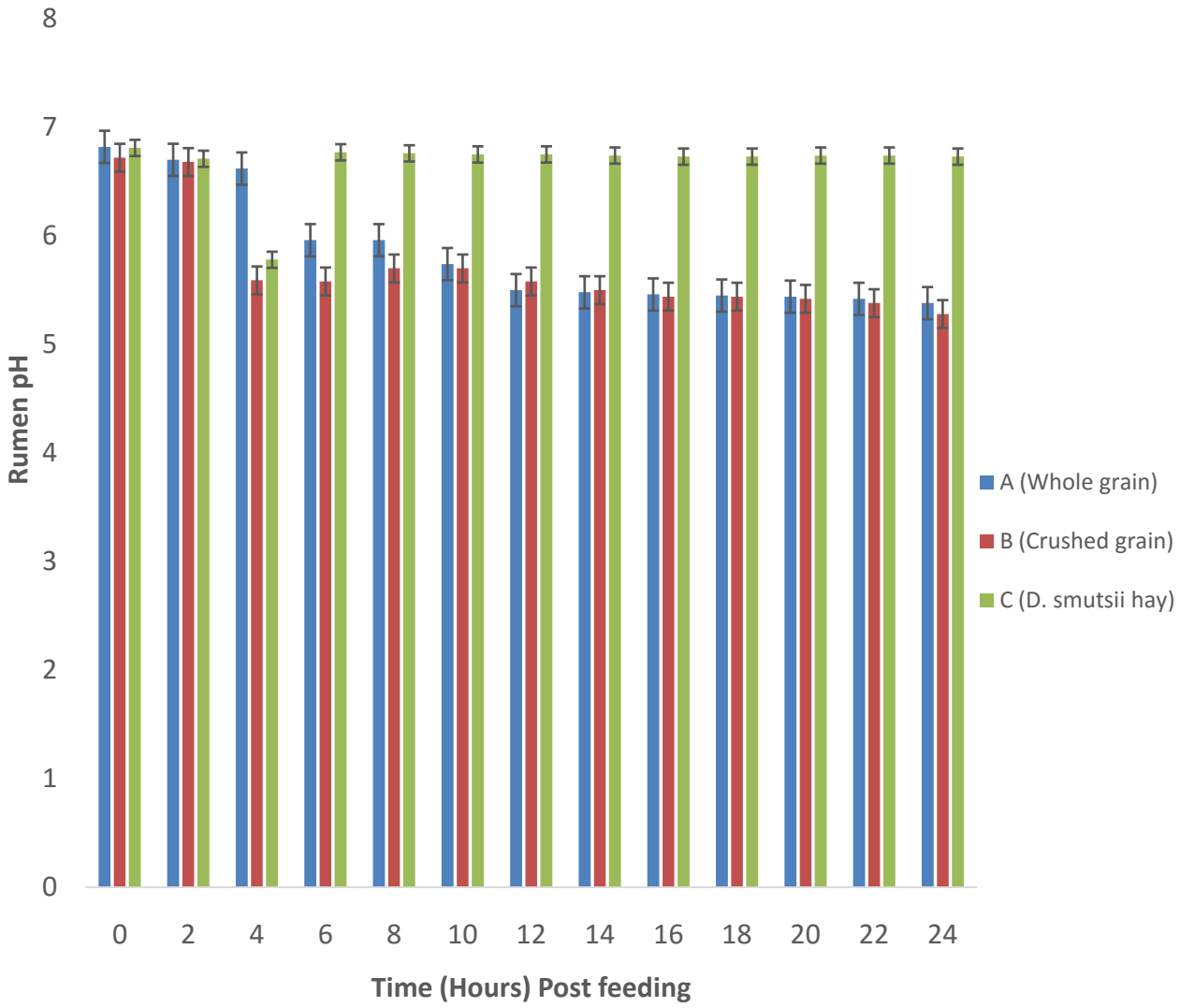

Figure 4.10: Effect of feeding whole or crushed maize grain on Mean values of rumen pH of Yankasa sheep for 24 hrs in the first week of feeding trial

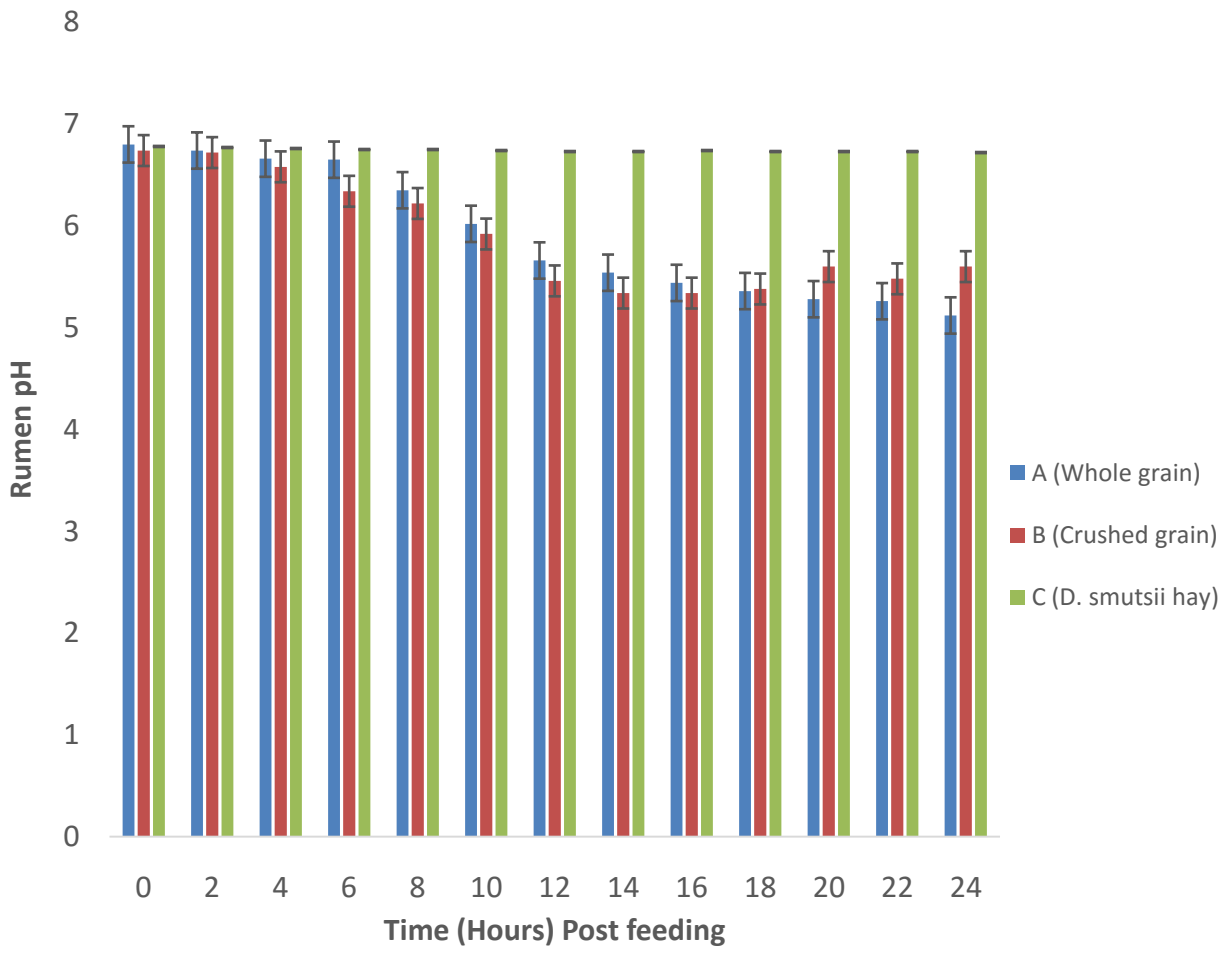

Figure 4.11: Effect of feeding whole or crushed maize grain on Mean values of rumen pH of Yankasa sheep for 24 hrs in the second week of feeding trial

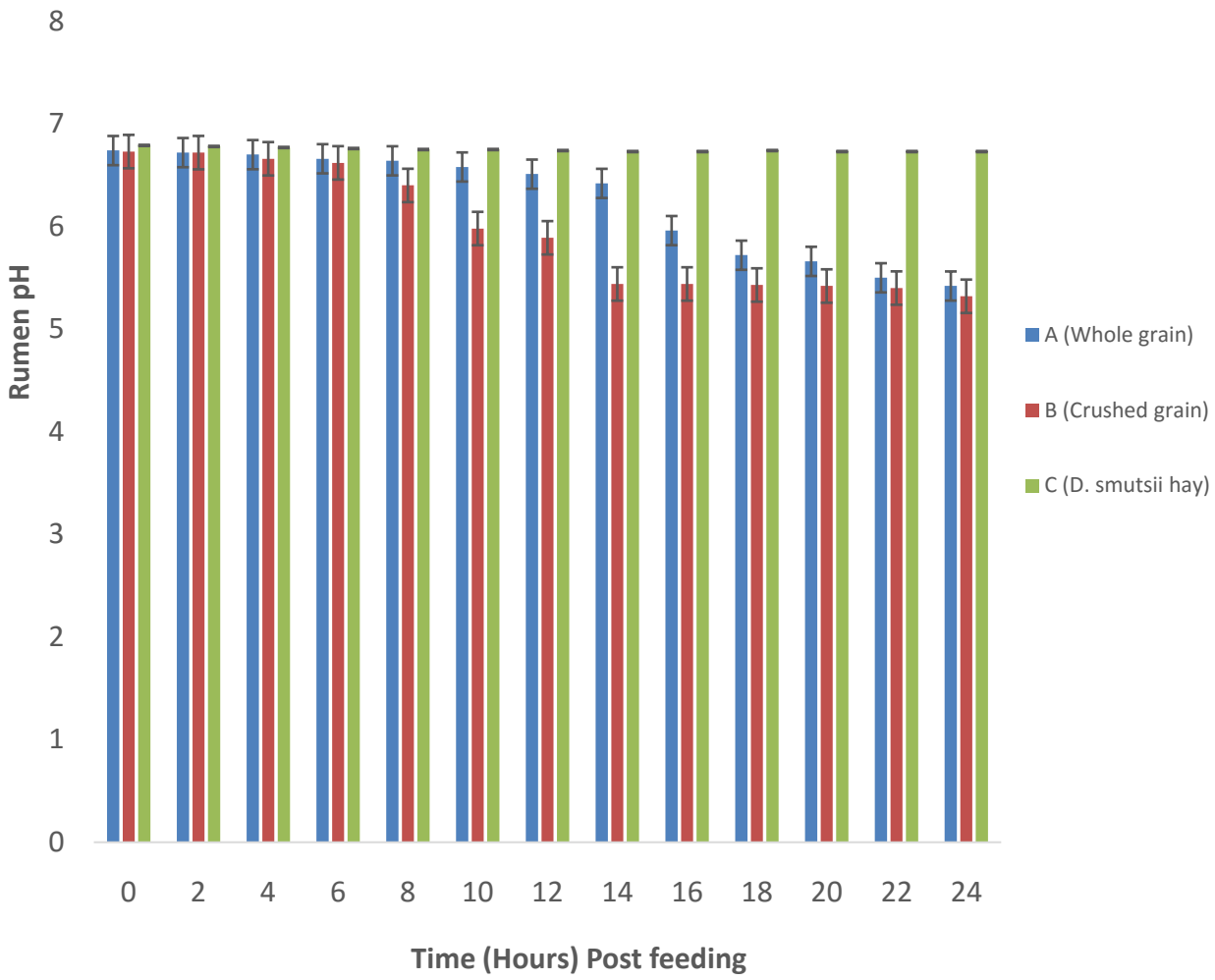

Figure 4.12: Effect of feeding whole or crushed maize grain on Mean values of rumen pH of Yankasa sheep for 24 hrs in the third week of feeding trial

4.4 Blood pH

The blood pH decreases non-significantly ($p > 0.05$) in group A (fed whole maize grain) from 6^{th} to 12^{th} hour then significantly decreased from 7.46 ± 0.04 to 7.04 ± 0.02 at 24^{th} hour of the experimental period. Blood pH decreased significantly ($p<0.05$) in group B (fed crushed maize grain) from 7.45 ± 0.04 to 7.16 ± 0.02 at 18^{th} hour, non-significantly decreased ($p > 0.05$) were observed 12^{th} and 24^{th} hours in the first week of the experimental period. A non-significantly decreased ($p > 0.05$) was observed in blood pH at 6^{th} and 12^{th} hours of group A, then a significantly decreased ($p<0.05$) were observed from 7.44 ± 0.02 to 7.12 ± 0.02 at 18^{th} and 7.10 ± 0.00 at 24^{th} hours of the experimental period. Blood pH decreased significantly ($p<0.05$) in group B (fed crushed maize grain) from 7.12 ± 0.02 at 12^{th} hour to 7.10 ± 0.00 at 18^{th} hour in the second week of the experimental period. There were non-significantly decreased ($p > 0.05$) at 6^{th}, 12^{th} and 18^{th} hours in group A and B after the introduction of the experimental feed, but a significantly increased ($p<0.05$) from 7.38 ± 0.02 to 7.71 ± 0.04 at 24^{th} hour in group A and also a significant decreased ($p<0.05$) was also observed from 7.42 ± 0.02 to 7.06 ± 0.02 at 24^{th} hours in group B in the third week of the experimental period (Figures 4.13, 4.14 and 4.15 respectively and appendix V respectively).

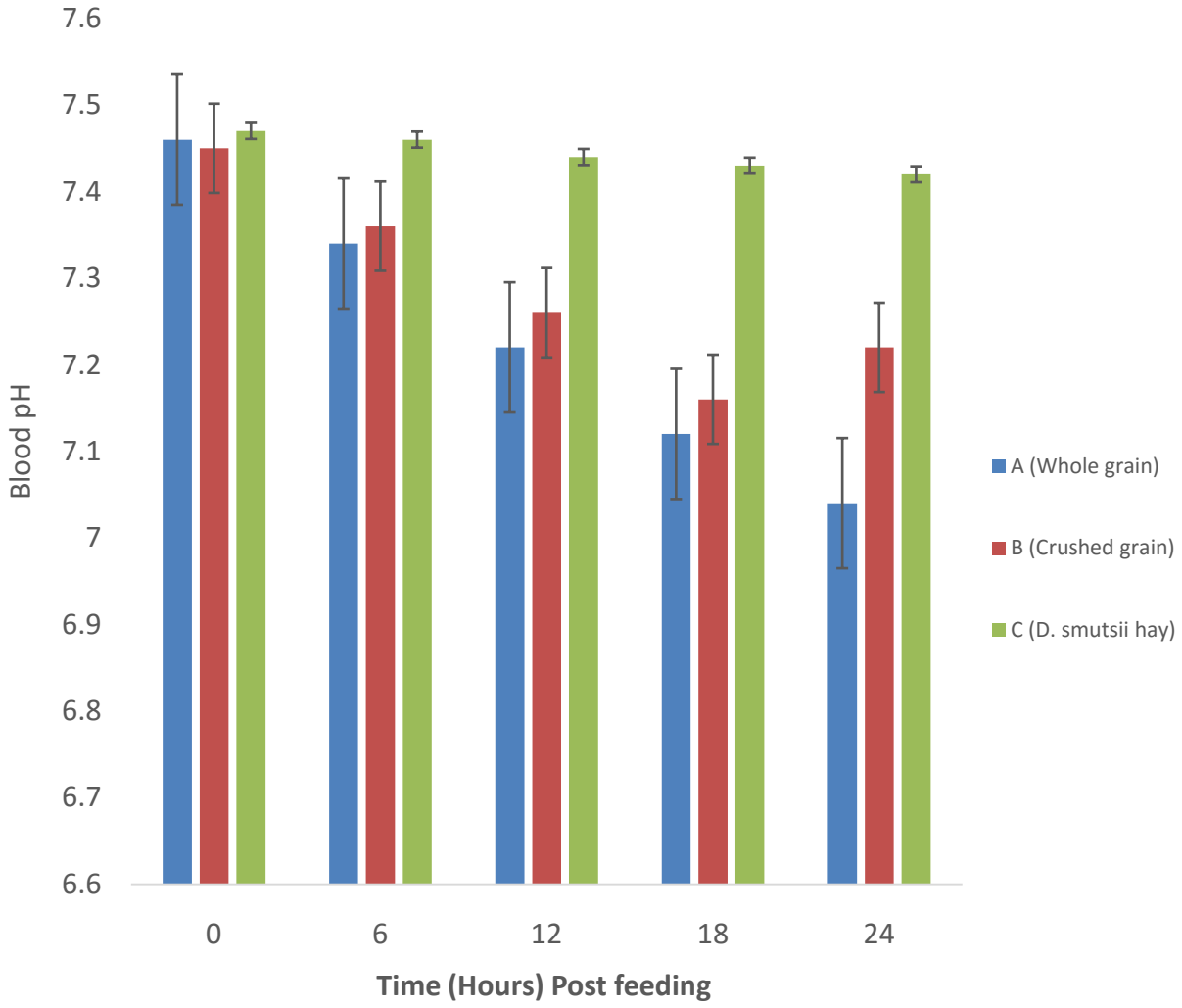

Figure 4.13: Effect of feeding whole or crushed maize grain on Mean values of blood pH of Yankasa sheep for 24 hrs in the first week of feeding trial

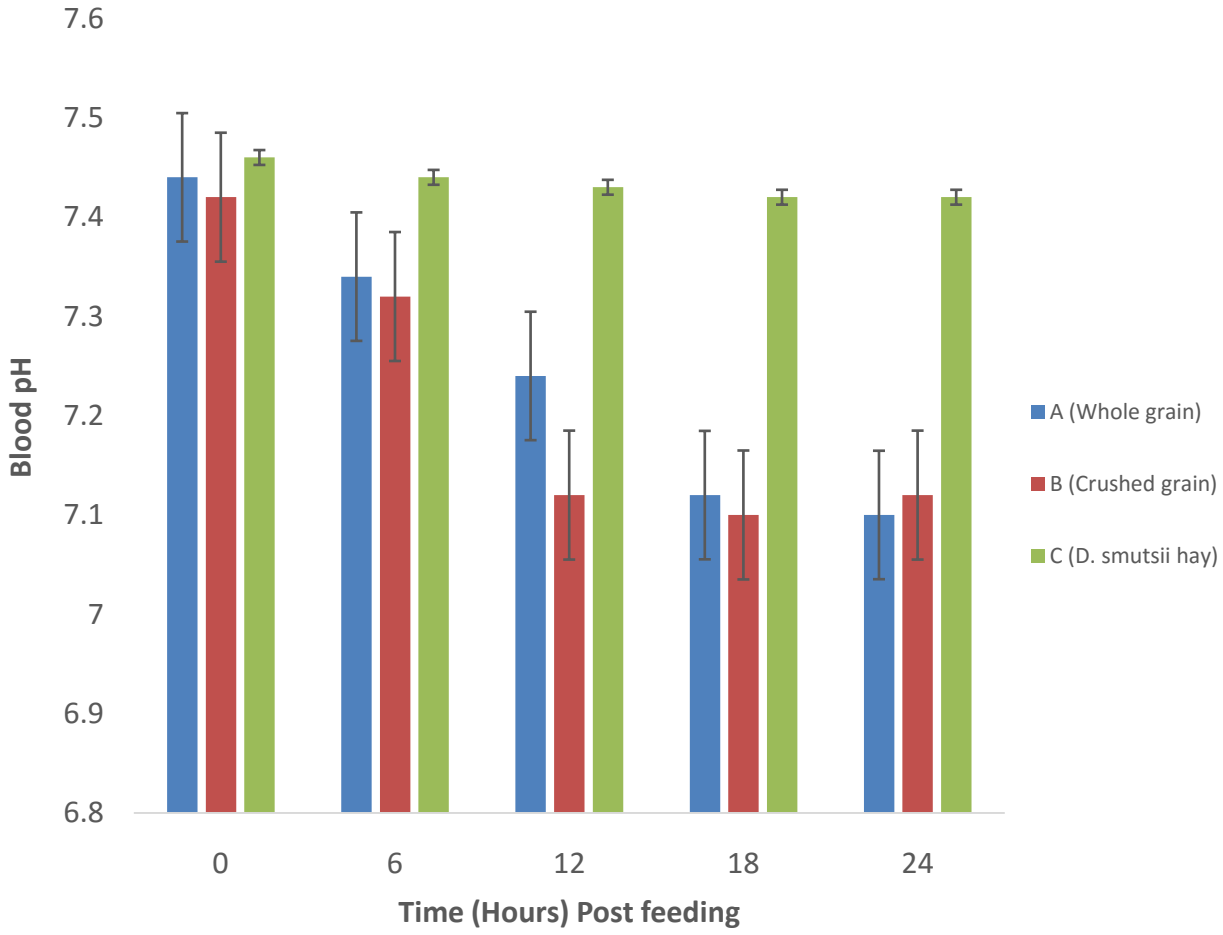

Figure 4.14: Effect of feeding whole or crushed maize grain on Mean values of blood pH of Yankasa sheep for 24 hrs in the second week of feeding trial

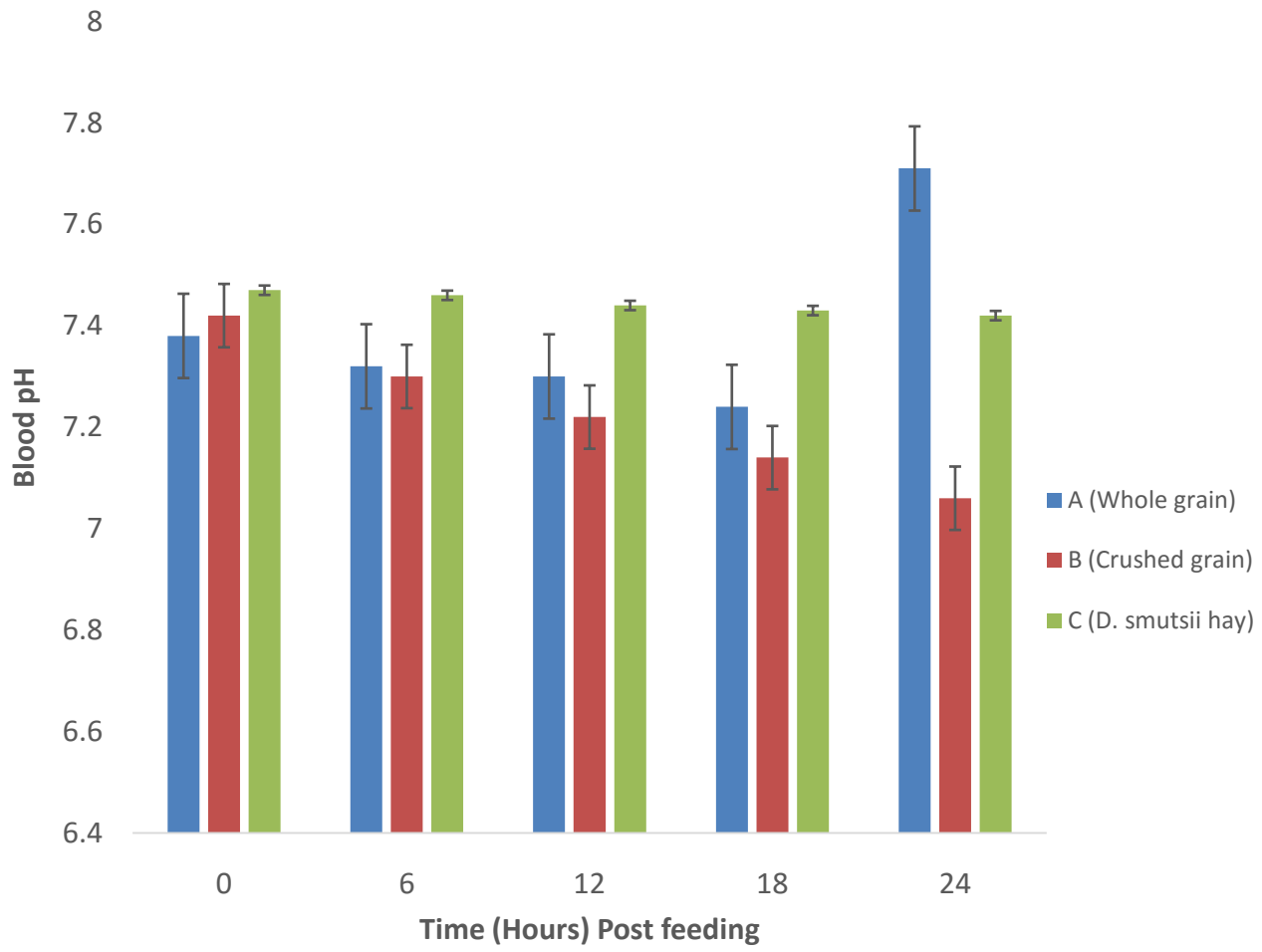

Figure 4.15: Effect of feeding whole or crushed maize grain on Mean values of blood pH of Yankasa sheep for 24 hrs in the third week of feeding trial

4.5 Acetic Acid

The mean Acetic acid significantly increased ($p< 0.05$) from 231.30 ± 18.29 to 825.04 ± 170.91 in 8^{th}, hour through the experimental period in group A and from 147.56 ± 14.46 to 718.74 ± 162.09 in B then begin to decreased non-significantly ($p> 0.05$) from 10^{th} to 14^{th} hour and increased significantly ($p< 0.05$) from 815.74 ± 320.07 in 16^{th} hours to 952.30 ± 211.21 in 20^{th} for group A and from 718.74 ± 162.09 to 877.80 ± 240 in group B of the experimental animals in the first weeks. There were non-significantly increased ($p> 0.05$) in acetic acid at the 6^{th} hours of the experimental period in both group A and B, then begins to decreased significantly ($p< 0.05$) from 428.02 ± 103.63 to 202.34 ± 23.10 at 14^{th} hour and significantly increased ($p< 0.05$) from 202.34 ± 23.10 to 613.36 ± 246.78 in 18^{th} hours of both group A and from 260.14 ± 35.03 to 576.66 ± 280.63 in group B of the experimental period in the second week. There were significantly increased ($p< 0.05$) from 628.36 ± 136.04 to 1352 ± 338.24 at 18^{th}hours in group B (fed crushed maize grain) and from 559.14 ± 128.43 to 1514.22 ± 330.77 in A during the 2^{nd} hour then a highly significant increased ($p< 0.05$) at 1938.72 ± 212.07 in 24^{th} hour of the experimental period in group in the third week (Figures 4.16, 4.17, 4.18 and appendix VI respectively).

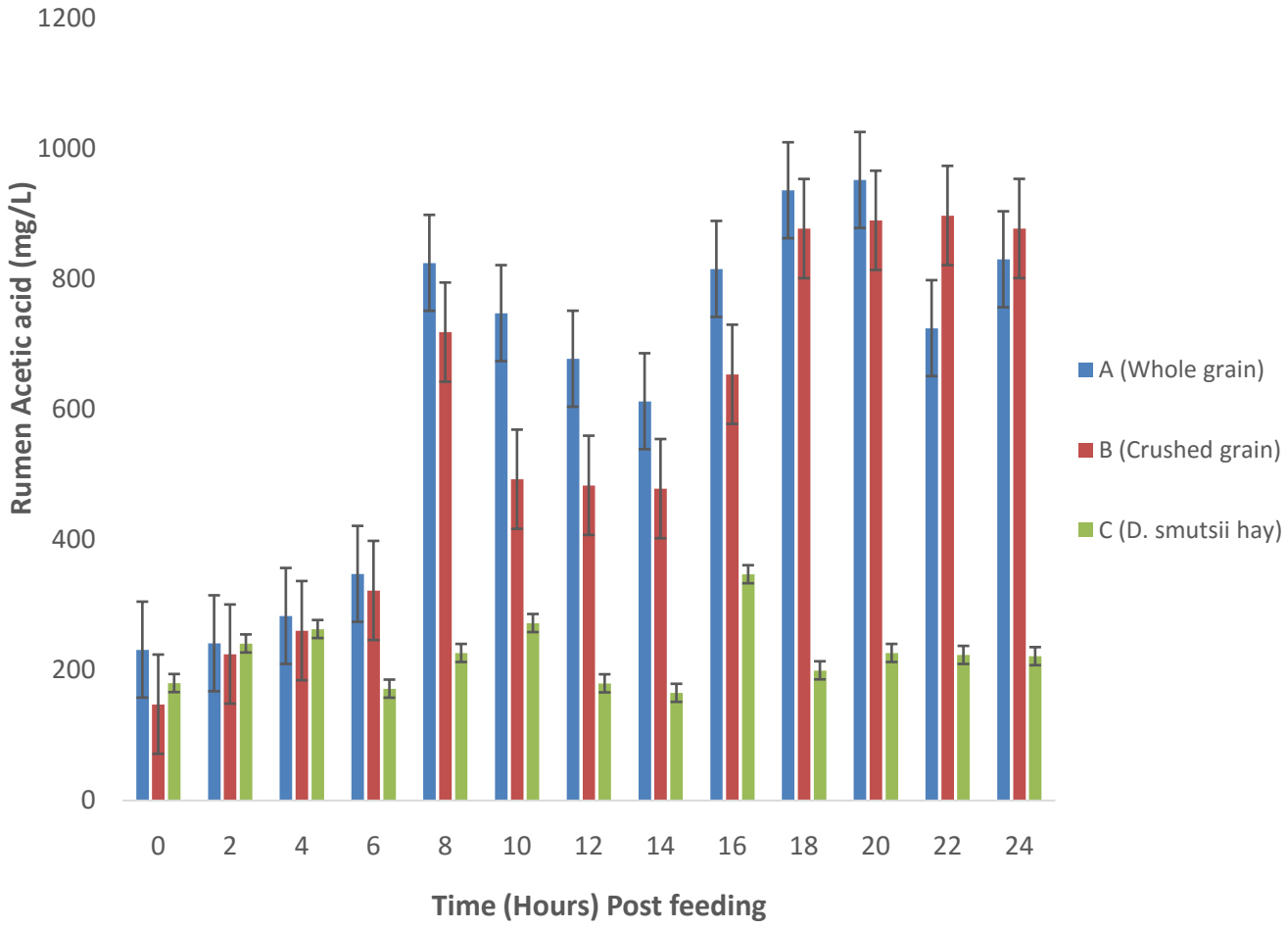

Figure 4.16: Effect of feeding whole or crushed maize grain on Mean values of Acetic acid of Yankasa sheep for 24 hrs in the first week of feeding trial

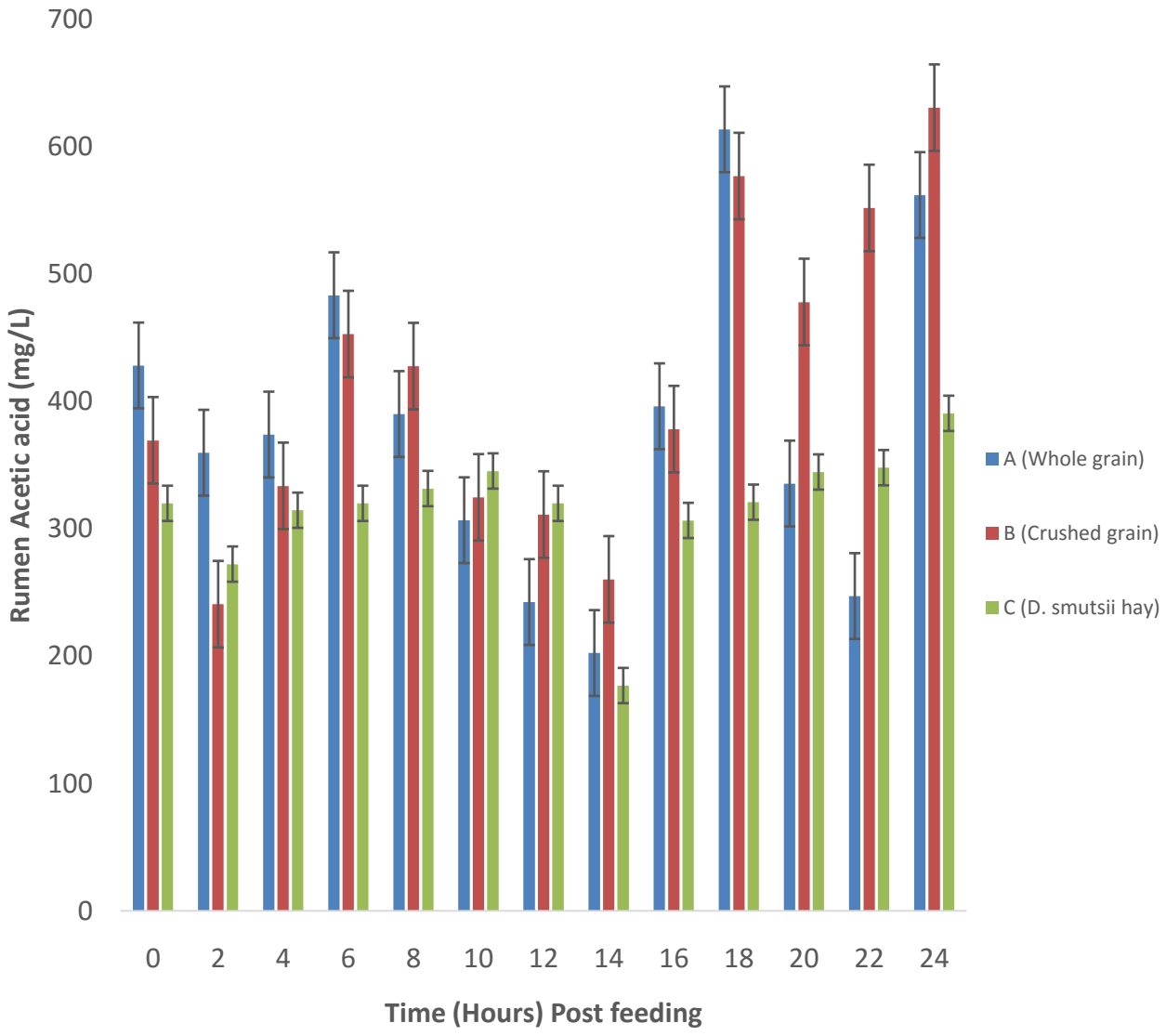
700
600
500
400
300
200
100
0
Rumen Acetic acid (mg/L)
0
2
4
6
8
10
12
14
16
18
20
22
24
Time (Hours) Post feeding
A (Whole grain)
B (Crushed grain)
C (D. smutsii hay)

Figure 4.17: Effect of feeding whole or crushed maize grain on Mean values of Acetic acid of Yankasa sheep for 24 hrs in the second week of feeding trial

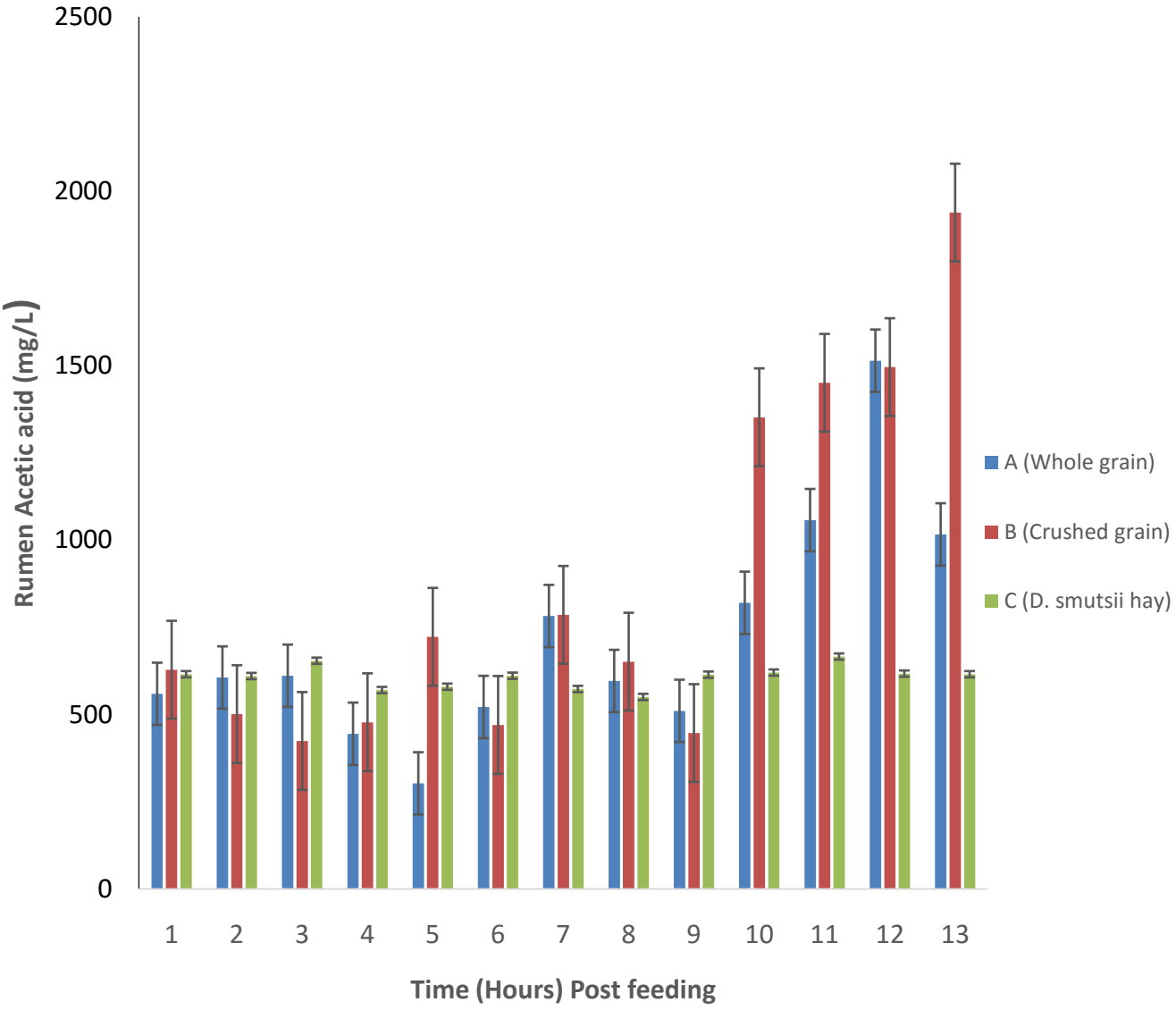

Figure 4.18: Effect of feeding whole or crushed maize grain on Mean values of Acetic acid of Yankasa sheep for 24 hrs in the third week of feeding trial

4.6 Lactic Acid

The mean values of Lactic acid increased significantly ($p < 0.05$) from 35.84 ± 0.36 to 44.60 ± 0.87 at 10^{th}, hours in group A and from 36.84 ± 1.87 to 45.18 ± 3.49 in group B then decreased non-significantly ($p > 0.0$) at the 16^{th} hours of the experimental period in the first week. Lactic acid increased significantly ($p < 0.05$) from 44.96 ± 2.61 in 8^{th} to 57.67 ± 7.78 in 16^{th}, hours in group B and there were no-significant decreased ($p > 0.05$) in group A at the same period of the experimental period in the second week. There were no-significant decreased ($p > 0.05$) at 20^{th} and 24^{th} hours in group A and B of the experimental period in the third week (Figures 4.19, 4.20, 4.21 and appendices VII respectively).

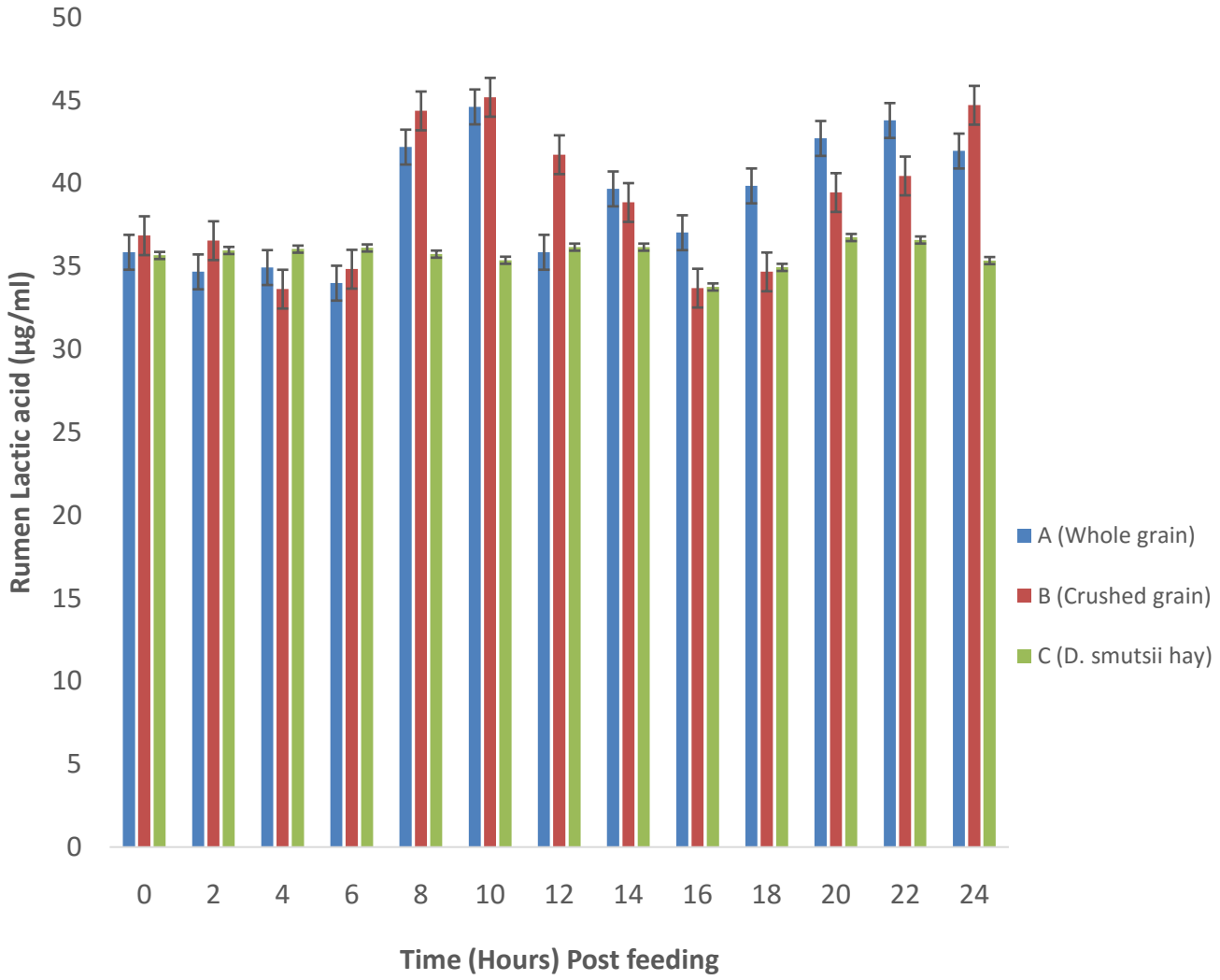

Figure 4.19: Effect of feeding whole or crushed maize grain on Mean values of Lactic acid of Yankasa sheep for 24 hrs in the first week of feeding trial

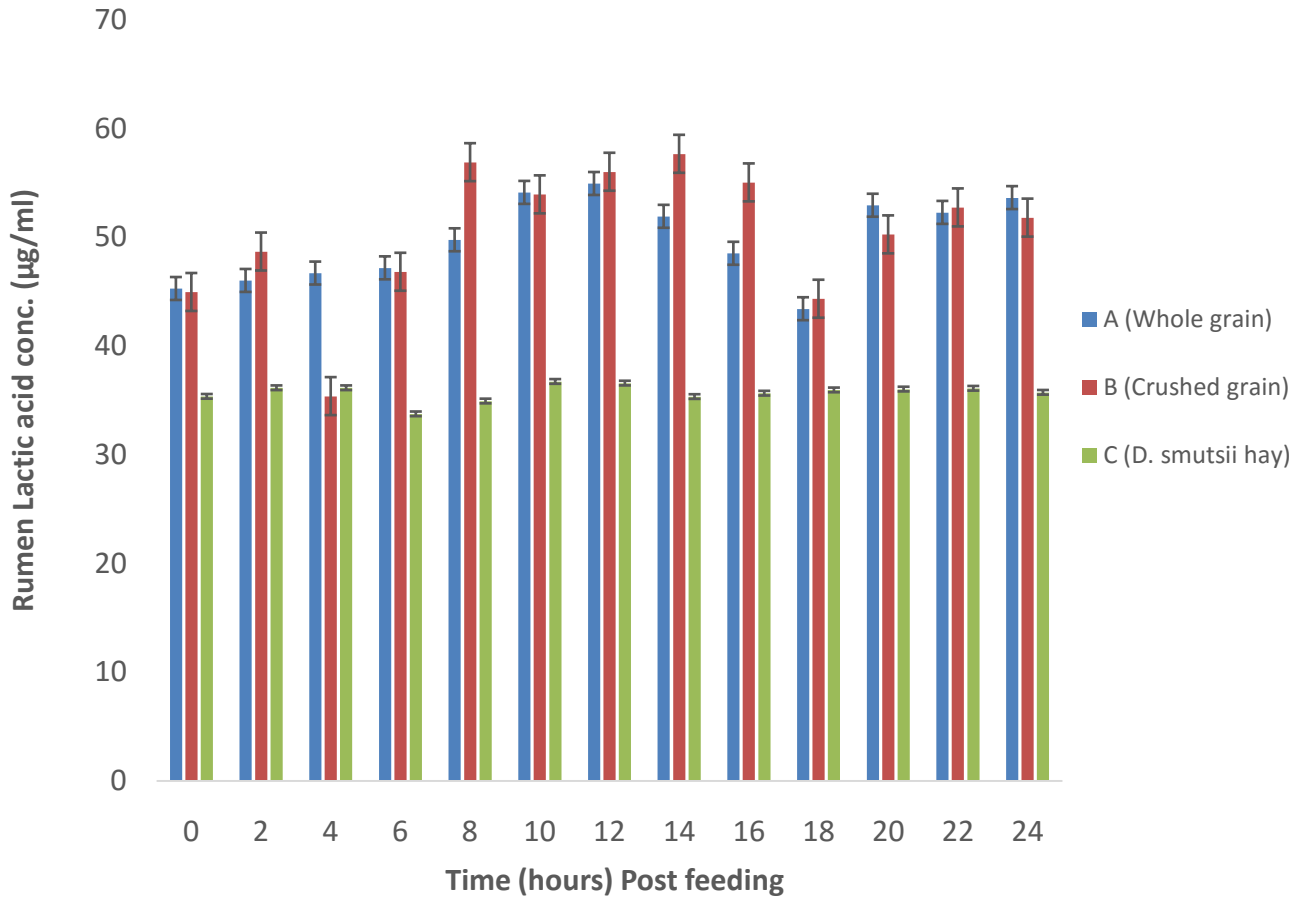

Figure 4.20: Effect of feeding whole or crushed maize grain on Mean values of Lactic acid of Yankasa sheep for 24 hrs in the second week of feeding trial

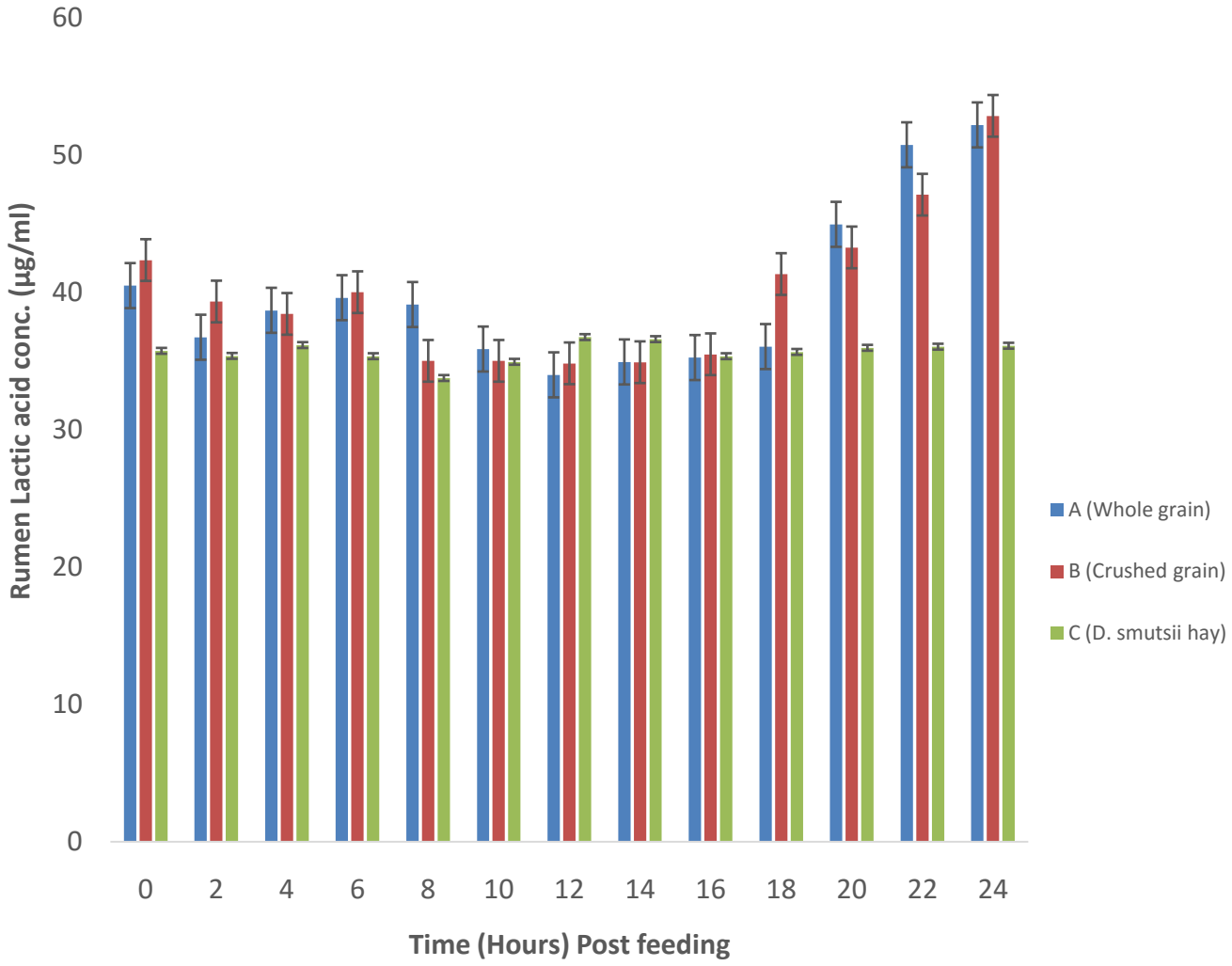

Figure 4.21: Effect of feeding whole or crushed maize grain on Mean values of Lactic acid of Yankasa sheep for 24 hrs in the third week of feeding trial

4.7 Rumen microbial Count

The mean aerobic count increased significantly ($p< 0.05$) 4.16 ± 0.22 to 4.50 ± 0.08 in 8^{th} hour in group A, and decreased significantly ($p< 0.05$) from 4.37 ± 0.05 to 4.02 ± 0.18 at 6^{th} hour in group B, then increased significantly ($p< 0.05$) to 4.61 ± 0.08 at 22^{nd} hour, in group B. Rumen microbes decreased significantly ($p< 0.05$) from 4.50 ± 0.08 to 4.04 ± 0.19 at the 14^{th} hours in group A of the experimental period in weeks one. The mean aerobic count increased significantly ($p< 0.05$) from 4.14 ± 0.22 to 4.46 ± 0.09 at10^{th} hour in group A and from 4.27 ± 0.08 to 4.51 ± 0.04 at 12^{th} hour in group B then decreased significantly ($p< 0.05$) to 4.34 ± 0.11 at the 20^{th} hours and increased significantly ($p< 0.05$) to 4.57 ± 0.10 at the 24^{th} hour in group B of the experimental animals at week two. The mean aerobic count increased significantly ($p< 0.05$) from 4.16 ± 0.24 to 4.57± 0.13 at the 16^{th}, hour in group A and from 4.26 ± 0.03 to 4.50 ± 0.11 in group B and then decreased significantly ($p< 0.05$) to 4.17 ± 0.02 at the 24^{th} hours in both group A and B in week three of the experiment period (Figures 4.22, 4.23, 4.24 and appendix VIII respectively)..

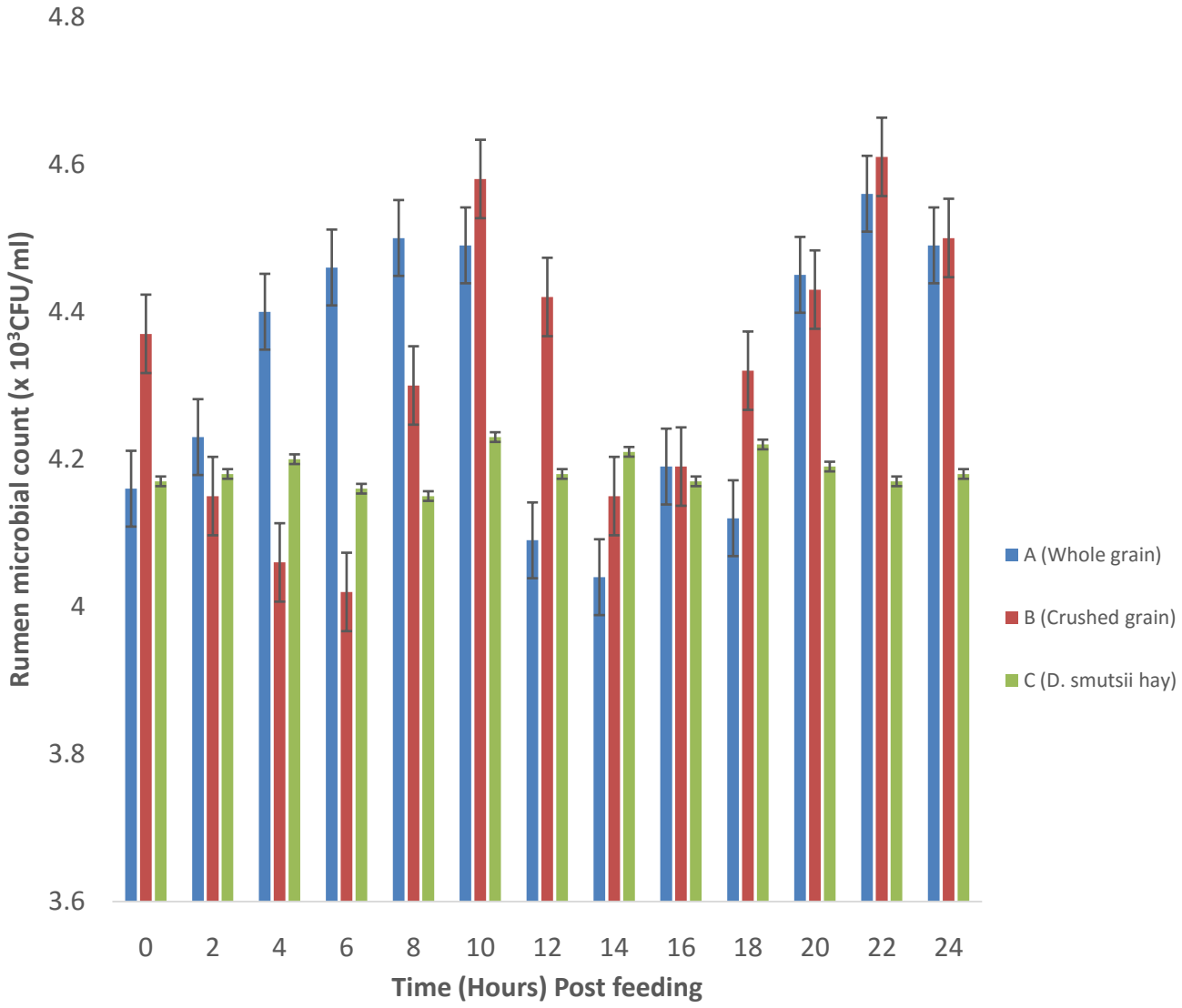

Figure 4.22: Effect of feeding whole or crushed maize grain on Mean values of Rumen microbial counts of Yankasa sheep for 24 hrs in the first week of feeding trial

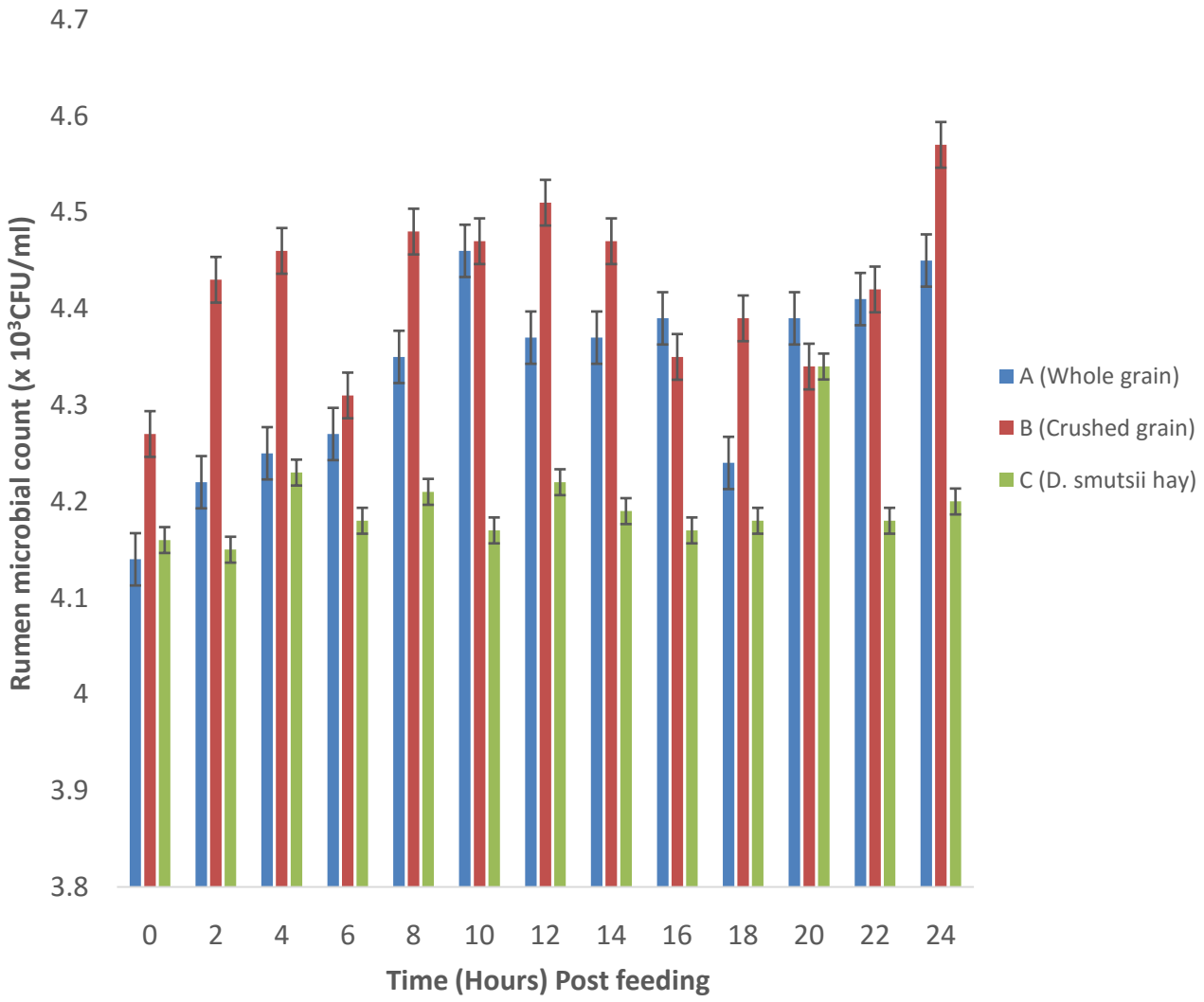

Figure 4.23: Effect of feeding whole or crushed maize grain on Mean values of Rumen microbial counts of Yankasa sheep for 24 hrs in the second week of feeding trial.

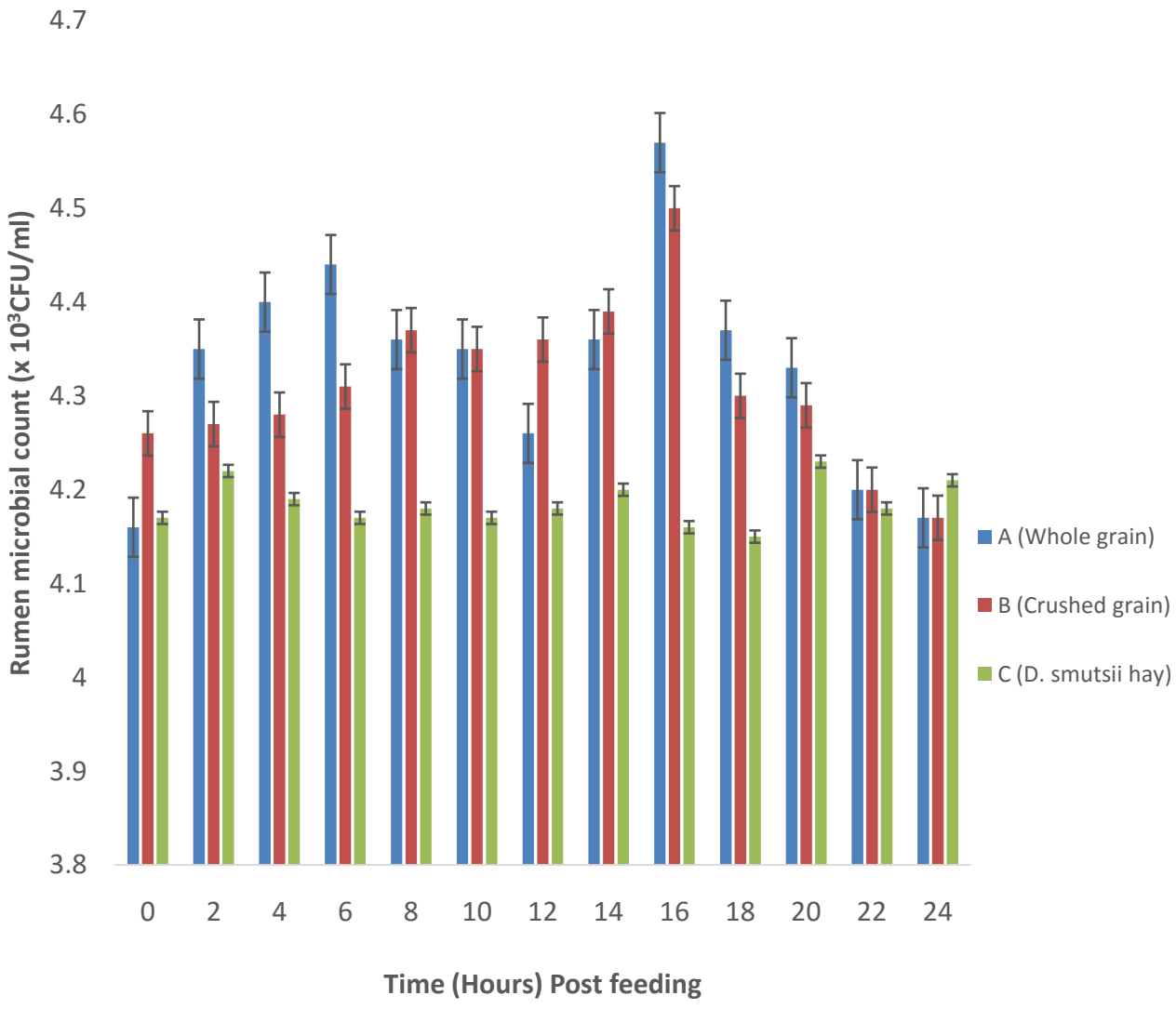

Figure 4.24: Effect of feeding whole or crushed maize grain on Mean values of Rumen microbial counts of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.8 Haematological Parameters

4.8.1 Packed Cell Volume (PVC)

The Packed Cell Volume (PVC) decreased non-significantly ($p<0.05$) with the introduction of the experimental feed at the 6^{th} and 24^{th} hours in both group A and B and significantly decreased ($p<0.05$) from 36.00 ± 2.79 to 23.80 ± 5.02 at 12^{th} hour in group A of the experimental animals in week 1. There is non-significant increased ($p<0.05$) 35.20 ± 3.15 to $39.69 \pm 0.3.87$ at 24^{th} hour in group A in week 3 (Figures 4.25, 4.26, 4.27, appendices IX, XI, and XIII respectively).

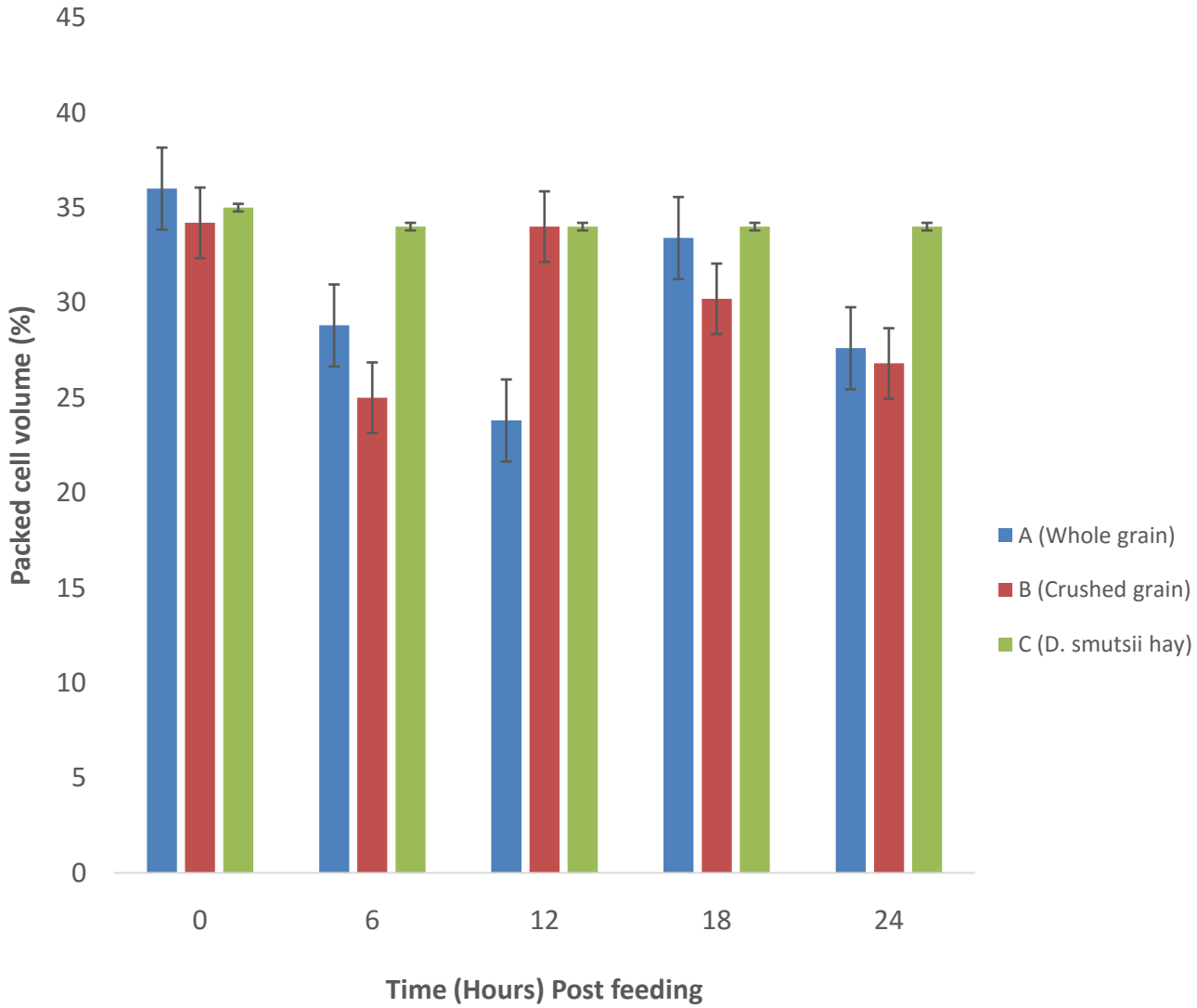

Figure 4.25: Effect of feeding whole or crushed maize grain on Mean values of Packed Cell Volume (PCV) of Yankasa sheep for 24 hrs in the first week of feeding trial.

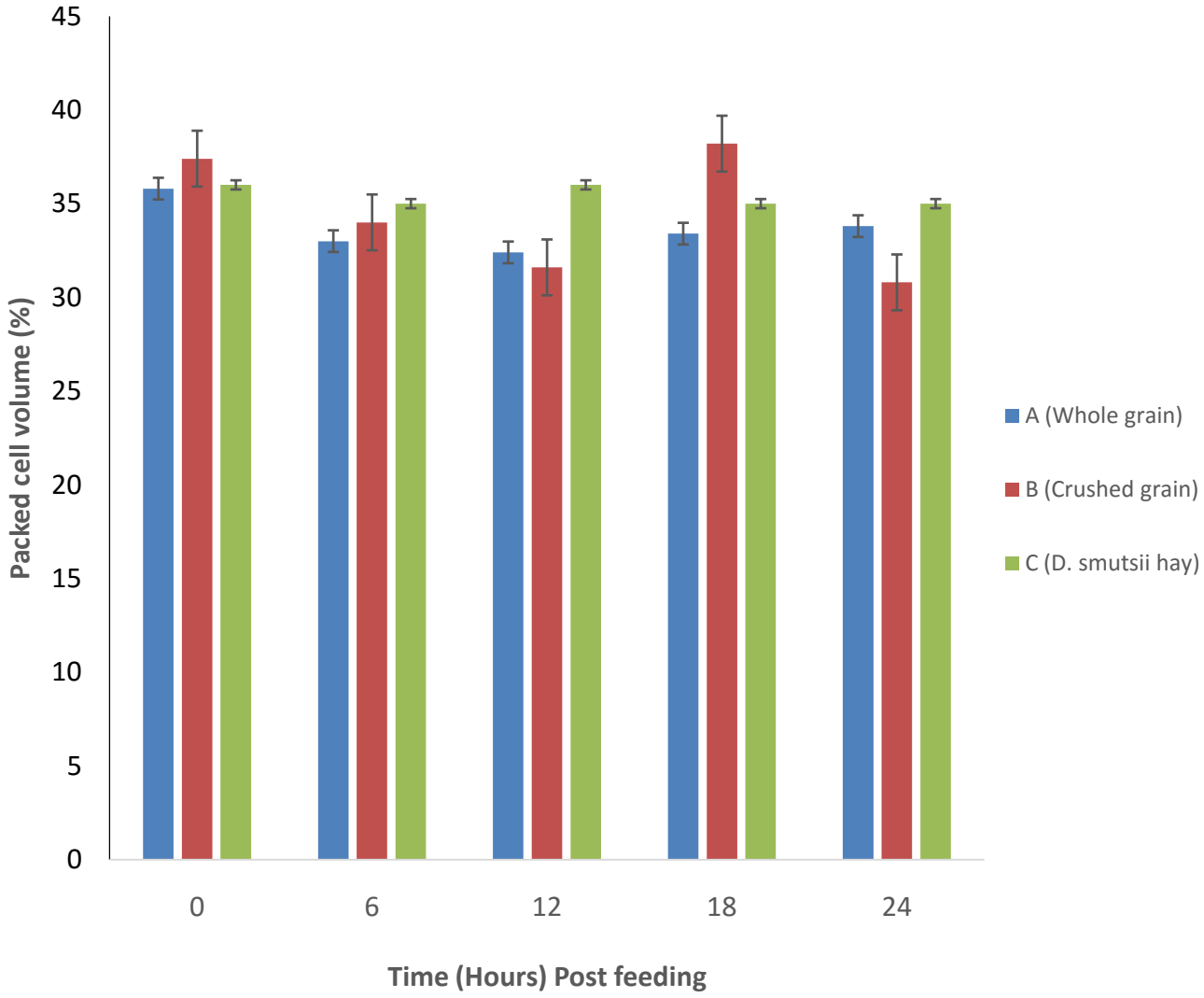

Figure 4.26: Effect of feeding whole or crushed maize grain on Mean values of Packed Cell Volume (PCV) of Yankasa sheep for 24 hrs in the second week of feeding trial.

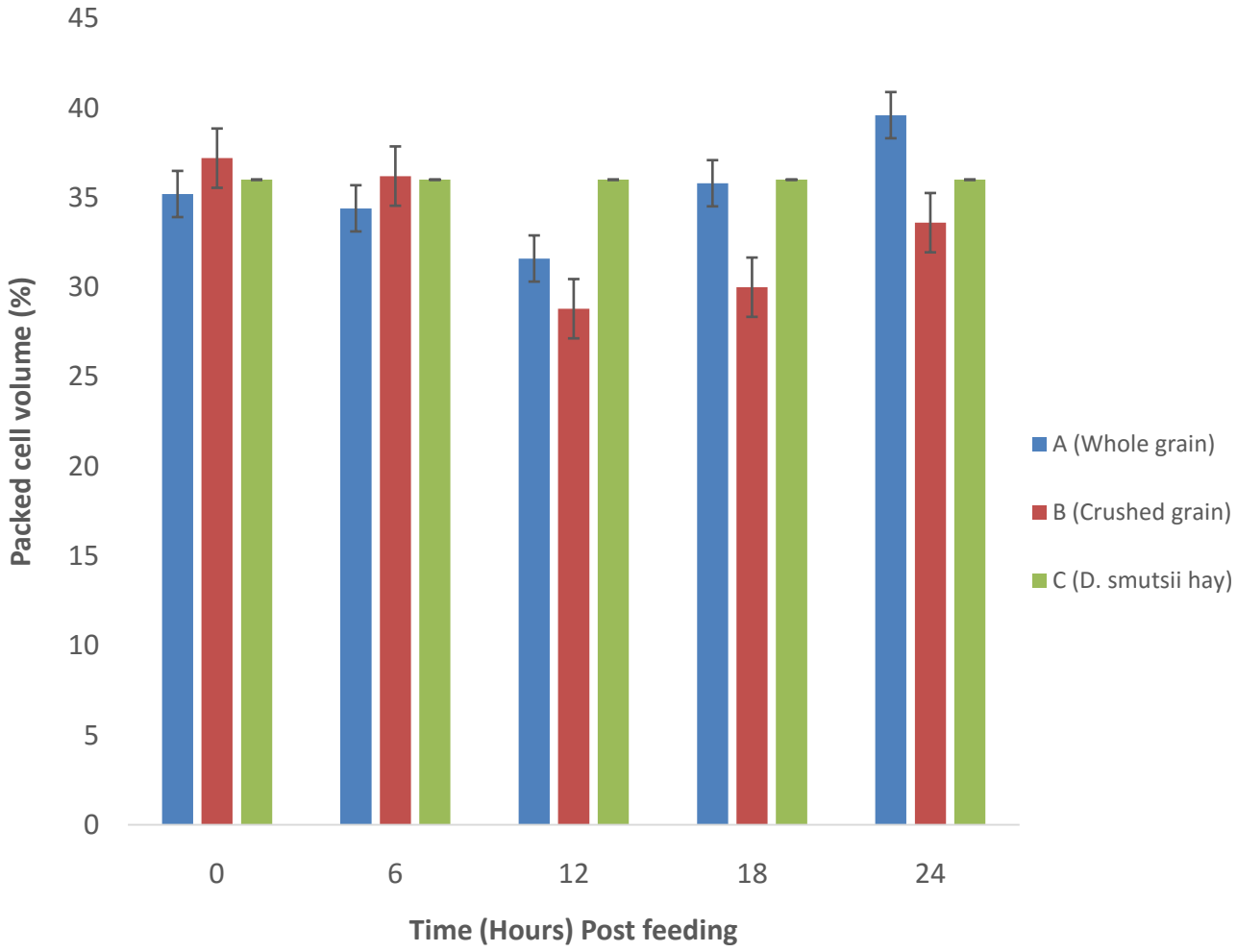

Figure 4.27: Effect of feeding whole or crushed maize grain on Mean values of Packed Cell Volume (PCV) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.8.2 Haemoglobin concentrations

There were non-significant decreased ($p>0.05$) in haemoglobin concentrations at the 6^{th} and 24^{th} hours in both group A (fed whole maize grain and B (fed crushed maize grain) and a non-significant increased ($p>0.05$) from 11.98 ± 0.93 to 13.84 ± 4.94at 12^{th} hour of the experimental period in week 1. Non-significant decreased ($p>0.05$) in haemoglobin concentrations was observedat the 6^{th}, 12^{th} and 24^{th} hours in both group A and B and a non-significant increased ($p>0.05$) was also observed at 18^{th} hour in group B of the experimental animals in week 2. There were non-significant decreased ($p>0.05$) in haemoglobin concentrations at the 12^{th}, 18^{th} and 24^{th} hours in both group A and Bof the experimental animals in week 3 (Figure: 4.28, 4.29, and 4.30, appendices IX, XI and XIII respectively).

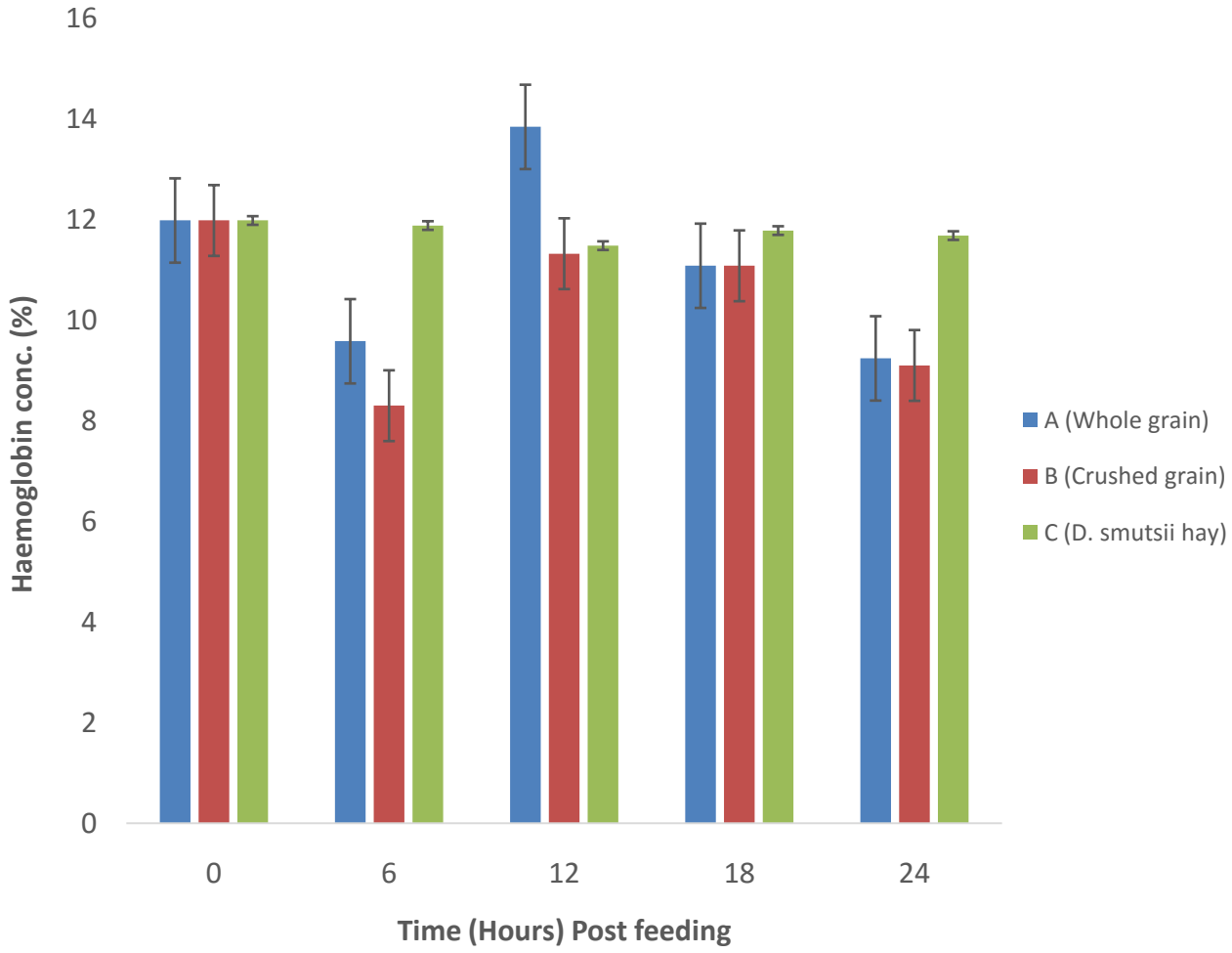

Figure 4.28: Effect of feeding whole or crushed maize grain on Mean values of Haemoglobin (HGB) of Yankasa sheep for 24 hrs in the first week of feeding trial.

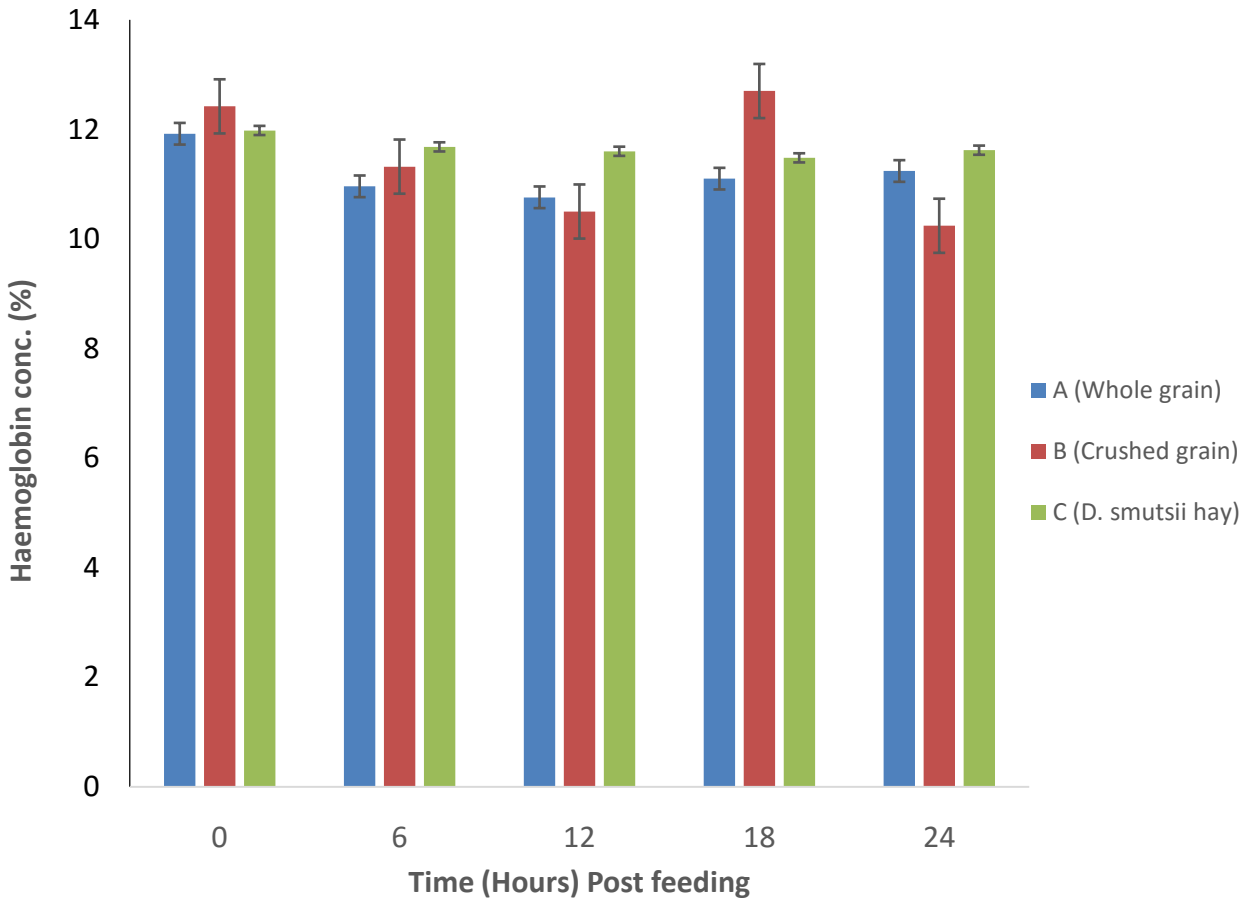

Figure 4.29: Effect of feeding whole or crushed maize grain on Haemoglobin (HGB) of Yankasa sheep for 24 hrs in the second week of feeding trial.

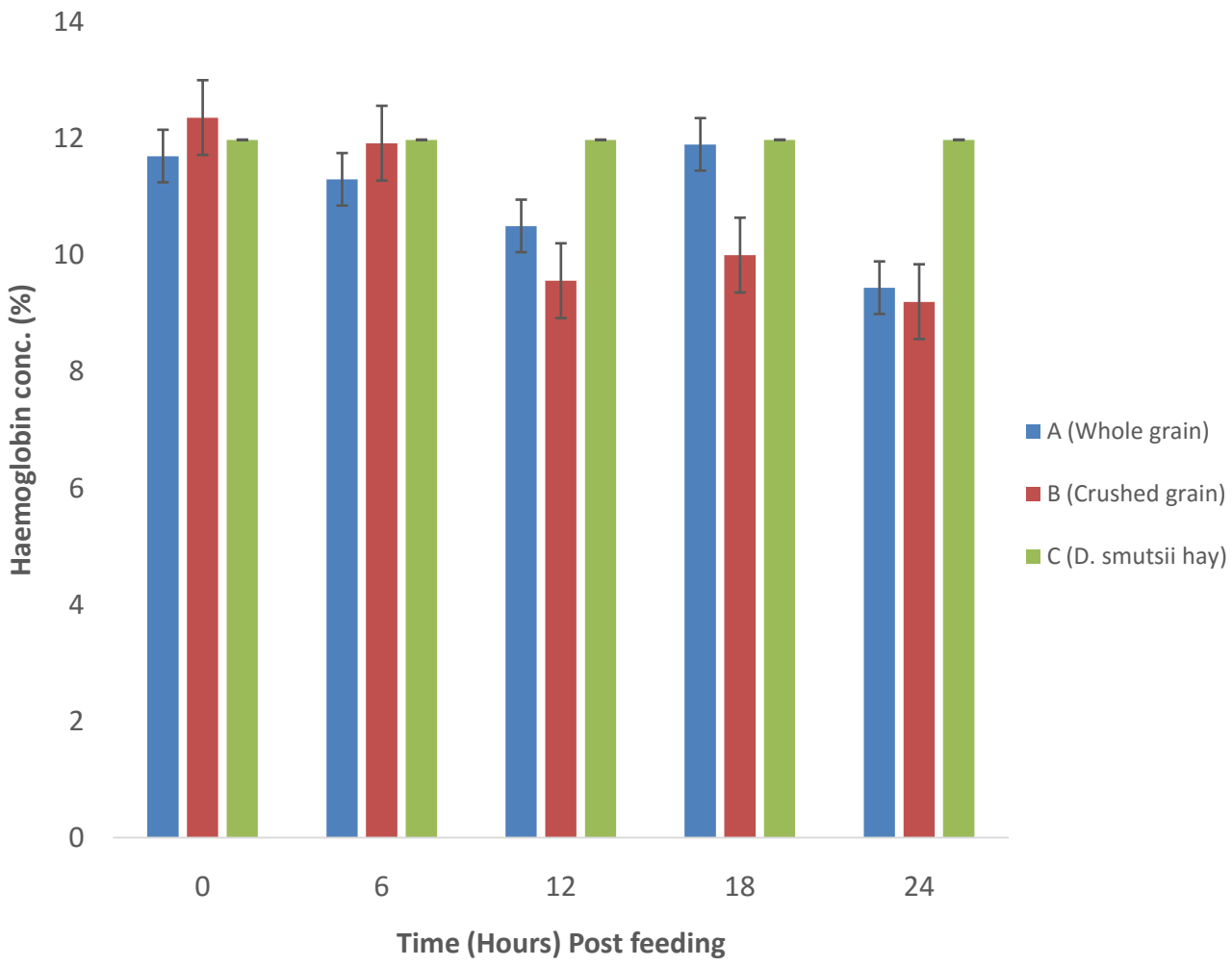

Figure 4.30: Effect of feeding whole or crushed maize grain on Mean values of Haemoglobin (HGB) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.8.3 Red Blood Cell (RBC)

The Red Blood Cell (RBC) count increased non-significantly ($p>0.05$) from 7.08 ± 1.12 to 9.00 ± 4.27 at the 6^{th} hour in group B (fed crushed maize grain) and decreased non-significantly ($p>0.05$) at the 6^{th} hour in group A (fed whole maize grain) then 12^{th}, $18^{th}$and 24^{th} hours in both group A and Bof the experimental period in week 1. There were no-significant decreased ($p>0.05$) at the 6^{th} to 24^{th} hours in both group A and B of the experimental animals in week 2. Then a non-significant increased ($p>0.05$) at the 24^{th} in group A of the experimental period in week 3 (Figure: 4.31, 4.32, and 4.33, appendices IX, XI and XIII respectively).

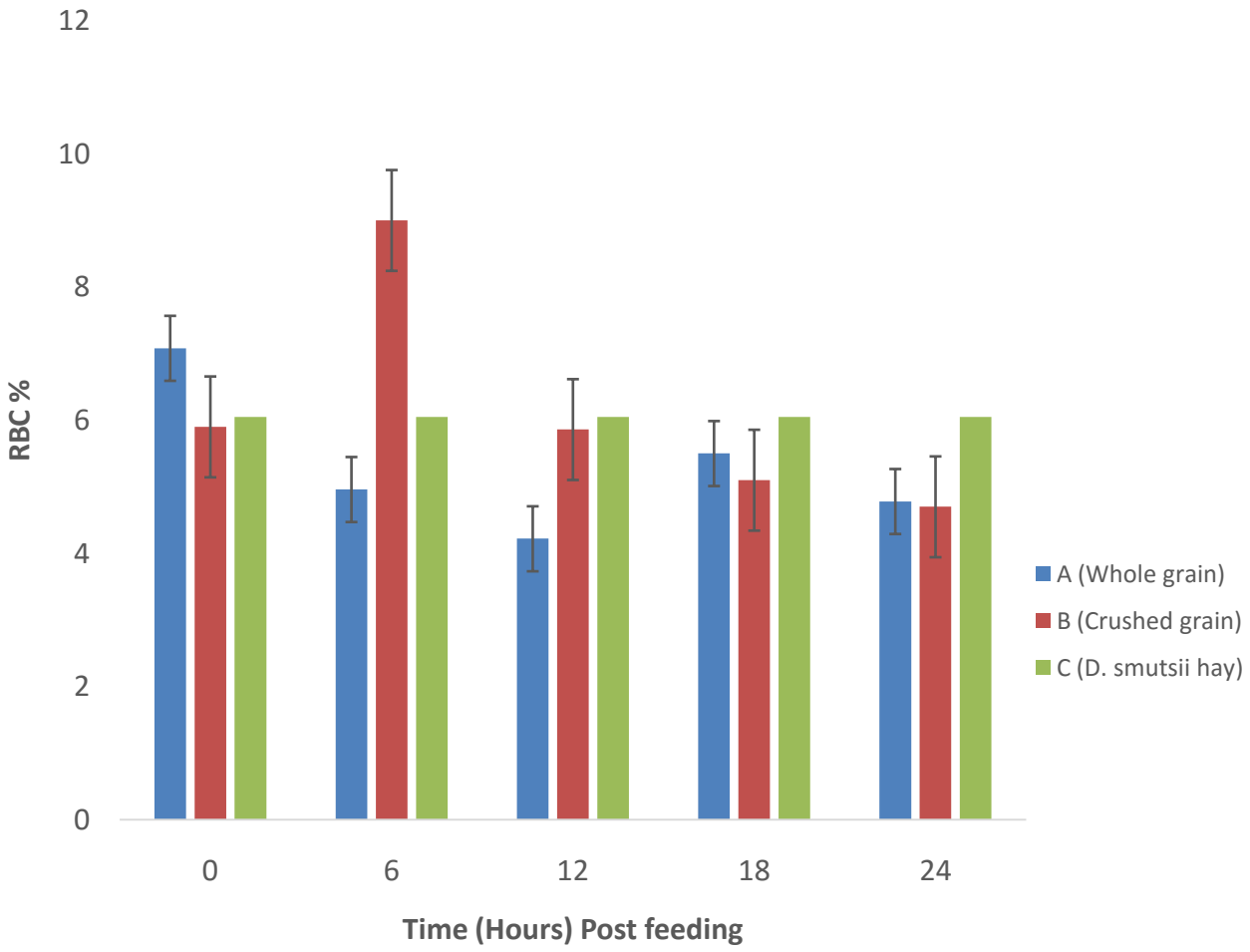
12
10
8
6
4
2
0
RBC %
0
6
12
18
24
Time (Hours) Post feeding
A (Whole grain)
B (Crushed grain)
C (D. smutsii hay)

Figure 4.31: Effect of feeding whole or crushed maize grain on Mean values of Red Blood Cell (RBC) of Yankasa sheep for 24 hrs in the first week of feeding trial.

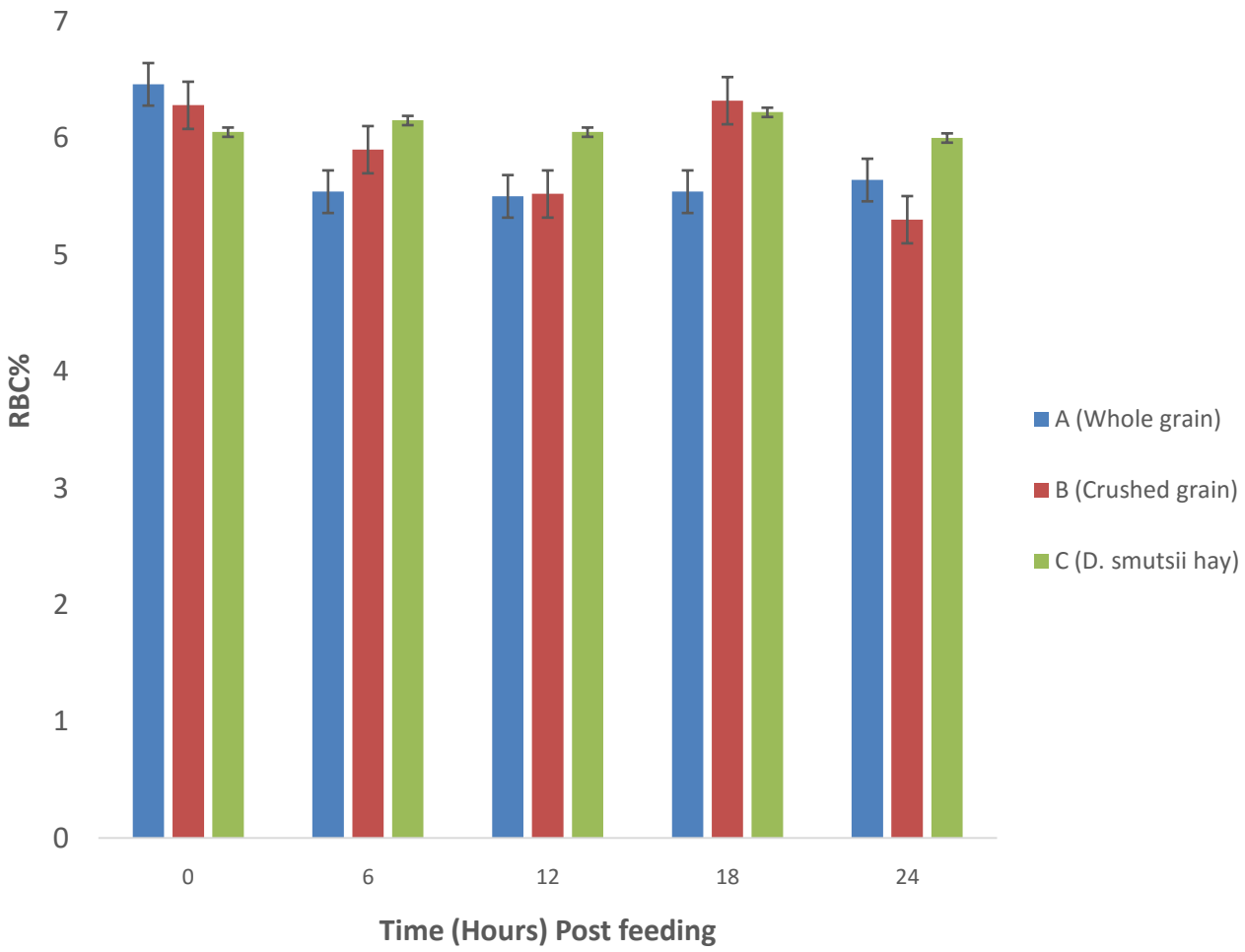

Figure 4.32: Effect of feeding whole or crushed maize grain on Mean values of Red Blood Cell (RBC) of Yankasa sheep for 24 hrs in the second week of feeding trial.

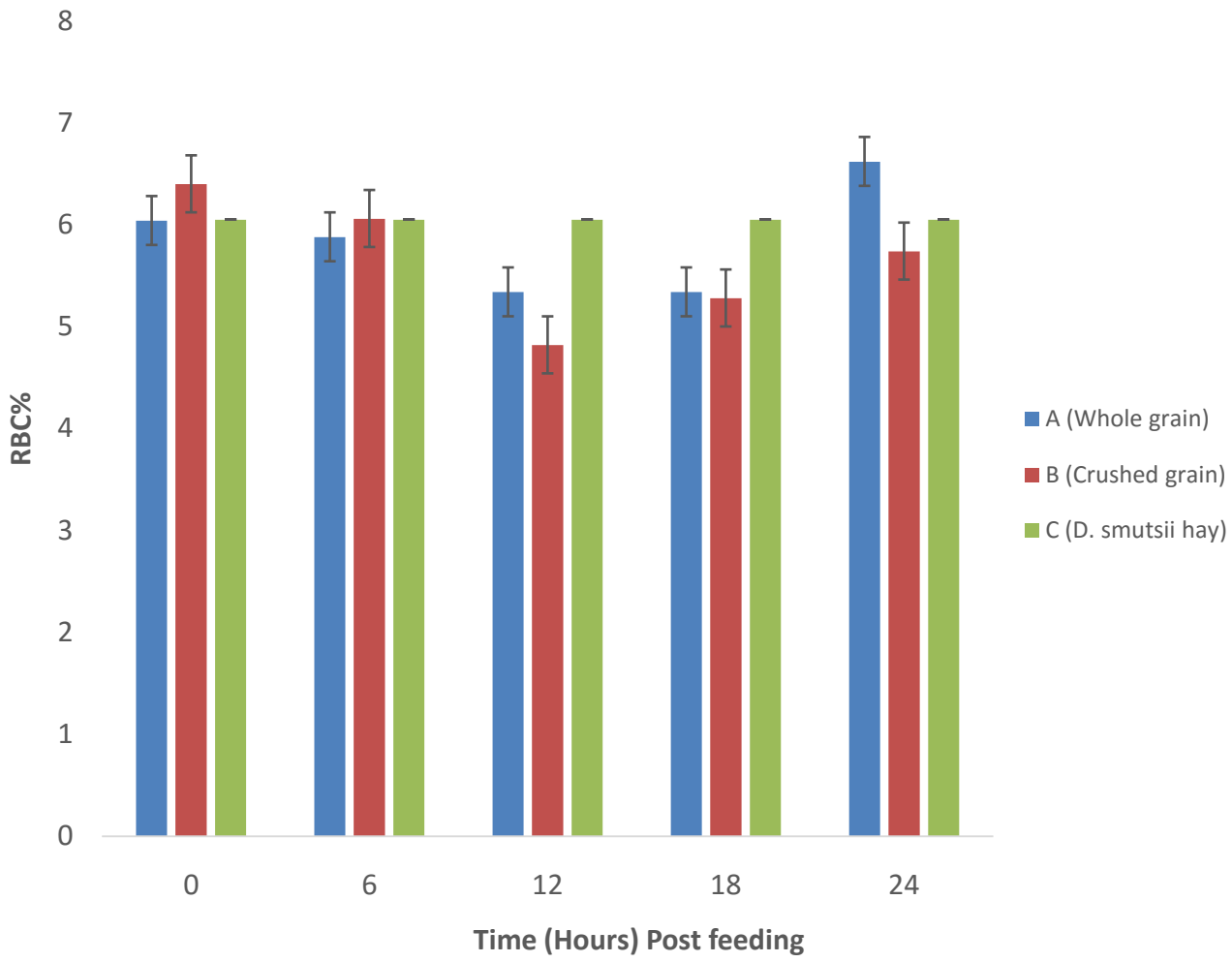

Figure 4.33: Effect of feeding whole or crushed maize grain on Red Blood Cell (RBC) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.8.4White blood cells (WBC)

White blood cells (WBC) increased significantly ($p < 0.05$) from 10.18 ± 1.37 at 6th hour to 10.80 ± 2.06 at 18th hour in group B, and from 6.94 ± 0.93 to 11.64 ± 1.29 at 24th hour in group A in week 1 of the experimental period. White blood cells increased significantly ($p < 0.05$) from 9.64 ± 1.45 to 10.72 ± 1.06 at 12th and to 13.04 ± 1.69 at 24th hour in group B in week 2 (Figure 4.34, 4.35, 4.36, appendices X, XII and XIV respectively).

4.8.4.1 Neutrophil

There were non-significant decreased in Neutrophil ($p > 0.05$) at 24th hour in group B and at 12th hours in group A in week 3 and a non-significant decreased ($p>0.05$) at 24th hours in group B in week 2 (appendices XII).

4.8.4.2 Lymphocyte

The values of lymphocytes increased non-significantly ($p>0.05$) at 12th hours with the introduction of experimental feed in group A, 24th hour in group B in week 1, there were no-significant increased ($p>0.05$) at 6th and 12th hours in group A and 24th hour in group B respectively in week 2. A non-significant decreased ($p>0.05$) was observed at 12th hour in group B and A non-significant increased ($p>0.05$) was observed at 12th hour in group A of the experimental period (appendices X, XII and XIV respectively).

4.8.4.3 Monocyte

Monocytes increased non-significantly ($p >0.05$) at 18th hour in group A, B and 24th hour in group A week 2 of the experimental period. A non-significant decreased ($p>0.05$) was observed at 22nd hours in group B of the experimental animals (appendices XII).

4.8.4.4 Total protein

Total protein increased non-significantly ($p > 0.05$) with the introduction of the experimental feed at 24^{th} hour in group A in week 1(appendices X).

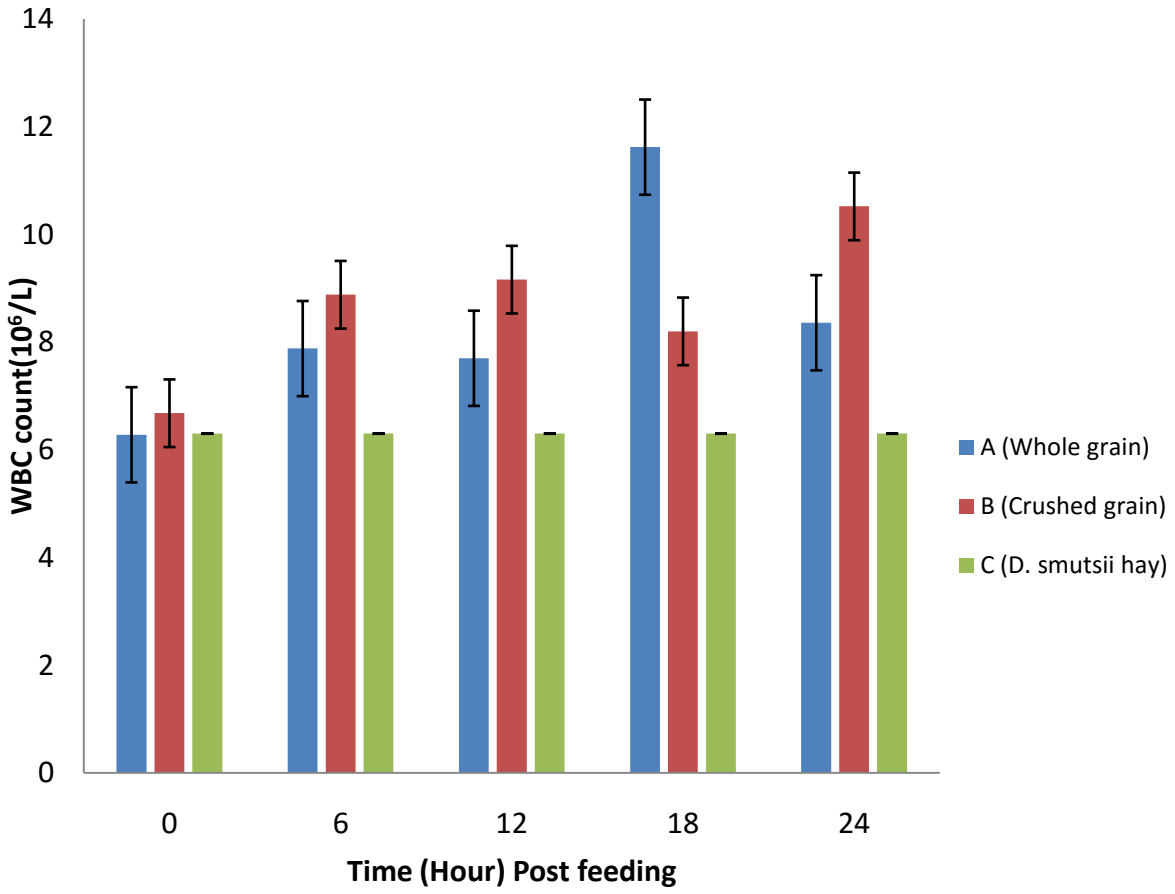

Figure 4.34: Effect of feeding whole or crushed maize grain onMean values of White Blood Cell (WBC) of Yankasa sheep for 24 hrs in the first week of feeding trial.

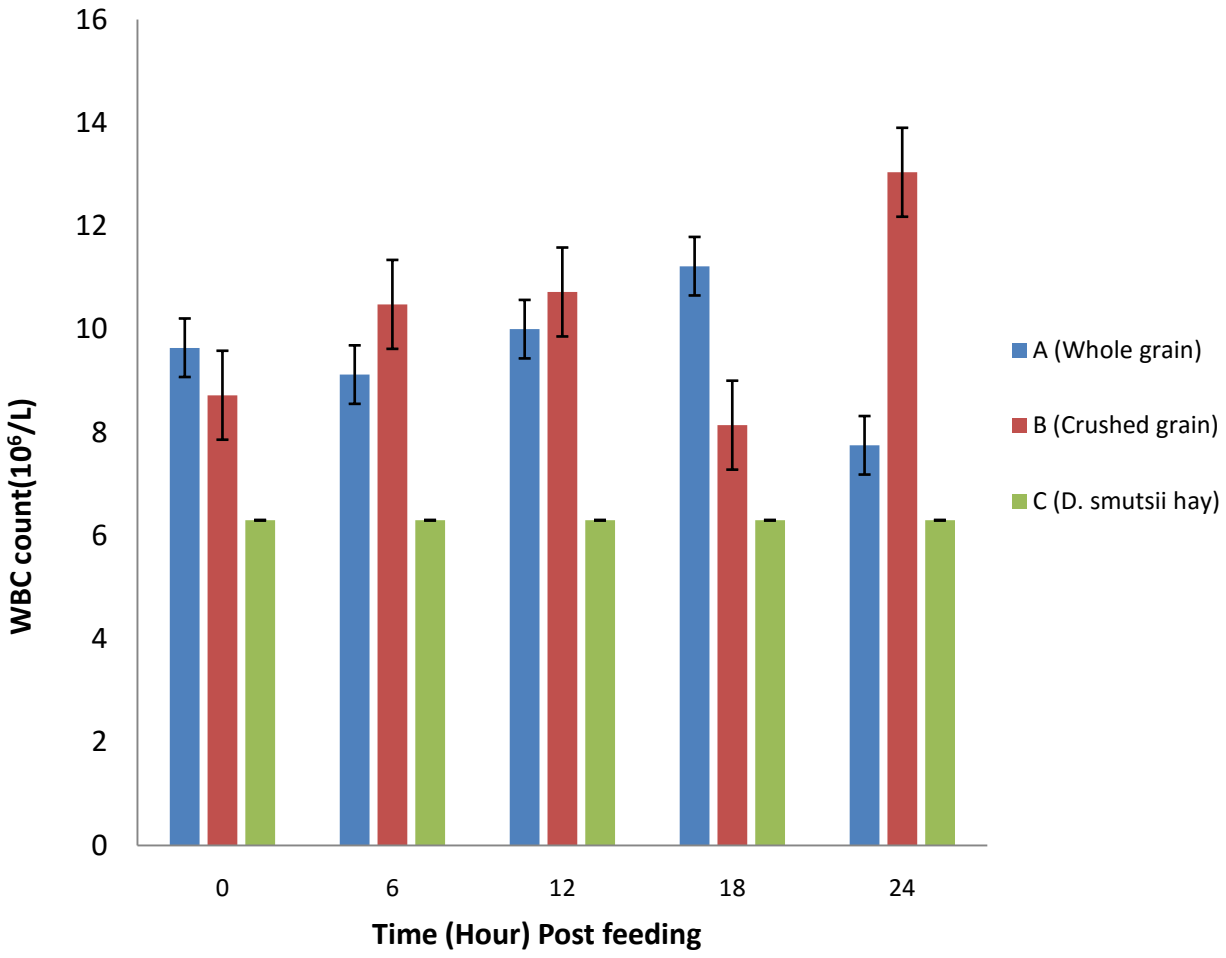

Figure 4.35: Effect of feeding whole or crushed maize grain on Mean values of White Blood Cell (WBC) of Yankasa sheep for 24 hrs in the second week of feeding trial.

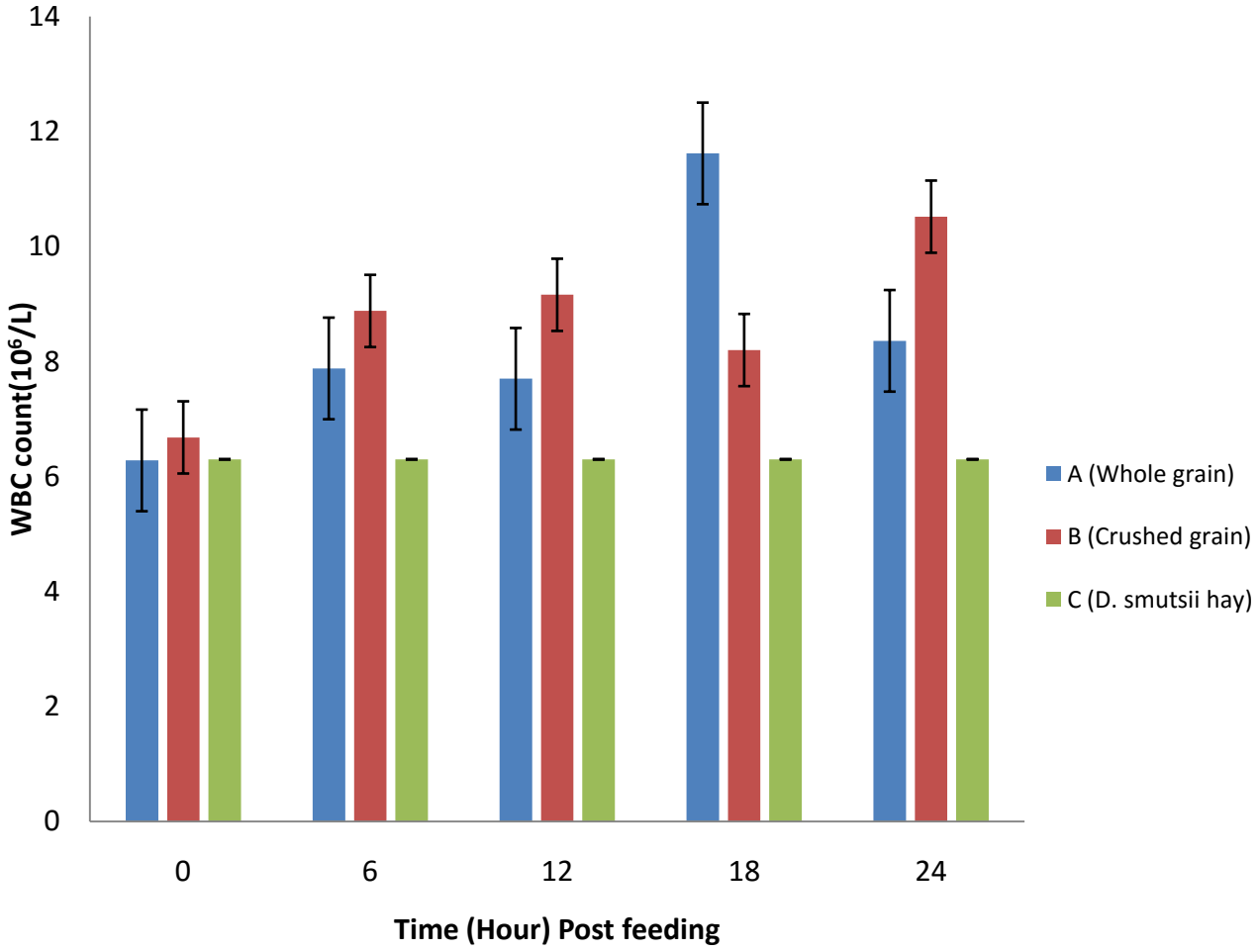

Figure 4.36: Effect of feeding whole or crushed maize grain on Mean values of White Blood Cell (WBC) of Yankasa sheep for 24 hrs in the third week of feeding trial.

4.9 Post Mortem finding

The dead experimental sheep were taken to the Department of Veterinary Pathology Ahmadu Bello University Zaria necropsy room for post mortem examination. Sloughed off rumen mucosa were observed during the examination

4.9.1 Gross Pathological changes

Gross lesion observed in photomicrographs of the sheep group A and B were sloughing off of the rumen mucosal wall (Plate II and III).

4.9.2 Histopathological changes

The observed rumen histopathological changes in groups A and B were areas of necrosis (Plates IV and V, respectively).

Plate II: Gross photograph of the rumen of sheep (group A) fed whole maize grain showing sloughing off of rumen mucosa surface (arrowed indicate).

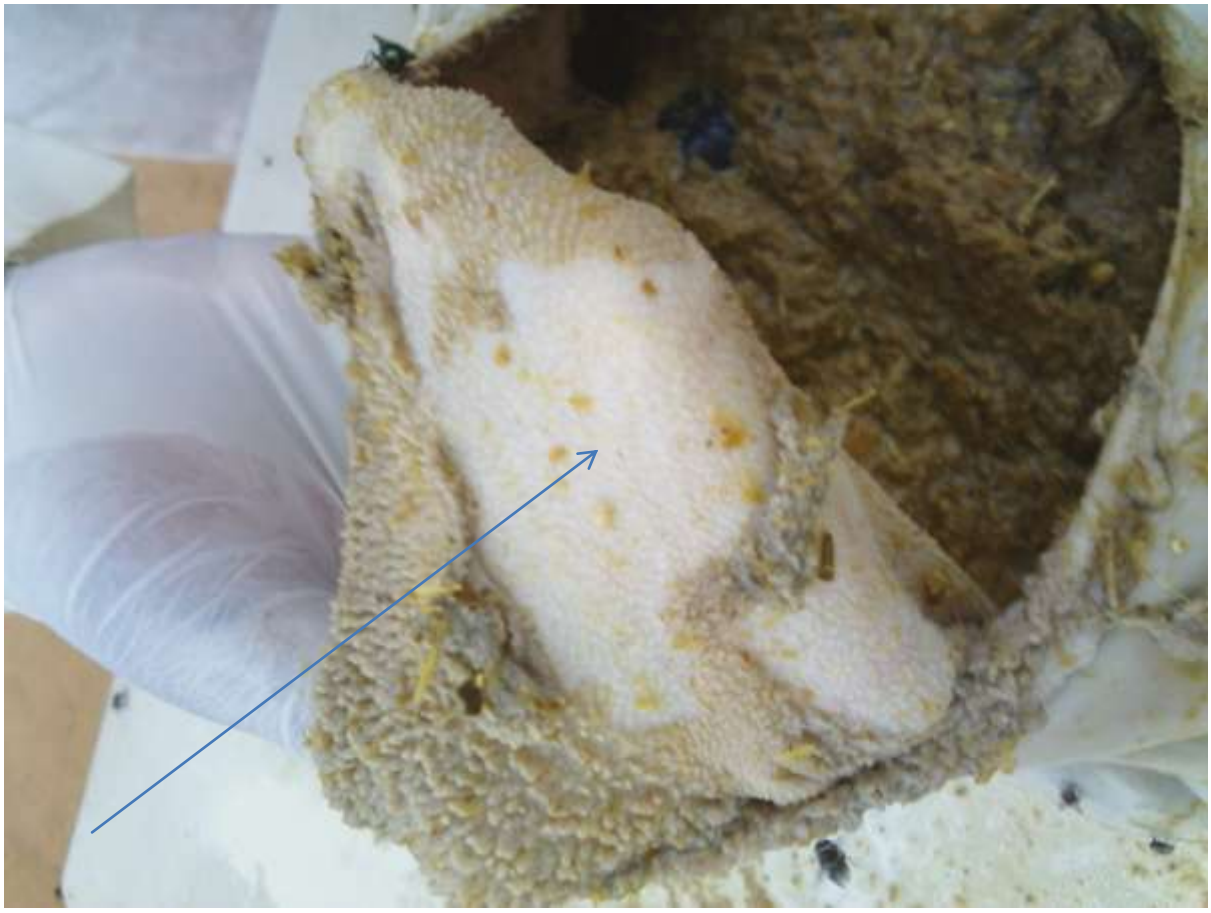

Plate III: Gross photograph of the rumen of sheep (group B) fed crushed maize grain showing sloughed offmucosal surface (arrowed indicate).

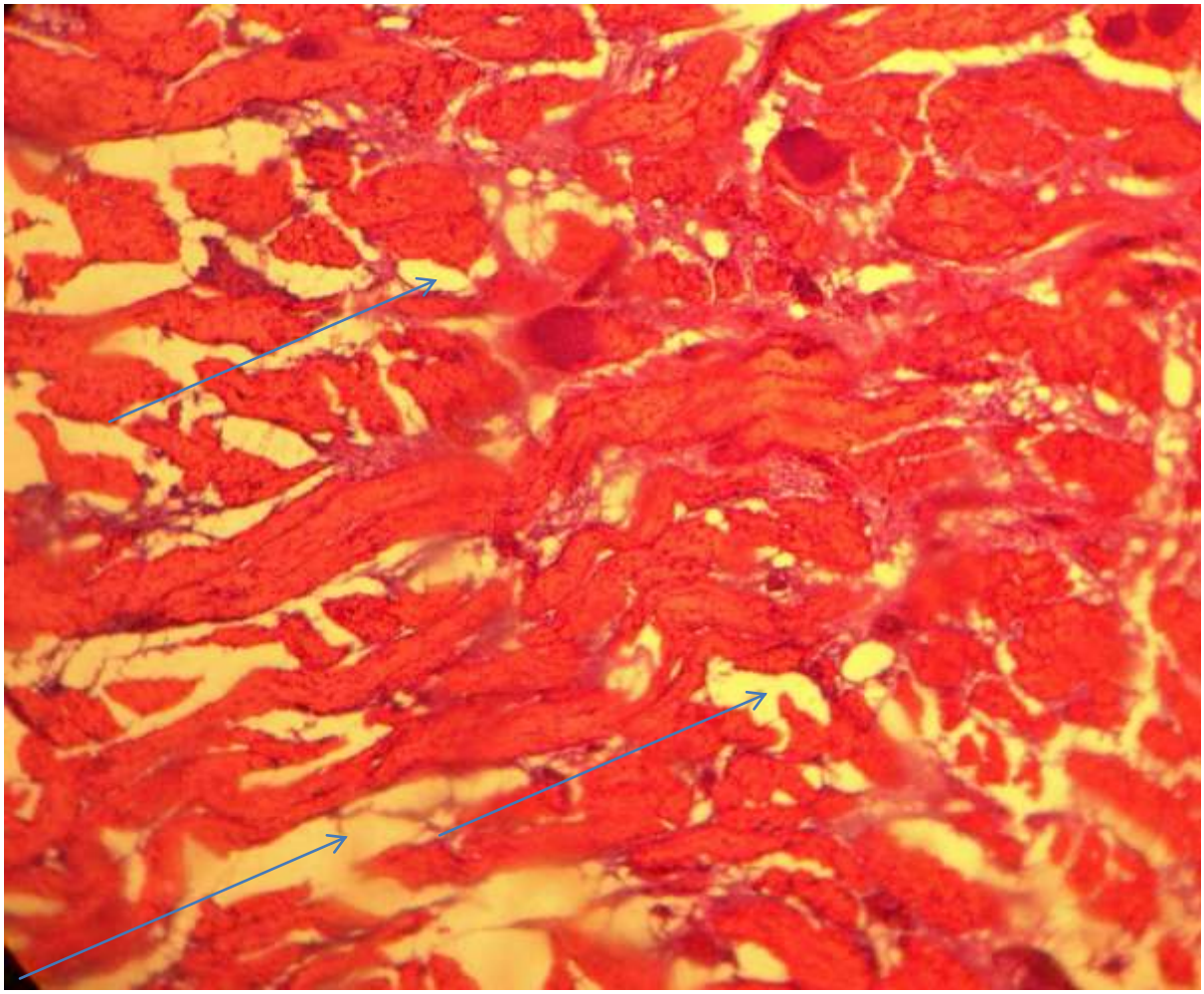

Plate IV: Photomicrograph of a section of the rumen wall of sheep (group A) fed whole maize grain showing necrosis of the rumen mucosa (Arrowed) (H and E x 400)

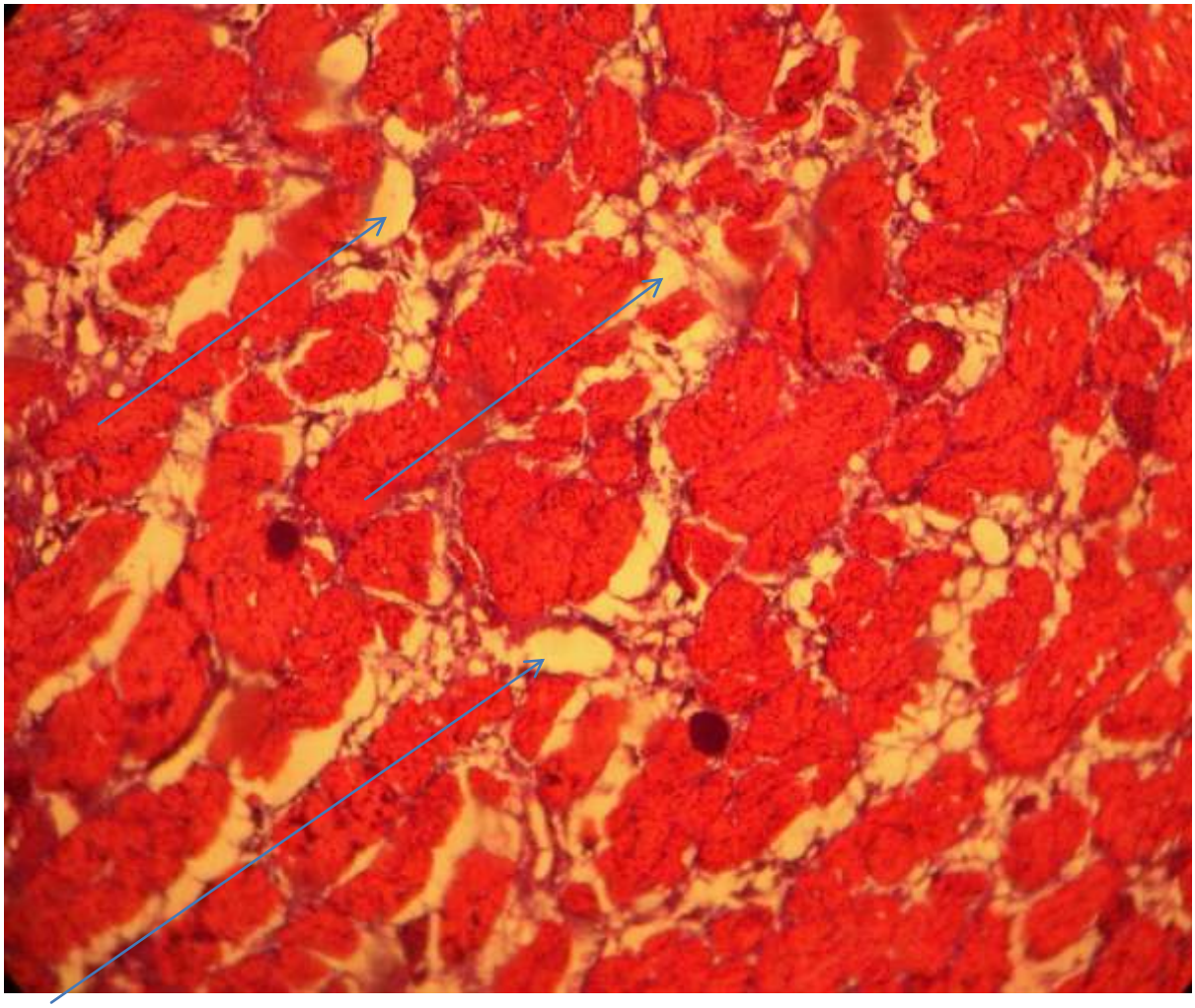

Plate V: Photomicrograph of a section of the rumen of sheep (group B) fed crushed maize grain showing necrosis of the rumen mucosa (Arrowed) (H and E x 400)

CHAPTER FIVE

5.0 DISCUSSION

On the vital parameters, the observed increased in therespiratory rate valueswith the introduction of maize grain preparations (whole and crushed) at 24th hour post feeding in both groups A and B.This increase in respiration might be due to stimulation of respiratory centre by increased carbon dioxide (CO_2) tension and blood/or decreased blood pH (Huber, 1976; Radostits *et al.,* 2007) and could also be attributed to metabolic and respiratory acidosis consequent to lactic acid accumulation in rumen (Samad, 2000). Similar increase in respiratory rate has been reported by Tanwar and Mthur (1983) and Ullah *et al.,* (2013).

Pulse rate slightly increased at 12th hour post introduction of the experimental feed in group A (fed whole maize grain) and group B (fed crushed maize grain) and increased significantly ($p< 0.05$) at 24thhour of the experimental period. Variation in pulse rate has been reported to reflect the rate at which the heart pumps blood through the body (Adetunji *et al.*, 2002). This could also be due to the fact that metabolic acidosis activates sympathetic nervous system and causes tachycardia (Samad, 2000). However, the higher values found in pulse rate in animals in group A and B posibly indicated of greater stress at that period as reported by Badru *et al.*(2009)that rectal temperature and pulse rates are both used to determine the health status and adaptability of domestic animals to stressful condition.

The decreased in rectal temperature in this study after the feeding whole or crushed maize grain could be due to acidosis, leading to dehydration, fall in total plasma volume and to some extent, depression of the cardiovascular system (Radostits *et al.*, 2007). This also agrees with the findings of Nour *et al.* (1998); Hajikolaei *et al.* (2006) and Tufani *et al.* (2013) who reported similar decreases in rectal temperature due to rumen acidosis in sheep and goats

following experimental induction with rice grain and fruits. It could also be attributed to the absorption of endotoxin in circulation due to carbohydrate engorgement (Coa *et al*., 1987). The rectal temperature values obtained in this finding agrees with the earlier report by Sanusi *et al* (2011) who reported rectal temperature values (38.40 to 39.30^{0}C) in West African dwarf sheep.

The study produced subacute ruminal acidosis which manifested clinical signs, such as loss of appetite in some animals, depression, diarrhoea, and reduction in rumen pH to below 5.5 from the 16th hour after the experimental feeding. These signs agree with the report of Mahmood *et al.* (2013) who reported similar clinical signs as anorexia, apathy, teeth grinding (bruxism), muscle twitching, ruminal stasis and the excretion of soupy or watery faeces in lactic acidosis in goats. These signs could be attributed to the toxic effect of ruminal acidosis associated with rapid fermentation of carbohydrates which alters the ruminal function through proliferation of acid resistant bacteria and an increase in the production of volatile fatty acids and lactate, which cause a sharp drop in ruminal pH leading also to ruminitis, liver abscesses and laminitis (Oetzel *et al.*, 2003:Gozho *et al*., 2005: Penner *et al.*, 2009: Gonzalez *et al.*, 2012). The decreased in rumen pH below 5.5 also talies the report by Owens*et al.* (1998);Kleen*et al.* (2003);Beauchemin and Yang,(2005)who reported that normal ruminal pH in sheep is between 6.4 and 6.8 and that values less than 5.5 or greater than 7.0 are considered abnormal; ruminal pH with values less than 5.5 being defined as subacute rumen acidosis.

On the rumen pH, the decrease in the rumen pH values observed in animals in both group A and B after the introduction of experimental feed could probably due to faster fermentation by cellulolytic or amylolytic bacteria activities leading to lactic acid production as earlier reported by Jyoti *et al.* (2000). The observed decrease in rumen pH also agrees with the earlier work of Haji *et al.,* (2006) who reported the rumen pH of 6.21 ± 0.26 at 3hours and 5.35

± 0.03 at 6 hour in sheep post feeding and also similar to report by Basak *et al*. (1993); Mohamed Nour *et al.* (1999) in experimentally induced Lactic acid in Nubian goat using sorghum flour. .

The insignificant decrease ($p > 0.05$) observed in blood pH in this study with the minimum lowest value of 7.04 (± 0.02) at the 24th hour is in agreement with the findings of Haji *et al.* (2006) who reported a decrease in the blood pH from 7.41 ± 0.03 at 0 hour to 7. 23 ± 0.06 at 48th hours post feeding in lactic acidosis induced in sheep using sugar. The decrease may be attributed to the fact that the animals have been feeding on maize grain feed for 24 hours a week for 3 weeks and therefore, some level of acidamia might have developed, as explained by Ortolani *et al*. (2003).

The significant increase ($p < 0.05$) at the 8th hour in both group A and B of acetate acid level obtained in this study could be due to the hay that was included in the feed to the animals for six days after feeding the experimental diet before the following weeks for the repetition of the experiment. This is similar to the report of Joshua (2016) who reported that sheep with low energy density papillae had a greater increase in acetic acid level in the first 6hours compared to sheep with high density energy papillae.

The observed significant increase ($p < 0.05$) in the Lactic acid level at 8th and 10th, hours post feeding in both group A and B and the later decrease but non-significantly ($p > 0.0$) at the 16th hour after feeding whole or crushed maize grain feed may be attributed to the increased production of lactic acid by the lactic acid-producing bacteria *Streptococcus bovis.* This observation also agrees with the finding of Joshua (2016) who reported that Lactic acid concentration increased over 24 hours period but started decreasing after 18th hour in group with low energy density.

Regarding the rumen microbial count significant increase ($p < 0.05$) from 10th hour, in group B and the later significant decreased ($p < 0.05$) at the 16th hours in both group A and B could be due to the variation in the processing method of the diet, ruminal fluid pH and adaptation

of the bacteria to the rumen environment. This is similar to the reports Russel and Hino, (1985); Bramley *et al.* (2008) that as more fermentable carbohydrate is fed, the growth rate of all bacteria increases, thereby increasing total VFA production and subsequent decreases in rumen pH, favouring growth of *Streptococcus bovis.*

On haematological parameters, the observed packed cell volume (PCV) and haemoglobin (Hb) non-significantly increase ($p>0.05$) at 23^{rd} hours after feeding whole or crushed maize grain could be attributed to some level of haemoconcentration as a result of dehydration following drawing of systemic fluid in the rumen and diarrhoea that were encountered during this study and also reported by Radostis *et al.* (2007) in sheep fed with grain.

The insignificant decreases ($p>0.05$) in red blood cell (RBC) counts at the 6^{th} hour after feeding the maize grains may be associated with iron deficiency, internal bleeding, some types of anemia or some vitamin deficiency (Njidda *et al* 2014). However the RBC values obtained in this study were within the normal values reported by Campbell *et al.* (2010) in steers doses with Megasphera elsdnii fed high starch diets.

The WBC values obtained in this study were within the reference values of sheep (4 to $12x10^3/\mu l$) as reported byNseAbasi*et al.,* (2014).

The sloughing off of the rumen mucosa observed in the present study is similar to the findings by Steele *et al.* (2009) who reported that decreased ruminal fluid pH could be the cause of ruminitis along with ruminal parakeratosis and ruminal mucosa slough off.

CHAPTER 6

6.0 CONCLUTION AND RECOMMENDATIONS

6.1 CONCLUSION

i. Sub-clinical acidosis was obtained during the experiment from the decreased ruminal fluid pH which decreases from 6.75 ± 0.03 to 5.12 ± 0.12 at the 16^{th} hour after the experimental feed.

ii. Crushed maize grain fermentation was faster (5.59 ± 0.15 by the 4^{th} hour after the experimental feed) than whole maize grainfermentation (5.96 ± 0.07 by the 6^{th} hour after the experimental feed).

iii. A decreased in rumen fluid pH (from 7.47 ± 0.04 to 7.04 ± 0.02) from 6^{th} to 10^{th} hour, blood pH (from 7.47 ± 0.04 to 7.04 ± 0.02) from 6^{th} to 12^{th} hour and rectal temperature (from 39.22 ± 0.20 to 38.12 ± 0.09 0^{C}) from 6^{th} to 12^{th} hour was observed while an increased in pulse rate (from 74.80 ± 1.00 to 87.8 ± 2.58 beat/sec) and respiratory rate (from 20.40 ± 0.40 to 25.80 ± 0.73 beat/sec) 12^{th} hour respectively was observed during the experimental period.

iv. The level of Acetic acid concentration in the rumen increased from (939 ± 32.07mg/L to1938.72 ±212.07mg/L) between the 18^{th} hour to 24^{th} hour after feeding both whole and crushed maize grain

v. The sloughing off of the rumen mucosa may be due to the decreased rumen fluid pH (from 6.82 ± 0.03 to 5.12 ± 0.12) thus making the resultant acidic environment detrimental to the rumen mucosa.

6.2. RECOMMENDATIONS

1. High levels (75% and above) of 4% body weight for whole or crushed maize grain should not be given to sheep for what so ever reason.

2. Sheep should be reared under intensive farming system to avoid accidental excess consumption of any form of gain.

3. Regular review of feeding program and management practices is important to prevent occurrences of rumen lactic acidosis.

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APPENDICES

Appendix I Respiratory Rate, PulseRate andTemperature of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in the First Week

	Respiratory Rate(cycle/minute)			**Pulse Rate(beat/minute)**			**Temperature(^{0}C)**		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	20.6 ± 0.4^a	21.0 ± 0.95^a	20.7 ± 0.95^a	76.0 ± 1.04^a	79.8 ± 2.92^a	76.5 ± 1.19^a	39.04 ±0.20^a	39.22 ±0.20^a	39.2 ± 0.27^a
6	21.8 ± 0.49^a	22.0 ± 0.55^a	20.9 ± 0.75^a	78.0 ± 1.05^a	81.8 ± 2.92^a	76.5 ± 2.19^a	39.70 ±0.20^a	39 .98±0.20^a	39.2 ± 0.18^a
12	22.4 ± 0.68^a	23.0 ± 0.55^b	21.0 ± 0.50^a	80.8 ± 0.97^b	84.2 ± 2.71^b	77.5 ± 1.19^a	38.66 ±0.15^a	38.82 ±0.19^a	39.0 ± 0.17^a
18	23.8 ± 0.8^b	24.6 ± 0.68^b	21.75 ±0.75^a	80.5 ± 1.39^b	83.0 ± 1.05^b	78.5 ± 2.71^a	38.42 ±0.18^a	38.44 ±0.15^a	39.0 ± 0.15^a
24	25.0 ± 0.89^b	25.8 ± 0.73^b	21.95 ±0.75^a	85.0 ± 0.95^b	87.8 ± 2.58^b	79.0 ± 1.19^a	38.18 ±0.17^b	38.24 ±0.19^b	39.0 ± 0.18^a

^{a,b}Means in the same row with different superscripts are significantly different ($p < 0.05$)

Appendix II Respiratory Rate, PulseRate andTemperature of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in the second Week

	Respiratory rate (cycle/minute)			**Pulse rate (beat/minute)**			**Temperature(^{0}C)**		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	21.20 ± 0.20^a	21.00 ± 0.00^a	21.00 ± 0.30^a	75.20 ± 0.80^a	78.20 ± 3.10^a	75.00 ± 1.91^a	38.74 ± 0.24^a	39.12 ± 0.20^a	39.10 ± 0.23^a
6	21.40 ± 0.40^a	21.20 ± 0.37^a	21.20 ± 0.00^a	77.80 ± 0.97^a	80.40 ± 2.87^a	76.20 ± 1.71^a	39.60 ± 0.23^a	40.10 ± 0.19^a	39.00 ± 0.24^a
12	22.20 ± 0.58^b	22.20 ± 0.58^b	21.00 ± 0.20^a	80.20 ± 0.58^a	82.80 ± 2.71^a	77.00 ± 1.97^a	38.46 ± 0.22^a	36.98 ± 1.72^a	38.80 ± 0.22^a
18	23.60 ± 0.75^b	21.20 ± 0.37^b	21.20 ± 0.00^a	82.20 ± 0.80^b	84.80 ± 2.46^b	78.00 ± 2.10^a	38.24 ± 0.19^a	38.30 ± 0.10^a	38.72 ± 0.23^a
24	24.80 ± 0.80^b	25.20 ± 0.8^b	21.40 ± 0.40^a	83.20 ± 0.58^b	85.80 ± 1.80^b	78.20 ± 1.97^a	38.08 ± 0.08^b	38.12 ± 0.09^b	38.60 ± 0.23^a

^{a,b}Means in the same row with different superscripts are significantly different ($p < 0.05$)

Appendix III Respiratory Rate, PulseRate andTemperature of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in the Third Week

	Respiratory rate (cycle/minute)			**Pulse rate (beat/minute)**			**Temperature(^{0}C)**		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	20.40 ± 0.24^a	$20.80 \pm 0{,}20^a$	21.75 ± 0.37^a	74.80 ± 1.00^a	78.60 ± 2.78^a	75.00 ± 2.71^a	38.98 ± 0.19^a	39.22 ± 0.20^a	39.15 ± 0.18^a
6	21.20 ± 0.37^a	21.80 ± 0.05^a	21.45 ± 0.75^a	77.00 ± 0.89^a	80.20 ± 1.12^a	77.20 ± 0.91^a	39.80 ± 0.18^a	40.30 ± 0.12^a	38.98 ± 0.19^a
12	22.00 ± 0.45^b	22.80 ± 0.34^b	20.75 ± 0.24^a	79.40 ± 0.68^b	83.60 ± 2.71^b	77.60 ± 1.12^a	38.58 ± 0.17^a	38.70 ± 0.20^a	38.85 ± 0.20^a
18	22.80 ± 0.58^b	22.80 ± 0.48^b	20.40 ± 0.24^a	81.40 ± 0.40^b	86.00 ± 2.28^b	78.00 ± 2.91^a	38.32 ± 0.15^a	38.44 ± 0.18^a	38.90 ± 0.18^a
24	24.40 ± 0.81^b	24.75 ± 0.75^b	20.20 ± 0.58^a	82.40 ± 0.40^b	87.4 ± 1.81^b	78.60 ± 2.10^a	38.20 ± 0.10^b	38.16 ± 0.15^b	38.75 ± 0.15^a

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix IV Rumen pH of Yankasa Sheep Fedwhole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days

	Week 1			Week 2			Week 3		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	6.82 ± 0.03^{a}	6.72 ± 0.02^{a}	6.81 ± 0.03^{a}	6.80 ± 0.04^{a}	6.74 ± 0.02^{a}	6.78 ± 0.02^{a}	6.74 ± 0.02^{a}	6.73 ± 0.03^{a}	6.79 ± 0.02^{a}
2	6.70 ± 0.00^{a}	6.68 ± 0.02^{a}	6.79 ± 0.02^{a}	6.74 ± 0.02^{a}	6.72 ± 0.02^{a}	6.77 ± 0.03^{a}	6.72 ± 0.02^{a}	6.72 ± 0.02^{a}	6.78 ± 0.02^{a}
4	6.62 ± 0.02^{a}	5.59 ± 015^{b}	6.78 ± 0.02^{a}	6.66 ± 0.02^{b}	6.58 ± 0.02^{b}	6.76 ± 0.00^{a}	6.70 ± 0.01^{b}	6.66 ± 0.02^{b}	6.77 ± 0.03^{a}
6	5.96 ± 0.07^{b}	5.58 ± 0.02^{b}	6.77 ± 0.03^{a}	6.56 ± 0.02^{b}	6.34 ± 0.02^{b}	6.75 ± 0.03^{a}	6.66 ± 0.02^{b}	6.62 ± 0.02^{b}	6.76 ± 0.00^{a}
8	5.96 ± 0.07^{b}	5.70 ± 0.11^{b}	6.76 ± 0.00^{a}	6.36 ± 0.02^{b}	6.22 ± 0.02^{b}	6.75 ± 0.00^{a}	6.64 ± 0.02^{b}	6.40 ± 0.03^{b}	6.75 ± 0.03^{a}
10	5.74 ± 0.06^{b}	5.70 ± 0.11^{b}	6.75 ± 0.03^{a}	6.02 ± 0.02^{b}	5.92 ± 0.04^{b}	6.74 ± 0.03^{a}	6.58 ± 0.02^{b}	5.98 ± 0.07^{b}	6.75 ± 0.00^{a}
12	5.50 ± 0.03^{b}	5.58 ± 0.02^{b}	6.75 ± 0.00^{a}	5.66 ± 0.02^{b}	5.46 ± 0.04^{b}	6.73 ± 0.05^{a}	6.51 ± 0.04^{b}	$5.89. \pm 0.17^{b}$	6.74 ± 0.03^{a}
14	5.48 ± 0.09^{b}	5.50 ± 0.03^{b}	6.74 ± 0.03^{a}	5.54 ± 0.04^{b}	5.34 ± 0.07^{b}	6.73 ± 0.02^{a}	6.42 ± 0.02^{b}	5.44 ± 0.02^{b}	6.73 ± 0.05^{a}
16	5.46 ± 0.02^{b}	5.44 ± 0.02^{b}	6.73 ± 0.05^{a}	5.44 ± 0.02^{b}	5.34 ± 0.07^{b}	6.74 ± 0.03^{a}	5.96 ± 0.09^{b}	5.44 ± 0.02^{b}	6.73 ± 0.02^{a}
18	5.45 ± 0.06^{b}	5.44 ± 0.02^{b}	6.73 ± 0.02^{a}	5.36 ± 0.02^{b}	5.38 ± 0.10^{b}	6.73 ± 0.07^{a}	5.75 ± 0.20^{b}	5.43 ± 0.06^{b}	6.74 ± 0.03^{a}
20	5.44 ± 0.02^{b}	5.42 ± 0.05^{b}	6.74 ± 0.03^{a}	5.28 ± 0.05^{b}	5.6 ± 0.08^{b}	6.73 ± 0.03^{a}	5.66 ± 0.06^{b}	5.42 ± 0.05^{b}	6.73 ± 0.07^{a}
22	5.42 ± 0.03^{b}	5.38 ± 0.07^{b}	6.73 ± 0.07^{a}	5.26 ± 0.08^{b}	5.48 ± 0.05^{b}	6.73 ± 0.07^{a}	5.50 ± 0.05^{b}	5.40 ± 0.04^{b}	6.73 ± 0.03^{a}
24	5.38 ± 0.02^{b}	5.28 ± 0.07^{b}	6.73 ± 0.03^{a}	5.12 ± 0.12^{b}	5.60 ± 0.11^{b}	6.72 ± 0.03^{a}	5.42 ± 0.05^{b}	5.32 ± 0.07^{b}	6.73 ± 0.07^{a}

[a,b]Means for each week on the same row with different superscripts are significantly different ($p < 0.05$).

Appendix V Blood pH of Yankasa Sheep Fedwhole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days

	Week 1			Week 2			Week 3		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	7.46 ± 0.04^{a}	7.45 ± 0.04^{a}	7.47 ± 0.04^{a}	7.44 ± 0.02^{a}	7.42 ± 0.02^{a}	7.46 ± 0.02^{a}	7.38 ± 0.02^{a}	7.42 ± 0.02^{a}	7.47 ± 0.04^{a}
6	7.34 ± 0.02^{b}	7.36 ± 0.02^{b}	7.46 ± 0.02^{a}	7.34 ± 0.02^{b}	7.32 ± 0.02^{b}	7.44 ± 0.02^{a}	7.32 ± 0.02^{b}	7.30 ± 0.03^{b}	7.46 ± 0.02^{a}
12	7.22 ± 0.04^{b}	7.26 ± 0.02^{b}	7.44 ± 0.02^{a}	7.24 ± 0.02^{b}	7.12 ± 0.02^{b}	7.43 ± 0.04^{a}	7.30 ± 0.03^{b}	7.22 ± 0.02^{b}	7.44 ± 0.02^{a}
18	7.12 ± 0.04^{b}	7.16 ± 0.02^{b}	7.43 ± 0.04^{a}	7.12 ± 0.02^{b}	7.1 ± 0.00^{b}	7.42 ± 0.04^{a}	7.24 ± 0.02^{b}	7.14 ± 0.02^{b}	7.43 ± 0.04^{a}
24	7.04 ± 0.02^{b}	7.22 ± 0.02^{b}	7.42 ± 0.04^{a}	7.10 ± 0.00^{b}	7.12 ± 0.02^{a}	7.42 ± 0.02^{a}	7.71 ± 0.04^{b}	7.06 ± 0.02^{b}	7.42 ± 0.02^{a}

[a,b]Means for each week in the same row with different superscripts are significantly different ($p < 0.05$)

Appendix VI Acetic Acid (mg/L) of Yankasa Sheep Fedwhole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days

	Week 1			Week 2			Week 3		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	231.30 ± 18.29^{a}	147.56 ± 14.46^{a}	180.00 ± 30.97^{a}	428.02 ± 103.63^{a}	369.26 ± 78.49^{a}	319.80 ± 26.75^{a}	559.14 ± 128.43^{b}	628.36 ± 136.04^{b}	615.35 ± 99.47^{a}
2	241.06 ±51.65 ^{a}	224.46 ± 35.98^{a}	240.80 ± 23.85^{a}	359.54 ± 40.09^{a}	240.66 ± 32.07^{a}	272.12 ±106.77^{a}	605.90 ± 117.81^{b}	500.92 ±113.15^{b}	610.18 ±149.80^{a}
4	283.08 ±90.84 ^{a}	260.48 ± 42.05^{a}	262.93 ± 31.34^{a}	373.78 ± 49.86^{a}	333.52 ± 63.40^{a}	314.4 ± 36.71^{a}	611.00 ± 110.02^{b}	424.00 ± 45.65^{b}	654.00 ±113.47^{a}
6	347.64 ± 109.24^{a}	322.12 ± 83.52^{a}	171.40 ± 23.22^{a}	483.14 ± 124.15^{a}	452.66 ± 110.10^{a}	319.80 ± 26.73^{a}	444.70 ± 92.82^{b}	477.72±52.87^{b}	570.05± 160.46^{a}
8	825.04 ± 150.91^{b}	718.74 ± 162.09^{b}	226.20 ± 22.61^{a}	389.90 ± 83.98^{a}	427.50 ± 106.82^{a}	331.43 ± 101.72^{a}	302.42 ± 87.84^{b}	722.58 ±199.98^{b}	579.53 ±140.42^{a}
10	747.92 ± 61.81^{b}	492.92 ± 168.62^{b}	272.13 ± 26.54^{a}	306.56 ± 109.96^{a}	324.50 ± 86.36^{a}	345.2 0± 25.71^{a}	521.40 ± 66.48^{b}	470.06 ± 98.15^{b}	610.96 ± 97.49^{a}
12	677.90 ± 170.99^{b}	483.60 ±272.79^{b}	179.60 ± 30.96^{a}	242.34 ± 23.10^{a}	311.01± 40.41^{a}	319.80± 26.73^{a}	782.17 ± 160.27^{b}	785.54 ± 21.19^{b}	573.05± 160.47^{a}
14	612.66 ± 188.81^{b}	478.44 ± 228.99^{b}	165.00 ± 17.48^{a}	202.34 ± 23.10^{a}	260.14 ± 35.03^{a}	176.80 ± 12.93^{a}	596.02 ± 67.46^{b}	651.40 ±179.45^{b}	550.25± 113.64^{a}
16	815.74 ± 320.07^{b}	653.98 ± 224.53^{b}	347.08 ± 67.50^{a}	396.00 ± 135.77^{a}	378.04 ± 152.12^{a}	306.38 ± 7105^{a}	510.30 ± 61.85^{b}	446.68 ± 67.01^{b}	614.18± 149.80^{a}
18	936.48 ± 320.07^{b}	877.68 ± 319.33^{b}	199.53 ± 50.38^{a}	613.36 ± 246.78^{b}	576.66 ± 280.63^{b}	320.70 ± 26.73^{a}	820.06 ± 234.16^{b}	1352±338.24^{b}	620.10 ±133.73^{a}
20	952.30 ± 211.21^{b}	890.36 ± 30.50^{b}	226.20 ± 22.61^{a}	335.38 ± 139.00^{a}	477.82 ± 105.38^{a}	344.40± 96.11^{a}	1057.16±271.81^{b}	1450.7± 255.98^{b}	666.03± 150.37^{a}
22	724.84± 103.90^{b}	897.80 ± 260.39^{b}	223.20 ± 23.85^{a}	247.04 ± 50.53^{a}	551.64 ± 79.16^{b}	347.80 ± 14.85^{a}	1514.22±330.77^{b}	1495.70±686.94^{b}	617.15±149.80^{a}
24	830.56 ± 42.57^{b}	877.80 ± 240.87^{b}	221.30 ± 26.50^{a}	561.76 ± 72.03^{b}	630.32 ±137.19^{b}	390.43 ± 26.53^{a}	1016.08±373.19^{b}	1938.72±212.07^{b}	615.35± 99.46^{a}

^{a,b}Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix VII Lactic Acid (mg/L) of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days

	Week 1			Week 2			Week 3		
Hour	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	35.84 ± 0.36^{a}	36.84 ± 1.87^{a}	35.65 ± 0.54^{a}	45.28 ± 3.62^{b}	44.96 ± 2.61^{b}	35.36 ± 0.73^{a}	40.48 ± 2.46^{b}	42.34 ± 1.40^{b}	35.73 ± 2.13^{a}
2	34.66 ± 2.58^{a}	36.54 ± 2.24^{a}	35.95 ± 0.32^{a}	46.02 ± 2.83^{b}	48.68 ± 2.91^{b}	36.15 ± 0.36^{a}	36.72 ± 2.28^{a}	39.32 ± 0.75^{b}	35.36 ± 0.73^{a}
4	34.92 ± 0.97^{a}	33.62 ± 0.69^{a}	36.03 ± 0.33^{a}	46.70 ± 3.19^{b}	35.38 ± 2.14^{a}	36.15 ± 0.28^{a}	38.68 ± 1.32^{b}	38.42 ± 1.20^{b}	36.15 ± 0.36^{a}
6	33.98 ± 0.25^{a}	34.82 ± 0.36^{a}	36.10 ± 0.42^{a}	47.18 ± 2.90^{b}	46.82 ± 4.87^{b}	33.75 ± 0.73^{a}	39.60 ± 1.12^{b}	40.00 ± 1.70^{b}	35.34 ± 0.32^{a}
8	42.18 ± 2.34^{b}	44.36 ± 0.85^{b}	35.73 ± 2.13^{a}	49.76 ± 3.10^{b}	56.90 ± 1.23^{b}	34.93 ± 0.67^{a}	39.10 ± 1.07^{b}	35.00 ± 0.57^{a}	33.75 ± 0.73^{a}
10	44.60 ± 0.87^{b}	45.18 ± 3.49^{b}	35.36 ± 0.73^{a}	54.12 ± 3.13^{b}	53.94 ± 1.98^{b}	36.73 ± 2.24^{a}	35.86 ± 0.56^{a}	35.00 ± 0.47^{a}	34.93 ± 0.67^{a}
12	35.84 ± 2.93^{a}	41.72 ± 2.77^{b}	36.15 ± 0.36^{a}	54.94 ± 1.00^{b}	56.02 ± 1.12^{b}	36.58 ± 0.74^{a}	33.98 ± 1.09^{a}	34.82 ± 0.20^{a}	36.73 ± 2.24^{a}
14	39.66 ± 3.99^{b}	38.84 ± 3.37^{b}	36.15 ± 0.28^{a}	51.92 ± 4.23^{b}	57.67 ± 7.78^{b}	35.34 ± 0.32^{a}	34.92 ± 0.90^{a}	34.90 ± 0.63^{a}	36.58 ± 0.74^{a}
16	37.02 ± 0.64^{b}	33.68 ± 1.46^{a}	33.75 ± 0.73^{a}	48.52 ± 7.77^{b}	55.04 ± 4.90^{b}	35.65 ± 0.54^{a}	35.24 ± 1.41^{a}	35.48 ± 0.74^{a}	35.34 ± 0.32^{a}
18	39.84 ± 3.87^{b}	34.66 ± 2.58^{b}	34.93 ± 0.67^{a}	43.42 ± 2.59^{b}	44.34 ± 4.89^{b}	35.95 ± 0.32^{a}	36.04 ± 0.87^{a}	41.32 ± 3.49^{b}	35.65 ± 0.54^{a}
20	42.70 ± 1.63^{b}	39.44 ± 1.23^{b}	36.73 ± 2.24^{a}	52.94 ± 1.27^{b}	50.26 ± 1.32^{b}	36.03 ± 0.33^{a}	44.94 ± 2.46^{b}	43.26 ± 2.89^{b}	35.95 ± 0.32^{a}
22	43.78 ± 1.49^{b}	40.44 ± 0.71^{b}	36.58 ± 0.74^{a}	52.28 ± 0.73^{b}	52.74 ± 0.62^{b}	36.10 ± 0.42^{a}	50.73 ± 1.43^{b}	47.10 ± 3.09^{b}	36.03 ± 0.33^{a}
24	41.94 ± 1.15^{b}	44.70 ± 1.35^{b}	35.34 ± 0.32^{a}	53.64 ± 1.67^{b}	51.80 ± 0.81^{b}	35.73 ± 2.13^{a}	52.18 ± 1.55^{b}	52.84 ± 1.50^{b}	36.10 ± 0.42^{a}

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix VIII Rumen microbial Count (Earobic) (x10^3 CFU/ml) of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days

Hour	Week 1			Week 2			Week 3		
	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)	A (Whole grain)	B (Crushed grain)	C (*D. smutsii* hay)
0	4.16 ± 0.22^a	4.37 ± 0.05^a	4.17 ± 0.13^a	4.14 ± 0.22^a	4.27 ± 0.08^a	4.16 ± 0.19^a	4.16 ± 0.24^a	4.26 ± 0.03^a	4.17 ± 0.18^a
2	4.23 ± 0.13^a	4.15 ± 0.20^a	4.18 ± 0.19^a	4.22 ± 0.03^a	4.43 ± 0.13^b	4.15 ± 0.28^a	4.35 ± 0.10^a	4.27 ± 0.04^a	4.22 ± 0.10^a
4	4.40 ± 0.05^b	4.06 ± 0.19^a	4.20 ± 0.12^a	4.25 ± 0.25^a	4.46 ± 0.12^b	4.23 ± 0.13^a	4.40 ± 0.05^b	4.28 ± 0.05^a	4.19 ± 0.06^a
6	4.46 ± 0.12^b	4.02 ± 0.18^a	4.16 ± 0.19^a	4.27 ± 0.07^a	4.31 ± 0.05^a	4.18 ± 0.17^a	4.44 ± 0.08^b	$4.31 \pm 0,07^a$	4.17 ± 0.19^a
8	4.50 ± 0.08^b	4.30 ± 0.13^a	4.15 ± 0.28^a	4.35 ± 0.10^b	4.48 ± 0.06^b	4.21 ± 0.15^a	4.36 ± 0.12^b	4.37 ± 0.12^b	4.18 ± 0.17^a
10	$4\text{-}49 \pm 0.09^b$	4.58 ± 0.10^b	4.23 ± 0.13^a	4.46 ± 0.09^b	4.47 ± 0.07^b	4.17 ± 0.18^a	4.35 ± 0.10^b	4.35 ± 0.09^b	4.17 ± 0.13^a
12	4.09 ± 0.18^a	4.42 ± 0.16^b	4.18 ± 0.17^a	4.37 ± 0.06^b	4.51 ± 0.04^b	4.22 ± 0.10^a	$4.26 \pm 0,11^a$	4.36 ± 0.15^b	4.18 ± 0.19^a
14	4.04 ± 0.19^a	4.15 ± 0.06^a	4.21 ± 0.15^a	4.37 ± 0.11^b	4.47 ± 0.04^b	4.19 ± 0.06^a	4.36 ± 0.12^b	4.39 ± 0.13^b	4.20 ± 0.12^a
16	4.19 ± 0.19^a	4.19 ± 0.13^a	4.17 ± 0.18^a	4.39 ± 0.07^b	$4.35 \pm 0,12^b$	4.17 ± 0.19^a	4.57 ± 0.13^b	4.50 ± 0.11^b	4.16 ± 0.19^a
18	4.12 ± 0.13^a	4.32 ± 0.14^a	4.22 ± 0.10^a	4.24 ± 0.20^a	4.39 ± 0.15^b	4.18 ± 0.17^a	4.37 ± 0.16^b	4.30 ± 0.09^a	4.15 ± 0.28^a
20	4.45 ± 0.15^b	4.43 ± 0.15^b	4.19 ± 0.06^a	4.39 ± 0.07^b	4.34 ± 0.11^b	4.17 ± 0.13^a	4.33 ± 0.11^b	4.29 ± 0.08^a	4.23 ± 0.13^a
22	4.56 ± 0.13^b	4.61 ± 0.08^b	4.17 ± 0.19^a	4.41 ± 0.06^b	4.42 ± 0.12^b	4.18 ± 0.19^a	4.20 ± 0.03^b	4.20 ± 0.04^b	4.18 ± 0.17^a
24	4.49 ± 0.06^b	4.50 ± 0.08^b	4.18 ± 0.17^a	4.45 ± 0.11^b	4.57 ± 0.10^b	4.20 ± 0.12^a	4.17 ± 0.02^b	4.17 ± 0.02^b	4.21 ± 0.15^a

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix IX Haematology of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in First Week

Hour	Group	PCV (%)	HGB (%)	RBC (%)
0	A (Whole grain)	36.00 ± 2.79	11. 98 ± 0.93	7. 08 ± 1.12
	B (Crushed grain)	34.20 ± 4.26	11. 98 ± 0.93	5.90 ± 0.75
	C (*D. smutsii* hay)	35.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
6	A (Whole grain)	28.80 ± 2.27	9.58 ± 0.75	4.96 ± 0.48
	B (Crushed grain)	25.00 ± 3.27	8.30 ± 1.08	9.00 ± 4.27
	C (*D. smutsii* hay)	34.00 ± 2.42	11.88 ± 0.83	6.05 ± 0.40
12	A (Whole grain)	23.80 ± 5.02	13.84 ± 4.94	4.22 ± 0.74
	B (Crushed grain)	34.00 ± 5.43	11.32 ± 1.80	5.86 ± 0.79
	C (*D. smutsii* hay)	34.00 ± 2.28	11.48 ± 0.64	6.05 ± 0.40
18	A (Whole grain)	33.40 ± 5.24	11.08 ± 1.74	5.50 ± 0.85
	B (Crushed grain)	30.20 ± 3.77	10.04 ± 1.26	5.10 ± 0.66
	C (*D. smutsii* hay)	34.00 ± 2.38	11.78 ± 0.82	6.05 ± 0.40
24	A (Whole grain)	27.60. ± 2.46	9.24 ± 0.83	4.78 ± 0.40
	B (Crushed grain)	26.80 ± 3.06	9.10 ± 1.10	4.70 ± 0.50
	C (*D. smutsii* hay)	34.00 ± 2.27	11.68 ± 0.24	6.05 ± 0.40

There were no significantly different ($p < 0.05$)

Appendix X Haematology of Yankasa Sheep Fed whole or crushed Maize Grain and for 24 hrs and *Digitaria smutsii* hay for 6 days in First Week

Hour	Group	WBC (10^6/L)	NEUT (%)	LYMP (%)	MONO (%)	EOSIN (%)	BASO (%)	BAND (%)	TP (%)
0	A (Whole grain)	6.94 ± 0.93[a]	26.40 ± 4.23[a]	74.40 ± 3.25[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	6.36 ± 0.31[a]
	B (Crushed grain)	6.64 ± 0.69[a]	29.80 ± 5.18[a]	69.80 ± 5.35[a]	0.40 ± 0.40[a]	0.00 ± 0.00a	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.85 ± 3.66[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
6	A (Whole grain)	7.66 ± 0.42[a]	29.80 ± 2.20[a]	69.40 ± 1.99$_a$	0.80 ± 0.58[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.60 ± 0.30[a]
	B (Crushed grain)	10.18 ± 1.37[b]	28.80 ± 5.03[a]	68.60 ± 3.84[a]	0.20 ± 0.20[a]	1.60 ± 1.67[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	4.68 ± 0.37[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
12	A (Whole grain)	9.34 ± 2.20[a]	27.00 ± 4.17[a]	71.60 ± 4.13[a]	0.60 ± 0.40[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.60 ± 0.40[a]	6.28 ± 0.24[a]
	B (Crushed grain)	10.00 ± 2.30[b]	31.80 ± 3.18[a]	67.20 ± 3.40[a]	0.75 ± 0.75[a]	0.20 ± 0.20[a]	0.00 ± 0.00[a]	0.20 ± 0.20[a]	6.28 ± 0.39[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
18	A (Whole grain)	7.72 ± 0.98[a]	29.20 ± 3.48[a]	68.20 ± 3.58a	2.20 ± 1.56[b]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.40 ± 0.40[a]	6.48 ± 0.22[b]
	B (Crushed grain)	10.80 ± 2.06[b]	32.60 ± 3.79[a]	65.20 ± 3.77[a]	1.25 ± 0.95[b]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	1.50 ± 1.19[a]	5.92 ± 0.33[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
24	A (Whole grain)	11.64 ± 1.29[b]	27.20 ± 2.08[a]	69.60 ± 0.03[a]	2.00 ± 1.14[b]	0.40 ± 0.40[a]	0.00 ± 0.00[a]	1.25 ± 0.95[a]	7.28 ± 0.46[b]
	B (Crushed grain)	8.80 ± 1.69$_a$	22.20 ± 1.71[a]	75.20 ± 1.66[a]	1.40 ± 0.68[b]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.50 ± 0.96[a]	6.56 ± 0.20[b]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix XI Haematology of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in Second Week

Hour	Group	PCV(%)	HGB(%)	RBC(%)
	A (Whole grain)	35.80 ± 6.50	11.92 ± 2.16	6.46 ± 0.97
0	B (Crushed grain)	37.40 ± 2.77	12.42 ± 0.92	6.28 ± 0.45
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
	A (Whole grain)	33.00 ± 4.76	10.96 ± 1.56	5.54 ± 0.75
6	B (Crushed grain)	34.00 ± 3.51	11.32 ± 1.16	5.90 ± 0.60
	C (*D. smutsii* hay)	35.00 ± 2.48	11.68 ± 0.82	6.15 ± 0.42
	A (Whole grain)	32.40 ± 4.38	10.76 ± 1.43	5.50 ± 0.72
12	B (Crushed grain)	31.60 ± 3.83	10.50 ± 1.27	5.52 ± 0.65
	C (*D. smutsii* hay)	36.00 ± 2.31	11.60 ± 3.24	6.05 ± 0.20
	A (Whole grain)	33.40 ± 1.08	11.10 ± 0.37	5.54 ± 0.23
18	B (Crushed grain)	38.20 ± 3.44	12.70 ± 1.45	6.32 ± 0.57
	C (*D. smutsii* hay)	35.00 ± 2.24	11.48 ± 0.92	6.22 ± 0.40
	A (Whole grain)	33,80 ± 5.12	11.24 ± 1.72	5.64 ± 0.88
24	B (Crushed grain)	30.80 ± 1.16	10.24 ± 0.39	5.30 ± 0.29
	C (*D. smutsii* hay)	35.00 ± 2.48	11.62 ± 0.34	6.00 ± 0.22

There were no significantly different ($p < 0.05$)

Appendix XII Leucocyte levels in Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 days in Second Week

Hour	Group	WBC(10^6/L)	NEUT(%)	LYMP(%)	MONO(%)	EOSIN(%)	BASO(%)	BAND(%)	TP(%)
	A (Whole grain)	9.64 ± 1.45[a]	29.20 ± 4.54[a]	69.20 ± 4.45[a]	0.40 ± 0.40[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	1.20 ± 0.80[a]	6.04 ± 0.19[a]
0	B (Crushed grain)	8.72 ± 1.68[a]	34.40 ± 2.62[a]	63.80 ± 2.58[a]	0.60 ± 0,24[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	1.20 ± 1.20[a]	5.80 ± 0.41[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	9.12 ± 0.55[a]	28.20 ± 1.53[a]	70. 80 ± 1.83[a]	1.00 ± 0.55[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.72 ± 0.29[a]
6	B (Crushed grain)	10.48 ±1.48[a]	33.80 ± 2.03[a]	73.60 ± 2.10[a]	1.00 ± 0.55[a]	1.60 ± 1.60[a]	0.00 ± 0.00[a]	2.00 ± 1.26[a]	5.88 ± 0.22[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	10.00 ±1.81[a]	29.20 ± 3.22[a]	69.20 ± 3.67[a]	1.00 ± 0.55[a]	0.20 ± 0.20[a]	0.00 ± 0.00[a]	0.40 ± 0.40[a]	5.70 ± 0.33[a]
12	B (Crushed grain)	10.72 ±1,06[b]	29.60 ± 3.26[a]	67.40 ± 3.94[a]	0.80 ± 0.58[a]	1.20 ± 0.58[a]	0.00 ± 0.00[a]	0.60 ± 0.60[a]	5.56 ± 0.29[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	11.22 ±1.78[b]	26.80 ± 2.27[a]	72.60 ± 2.44[a]	0.40 ± 0.40[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.20 ± 0.20[a]	6.20 ± 0.17[a]
18	B (Crushed grain)	8.14 ± 1.08[a]	29.40 ± 2.48[a]	69.40 ± 2.79[a]	1.00 ± 0.55[a]	0.00 ±0.00[a]	0.00 ± 0.00[a]	0.20 ± 0.20[a]	5.88 ± 0.40[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	7.75 ± 1.63a	30.60 ± 3.31[a]	66.60 ± 2.71[a]	0.80 ± 0.37[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	6.04 ± 0.38[a]
24	B (Crushed grain)	13.04 ±1.69[b]	23.80 ± 3.61[a]	73.00 ± 3.38[a]	1.00 ± 0.55[a]	0.20 ± 0.20[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.80 ± 0,31[a]
	C (*D. smutsii* hay)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)

Appendix XIII Haematological values of Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 in Third Week

Hour	Group	PCV(%)	HGB(%)	RBC(%)
	A (Whole grain)	35.20 ± 3.15	11.70 ± 1.05	6.04 ± 0.56
0	B (Crushed grain)	37.20 ± 2.71	12.36 ± 0.90	6.40 ± 0.44
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
	A (Whole grain)	34.40 ± 4.45	11.32 ± 1.54	5.88 ± 0.74
6	B (Crushed grain)	36.20 ± 7.21	11.92 ± 2.29	6.06 ± 1.13
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
	A (Whole grain)	31.60 ± 1.21	10.50 ± 0.41	5.34 ± 0.24
12	B (Crushed grain)	28.80 ± 3.18	9.56 ± 1.06	4.82 ± 0.48
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
	A (Whole grain)	35.80 ± 3.17	11.90 ± 1.07	5.34 ± 0.18
18	B (Crushed grain)	30.00 ± 3.29	10.00 ± 1.10	5.28 ± 0.53
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40
	A (Whole grain)	39.69 ± 3.87	9.44 ± 0.64	6.62 ± 0.65
24	B (Crushed grain)	33.60 ± 2.40	9.20 ± 2.20	5.74 ± 0.31
	C (*D. smutsii* hay)	36.00 ± 2.48	11.98 ± 0.84	6.05 ± 0.40

There were no significantly different ($p < 0.05$)

Appendix XIV Leucocyte levels in Yankasa Sheep Fed whole or crushed Maize Grain for 24 hrs and *Digitaria smutsii* hay for 6 in Third Week

Hour	Group	WBC(10^6/L)	NEUT(%)	LYMP(%)	MONO(%)	EOSIN(%)	BASO(%)	BAND(%)	TP(%)
	A (Whole grain)	6.28 ± 0.15[a]	37.20 ± 1.71[a]	61.80 ± 1.80[a]	0.20 ± 0.20[a]	0,20 ± 0.20[a]	0.00 ± 0.00[a]	0.80 ± 0.80[a]	7.08 ± 0.29[a]
0	B (Crushed grain)	6.68 ± 0.90[a]	42.00 ± 2.61[b]	57.80 ± 2.50[a]	0.00 ± 0.00[a]	0.00 ± 0'00[a]	0.00 ± 0.00[a]	0.20 ± 0.20[a]	6.60 ± 0.23[a]
	C (Control)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	7.88 ± 0.51[a]	35.40 ± 4.20[a]	61.20 ± 3.61[a]	1.40 ± 0.60[a]	1.00 ± 0.63[a]	0.00 ± 0.00[a]	1.00 ± 0.45[a]	5.88 ± 0.32[a]
6	B (Crushed grain)	8.88 ± 0.63[a]	37.80 ± 2.84[a]	61.60 ± 2.79[a]	0.60 ± 0.40[a]	0.00 ± 0.00[a]	0.00 ±0.00[a]	0.00 ± 0.00[a]	5.84 ± 0.23[a]
	C (Control)	6.30± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	7.70 ± 0.56[a]	29.20 ± 3.43[a]	68.80 ± 3.60[a]	0.80 ± 0.53[a]	0.60 ± 0.60[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	6.28 ± 0.29[a]
12	B (Crushed grain)	9.16 ± 1.93[a]	28.60 ± 5.46[a]	54.92 ± 1.59[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.84 ± 0.16[a]
	C (Control)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	11.62 ± 1.63[b]	25.20 ± 3.12[a]	73.00 ± 3.00[a]	0.80 ± 0.49[a]	0.80 ± 0.80[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	6.76 ± 0.51[a]
18	B (Crushed grain)	8.20 ± 1.28[a]	28.00 ± 2.35[a]	69.80 ± 3.29[a]	1.00 ± 0.77[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	1.00 ± 1.00[a]	5.36 ± 0.37[a]
	C (Control)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]
	A (Whole grain)	8.36 ± 0.44[a]	33.00 ± 3,13a	66.60 ± 2. 90[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	0.00 ± 0.00[a]	5.00 ± 0.50[a]	0.71 ± 0.36[a]
24	B (Crushed grain)	10.52 ± 2.23[b]	35.60 ± 3.70[a]	62.60 ± 3.82[a]	1.40 ± 0.87[a]	0.20 ± 2.00[a]	0.00 ±0.00[a]	0.20 ± 0.20[a]	6.00 ± 0.13[a]
	C (Control)	6.30 ± 0.47[a]	37.75 ± 1.65[a]	60.25 ± 2.02[a]	0.50 ± 0.50[a]	0.50 ± 0.50[a]	0.00 ± 0.00[a]	1.00 ± 0.58[a]	6.15 ± 0.22[a]

[a,b]Means in the same rows with different superscripts are significantly different ($p < 0.05$)