Sudan University
Of Science and Technology
College of Graduate Studies

EFFECT OF VITAMIN E AND SELENIUM ON REPRODUCTIVE EFFICIENCY IN NUBIAN GOATS

أثر فيتامين E والسيلنيوم في الكفاءة التناسلية للماعز النوبي

By
AHMED ABDELGADIR AHMED SIDDIG

B.V.Sc. (1980), Alexandria University,
M. Sc. (1999), University of Khartoum.

A
Thesis Submitted in Accordance with the Requirements of the Sudan University of Science and Technology for the Degree of Doctor of Philosophy

Supervisor:

Prof. Abdelaziz Mekkawi A/Rahman Mohammed
Department of Animal Production, Faculty of Agricultural studies,
Sudan University of Science and Technology.

Department of Animal Production,
Faculty of Agricultural studies,
Sudan University of Science and Technology.
Jan.' 2014.

TO

Hanan

and her son

Tarik

and daughter

Hanin

A.A.A.Siddig

Jan' 2014

بسم الله الرحمن الرحيم

قال تعالى :

{قَالُواْ سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا
إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ }

صدق الله العظيم
سورة البقرة - الآية 32

وقال تعالى :

{وَإِنَّ لَكُمْ فِي الأَنْعَامِ لَعِبْرَةً نُّسْقِيكُمْ مِّمَّا فِي بُطُونِهَا
وَلَكُمْ فِيهَا مَنَافِعُ كَثِيرَةٌ وَمِنْهَا تَأْكُلُونَ}

صدق الله العظيم
سورة المؤمنون - الآيه(21)

Table of Contents

Item	Page

List of Tables

List of Figures

LIST OF ABBREVIATIONS

Abbrev.	Denotation	Abbrev.	Denotation
ALP	Alkaline phosphatase	**MCV**	Mean Corpuscular volume
ALT	Alanine amino transferase	**ME**	Metabolizable energy
BCG	Bromocresol green	**mg**	Milligram
BW	Body weight	**NMD**	Nutritional myodegeneration
C	Concentration	**PCV**	Packed Cell Volume
g/L	Gram per liter	**ppb**	Parts per billion
GSH-px	Glutehione perioxide	**ppm**	Parts per million
Hb	Hemoglobin	**RBC**	Red blood cell
IU	International unit	**S E**	Standard error
Kg	Kilogram	**Se**	Selenium
MCal	Mega Calorie	**U/L**	Units per liter
MCH	Mean Corpuscular Hemoglobin	**WBC**	White blood cell
MCHC	Mean Corpuscular Hemoglobin Concentration	**WMD**	White muscle disease

ACKNOWLEDGEMENTS

Thanks to God firstly and in conclusion, I wish to express my gratefullness, thanks and appreciation to my Supervisor **Prof. Abdel Aziz Makawi**, Department of Animal Production, Faculty of Agricultural Studies (Shambat), Sudan University of Scince and Technology, who spent valuable time in followup of this research and offered valuable directions, besides supplying the selenium test material. Also thanks are extended to **Prof. Ahmed El Amin,** Department of Pharmacology and Toicology, Faculty of Veterinary Medicine, University of Khartoum, who devoted his time and dedicated precious efforts in revising this research.

My thanks also go my colleagues in the Animal Production Department, Faculty of Agricultural Studies (Shambat), Sudan University of Scince and Technology, for their encouragement throughout this research. Also thanks to Uncle Alhajaa, for continuous labour attendance whilst expirmenttion.

My warmest and sincere gratitude also go to the Small Ruminant Department, Animal Production Corporation, Ministry of Animal Resources and Fisheries who supplied fodder during the period of experimentation, specially Dr. Almahy and Dr. Almuiz (wishes to Allah bless them). Thanks are also due to Miss Sawsan Mustafa, Khartoum Education Hospital Labrotary, who geniusly analyzed blood samples and serum, and thanks also offered to the Technicians, Shambat Central Labarotary, University of Khartoum, who analysed the selenium material.

Thanks are extended to engineer Badraldin the agricultural engineers union for this advice and finance to the research let Allah reward for precious advice,

thank to all colleagues at Animal Production complex, kuku, specially the A.I. family for alloy were the best helpers for transporting feeds and advices, thanks appreciations and regardes to all many. Allah repays best you best. Let Allah reward them for their precious advice.

Last but not least, my thanks go to Engineer Hajir Modawy who typed this manuscript in rapid and efficient manner.

Thanks are always due to Allah in commencement and close.

A. Abdelgadir

ABSTRACT

Selenium found in plants is ideally suited to the animal digestation and metabolism because it is in the form of selenium acids. Unfortunately, livestock producers have been forced to relyon inorganic selenium sources, such as selenite, in regions where siol and plants produce feed ingrients with low selenium content. In this study, two experiments were conducted to evaluate the effect of only selenium or synergized with vit E on the reproductive performance of Nubian goats. Twenty two non pregnant goats were selected and kept under indoor *ad lib*. forageing plus concentrate offered on daily basis at the rate of 1 kg per group. The goats were divided to six groups according to similar body weight basis. Three groups A, B and C constituting 5, 3 and 3 heads respectively were assigned for each experiment. Group A was designated as control, selenium free. Selenium premix wad dosed at 0 (devoid), 5 (low) and 10 (high) mg/kg (first experiment) or synergized with vitamin E 0+0 (devoid), 5+250(low) and 10+500 (high) mg/kg+ I.U./g (second experiment) twice weekly , fed in concentrate by dilution to groups A, B and C respectively . Goats' health was observed throughout the experimental period during gestation and delivery. Blood samples were analyzed every three weeks for RBCs count, Hb concentration, PCV, WBCs count and their differential lymphocytes. Sera were analyzed for metabolic indicators glucose, total protein, albumen, globulin and cholesterol, electrolytes Ca, P and Se and Serum enzymes ALT and ALP. Gestation start was assessed by palpation. On delivery, neonate sex, average birth and one month weights (kg) were recorded.

All experimental goats were healthy althrough the experimental period with normal voluntary feed intake, behavioural patterns and deliveries. Hematological findings show non significant ($p>0.05$) differences among all treatments and goups. Values of LS were higher than those of HS, but always higher ($P> 0.05$) than the control except for lymphocytes of HS (4.90±2.99). WBCs varied ($p>0.05$) with different treatments HsHv 17.74±5.05 followed by LsLv17.17±5.51, Ls 14.48±3.45 and Hs 13.42±5.2 when compared to the control (13.55±4.93). Little effect ($p>0.05$) was imposed on Hb in the diffenent treatments compared to the control. All serum metabolites values were not significant ($P>0.05$) compared to the control. The glocuse level was higher ($p>0.05$) for the treatments Hs (9.25±5.02) and LsLv (8.00±4.98). For total protein, Albumin and Globulin showed a better ($p>0.05$) reponse to the treatment with Hs (7.26±0.43), LsLv (3.83±0.49), and Hs (3.81±0.69) respectively compared to all other treatments. Cholesterol values were higher ($p>0.05$) for all treatments except with HS. Selenium concentrations, high and low correlates insignificantly ($p>0.05$) with total protein, but HsHv and LsLv correlates significantly ($p<0.05$, $p<0.01$ respectively) with total protein. Hs, HsHv and LsLv correlates significantly ($p<0.01$) with cholesterol whereas Ls correlates also significantly ($p<0.05$) with cholesterol. On regression of selenium, total protein and cholesterol, coefficients were only significant ($p<0.05$) in selenium for Hs and Ls concentrations.

For minerals Ca and P, results showed nonsignificant ($p>0.05$) mean treatment differences, though Ca showed a better response with HsHv (9.23±0.97). Synergized Se values 36.67±32.62 (HsHv) and 36.47±13.73 (LsLv) were the only significant ($p<0.05$) of the experimental groups. Activities of ALP and ALT were nonsignificant ($p>0.05$) when compared with the control. Avarge birth and one month weights (kg) of newborns were ascending in values for control, Ls and Hs, wheras same values were descending for control, LsLv and HsHv.

The effect of Se alone or synergized with vitamin E on goat reproduction in this study was not significant, deflating positive expectations. The reason was attributed to the effect imposed on bioavailability and absorption of Se by the quality and quantity of feed supplied.

المستخلص

السلينيوم نباتي المصدر هو المناسب للهضم والأيض في الحيوان لأنه في الصيغة الحمضية. ولسؤ الحظ فإن المنتجين اجبروا علي الإعتماد علي مصادر السلينيوم غير العضوية كالسلينيتات في مناطق تعطي فيها التربة والنبات مصادر علفية متدنية محتوى السلينيوم. في هذه الدراسة ، اجريت تجربتان فى الماعز النوبى لدراسة تأثير عنصر السيلنيوم منفرداً أو متآزراً مع فيتمين هـ علي الأداء التكاثري. تم إختيار عدد 22 ماعز انثى غير حامل غذيت في الحظائر بمالىء بحرية علاوة علي مركز يعلف يومياً بمعدل واحد كيلوجرام للمجموعة. قسمت الماعز الى ست مجموعات مؤسسة علي التشابة في وزن الجسم. ثلاث مجموعات منها أ ، ب ، ج تحتوى كل مجموعة علي 3،3،6 رؤوس علي التوالي خصصت لكل تجربة. حيوانات المجموعة أ هى المجموعة المرجعية الخالية من السيلنيوم. غذي مخلوط السلينيوم بمستوى صفر (خالي)، 5 (منخفض) و 10 (عالي) ملليجرام/ كجم (التجربة الأولي) أو متآزراً مع فيتمين هـ صفر+ صفر (خالي)، 5+250 (منخفض) و 10+ 500 (عالي) ملليجرام/ كجم +وحدة دولية/جم (التجربة الثانية) مرتين اسبوعيا، مخلوطاً في المركز بالتخفيف للمجموعات أ ، ب ، ج علي التوالي. لوحظت صحة الماعز طوال فترة التجربة خلال الحمل والولادة. عينات الدم اخذت كل ثلاثة أسابيع وحللت لعددية الكريات الحمراء ، تركيز الهمجلوبين، حجم الخلايا المتراص، عددية الكريات البيضاء وتفريقها للخلايا اللمفية. تم تحليل المصل للمؤشرات الأيضية الجلوكوز، البروتين الكلي، الألبيومين، القلوبيولين والكوليستيرول، الكهارل الكالسيوم، الفوسور والسلينيوم، والإنزيمات ناقل ايمين الألانين والفوسفتيز القلوي. تم تقييم الحمل بالتحسس. عند الولادة، يتم تسجيل جنس المولود، متوسط وزن (كجم) الولادة وبعد شهر منها.

كانت الماعز جيدة الصحة خلال فترة التجربة ومأكولها الطوعى طبيعى ، نمط تصرفاتها وولاد اتها . أظهرت نتائج الدم فروقات غير معنوية (أ > 0.05) بين المجموعات فى التجربتين ، قيم السلينيوم المنخفض كانت أعلى من السلنيوم العالى لكنها دائماً أعلى (أ > 0.05) من المجموعه المرجعية فيما عدا الخلايا اللمفية للسنيوم العالى (4.90 ± 2.99). الكريات البيضاء تغيرت (أ > 0.05) مع المعاملات ، سلنيوم عالى وفتمين عالى 17.74 ± 5,05 يتبعه سلنيوم منخفض فتمين منخفض 17.17 ± 5.51 ، سلينيوم منخفض 14.48 ± 3.45 وسلنيوم عالى 13.4 ± 5.20 عند مقارنتها بالمجموعة المرجعية (13.55 ± 4.93). أثر ضئيل (أ > 0.05) حدث على الهمجلوبين فى المعاملات المختلفة مقارناً بالمجموعة المرجعية. لم تكن قيم المستقلبات فى المصل ذات دلالة (أ > 0.05) مقارنة بالمجموعة المرجعية . كان مستوى الجلوكوز عالياً (أ > 0.05) فى المعاملات سلنيوم عالى (9.25± 5.02) وسلنيوم منخفض (4.98 ± 8.00) والبروتين الكلى ، الألبيومين و القلوبيولين أظهروا أحسن (أ > 0.05) رد فعل فى المعاملات سلنيوم عالى (7.26 ± 0.43) ، سلنيوم منخفض وفتمين منخفض (3.83 ± 0.49) وسلنيوم عالى (3.81 ± 0.69) على التوالى مقارنين بكل المعاملات الأخرى . كانت قيم الكلوليسترول أعلى (أ > 0.05) لكل المعاملات ماعدا السلنيوم العالى . تركيزات السلنيوم العالية والمنخفضة أرتبطت إرتباطاً غير معنوى (أ > 0.05) مع البروتين الكلى ولكن السلنيوم العالى والفتمين العالى والسلنيوم المنخفض والفتمين المنخفض إرتباطاً معنوياً (أ > 0.05 ، أ > 0.01 على التوالى) مع البروتين الكلى . السلنيوم العالى ، السلنيوم العالى والفتمين العالى والسلنيوم المنخفض والفتمين المنخفض إرتبطت معنوياً (أ > 0.01) مع الكولسيترول بينما السلينيوم المنخفض ارتبط أيضاً معنوياً (أ > 0.05) مع الكوليستيرول. عند تحليل إنحدار السلنيوم ، البروتين الكلى والكوليستيرول ، فإن معاملات الإنحدار كانت معنوية (أ > 0.05) فقط فى السلنيوم فى التركيزات العالية والمنخفضة . بالنسبة لأملاح الكالسيوم والفسفور ، أظهرت النتائج فروقات متوسطات غير معنوية (أ > 0.05) مع أن الكالسيوم أظهر ردة فعل حسنة مع السلنيوم العالى والفتمين العالى (9.23 ± 0.97) . قيم السلنيوم المتآزر 36.67± 32.62 (سلنيوم عالى وفتمين عالى) و 36.47 ± 13.73 (سلنيوم منخفض وفتمين منخفض) كانت هذه الوحيدة الدلالة المعنوية (أ > 0.05) بين مجموعات التجربة . نشاط أنزيمى الفوسفتيز القلوى وناقل ايمن الألانين كان غير معنوى (أ > 0.05) الدلالة ع ند مقارنته بالمجموعة المرجعية . كان موسط وزن (كجم) الولادات وبعد شهر منه متصاعدة القيم للمجموعة المرجعية ، السلنيوم الم نخفض ثم السلنيوم العالى على التوالى بينما نفس القيم متنازلة للمجموعة المرجعية ، السلنيوم المنخفض والفتمين المنخفض ثم السلنيوم العالى والفتمين العالى على التوالى أيضاً .

كان أثر عنصر السلنيوم منفرداً أو متآزراً مع فيتمين هـ علي التكاثر في الماعز في هذه التجربة غير ذي دلالة، محبطاً للتوقعات الإيجابية. ارجع السبب للأثر الواقع علي التوافر البيولوجي وإمتصاص السلنيوم من كم وكيف العلايق المستخدمة.

INTRODUCTION

Selenium found in plants is ideally suited to animal digestion and metabolism because it is in the form of selenoamino acids. Unfortunately, livestock producers have been forced to rely on inorganic selenium sources, such as sodium selenite, in regions where soil and plants produce feed ingredients with low selenium content. Replacing the natural organic selenium is often unsuccessful. Despite inorganic supplementation, clinical and sub clinical selenium deficiencies remain widespread. Low selenium (**Se**) in soil and forages combined with difficulty in supplementing growing cattle on pasture often result in inadequate blood **Se** as the growing heifer approaches calving. Selenium fertilization is effective during the grazing season; however changes in availability occur due to factors such as time post application and the change from pasture to hay and silage as feed sources. An additional disadvantage of this method is the toxicity of the material and danger of over-application. The purpose of this field investigation was to compare blood **Se** levels in animals on **Se** -fertilized pasture with those received either injected **Se** or **Se** in the form of selenium yeast.

This element was considered primarily as a toxic element. The first reports of the effects of **Se** deficiency in ruminants are the studies with calves **(Muth, *et al.*, 1957)** and lambs **(Hogue, 1958)** demonstrating that **Se** prevented nutritional muscular dystrophy. Following these studies, there have been numerous studies showing the benefits of **Se** along with vitamin **E** in number of health related aspects of diary cattle and other species. Indeed, most of the selenium deficiency syndromes are (at least partially) responsive to an elevated supply of the antioxidant micronutrient vitamin **E**. thus the descriptive term selenium/ vitamin **E** deficiency symptoms might be more appropriate. Over selenium deficiency is a relatively rare finding in livestock due to the widespread use of dietary supplementation of selenium with inorganic and organic selenium sources. In the area of human health, knowledge about selenium deficiency symptoms has only been derived from

observation of naturally occurring selenium sources deficiency because unlike in animals, selenium deficiency can not be experimentally induced in human for ethical reasons. Most emphasis regarding selenium nutrition in humans has been directed to possible anticarcinorgenic effects. It should be emphasized, however, that these effects are only observed at supro physiological doses of selenium, i.e. at levels well above the nutritional requirement.

Selenium in forages and seed grains is normally present as organic selenium in the form of *selenocystine*, *selenocysteine*, and *selenomethionine*, shows that organic plant source of selenium are more potent than inorganic **(Frape, 1998).** New born ruminants are dependent upon the dam for selenium transfer via the placenta or mammary gland. Because feeds grown in many areas of the world are deficient in selenium for livestock, selenium supplementation is often necessary. Methods that have been used for selenium supplementation include injection, drenching, and administering selenium. Iaden boluses and trace minerally salt mixes. Inorganic selenium salts, primarily sodium selenite, are generally used in selenium supplements. Organic selenium sources, such as plants and selenium yeast (**sel-plex**) contain seleno amino acids that are beneficial as selenium supplements. Differences exist in the utilization of dietary selenium from the various chemical forms. Selenium in sodium selenate can be chemically reduced to insoluble form by rumen microorganisms, thus lowering selenium absorption.

Selenium has long been recognized as an essential trace element in livestock nutrition. Its function as a part of the chief intracellular antioxidant enzyme glutathione peroxides make selenium, along with vitamin **E**, important in reproduction and immunity along with a wide range of metabolic and digestive roles. Selenium deficiency in the dairy cow is often seen as retained placenta and reproductive failures of serious kinds calves born to deficient has an effect on milk production, but this effect may be blamed on other nutritional or environmental problems. One method by when selenium needs can be more adequately met is by

supplying an organic source of selenium in the diet. The importance of an adequate selenium supply to our livestock cannot be overemphasized. Selenium supplementation of low-selenium feed not only prevents deficiency symptoms in animals but also substantially contributes to the selenium intake of humans through animal-derived products such as meat and eggs. Before selenium was recognized as an essential micronutrient, its toxic properties were well known. Indeed, there is a relatively small range between the beneficial effects of selenium and selenium toxicity.

The goat is one of smallest domesticated ruminant which have served mankind earlier and longer than cattle and sheep. It is managed for the production of milk, meat, and wool, particularly in the arid, semitropical and mountainous regions of the world, such arears are poor for their carrying capacitys quality and quantity wise and deficient in most of the microelements particularly selenium and hence affecting the production and reproduction efficiency of the animals living in those areas particularly goats.

The purpose of this sutdy is to essess the effect of inorganic selenium (**sel-plex 50**) supplementation on health, metabolism and some reproductive traits. Parameters used for achieving this objective are synchronization of estrus, conception rate, gestation period, kidding rate, litter size weight, health observations, blood cells and metabolites.

CHAPTER ONE

LITERATURE REVIEW

1. Goat classification

1.1. Types of goats in Sudan

Goat population in Sudan is estimated to be 32.1million heads **(Ministry of Animal Resource, 2011).** There are breeds of goats distributed all over the country e.g. Nubian, Desert. Nilotic and Tigeri goats. Some other foreign breeds were introduced to the country resulting in many crossed.

1.2. Sudanese Nubian goats

This breed was developed along the Nile Valley of southern Egypt (Wawat) and northern Sudan (Kush). It is a dairy type goat characterized by fairly proportioned body size with small to medium size head, convex facial profile and large drooping ears usually turned out at the lower tips. The back is long and straight, the legs are long, strong and well proportioned. The udder is large and well shaped **(El Naim, 1979).**

1.3. General reproductive performance of goats

Puberty in females is defined as the age at the first expressed estrus with ovulation; it should not be considered sexual maturity **(Bearden and Fuquay, 1984).** The prepubertal females to respond to the pulsatile secretion of gonadotropins by gradually secreting estrogen, which is associated with behavioral estrus **(Hafez and Hafez, 2000). Hunter (1980)** indicated that the direct cause of sexual maturation at puberty is the secretion of pituitary hormones. The age of puberty is influenced by physical environment,

photoperiod, age and breed of dam, breed of sire, environmental temperature, and body weight as affected by nutrition and growth rates before and weaning **(Hafez and Hafez, 2000).** In seasonal breeders, the age of puberty depends on the birth season **(Hafez and Hafez, 2000).**

1.4. Estrous Cycle

Estrous is rhythmic behavior pattern that develops female animals during puberty **(MeDonald, 1975).** The repeating chain of events leading to regular estrus periods is called the estrus cycle. The estrus cycle is divided into two phases, the follicular phase and the luteal phase; estrus occurs during the later stages of the follicular phase. The follicular phase is 3-4 days and the luteal phase occupies the rest of the cycle (about 17 days) **(Evans and Maxwell, 1987).** Estrus is that time during which the female will accept the services of the male. This period is cyclic in nature and the length of estrus is species dependent and varies from one breed to another within the same species **(Hafez and Hafez, 2000).** It is brought about by estrogen, the female excitalory hormone **(Sorensen, 1979).** The ovum is shed soon thereafter. Throughout the cycle the various parts of the female reproductive tract undergo changes affected by pituitary and ovarian hormones. In addition to initiate the period of receptivity to the male, these hormones condition the reproductive tract to receive the sperm, producing eggs and nourishment of the embryo and fetus **(Salisbury *et al*,. 1978).** Estrus is the fertile period of the cycle and if the female doesn't conceive, it is repeated every 19-21 days (normal range 17-24) (8-10%) of some breeds of goats have short cycles of (6-1 days) **(Evans and Maxwel, 1987).** It may last for 48 hours (Hafez and Hafez, 2000). In the tropics the mean duration of estrus period ranges from 17-48hrs **(Devendra and Mcleroy, 1982).** Breed, age, season and presence of male influence the duration of estrus **(Hafez and Hafez, 2000).**

1.5. **Ovulation**

In goats it's spontaneous, which occurs regardless of whether or not mating takes place **(Evans and Maxwel, 1987).** Ovulation is the process, in which the mature Graafian follicle ruptures and releases the ovum, it occurs at the end of estrus followed by corpus luteum formation resulting in progesterone secretion **(Hafez and Hafez, 2000).** Growth maturation, ovulation, and luteirization of the Graafian follicle depend on appropriate patterns of secretion, sufficient concentrations, and adequate ratio of FSH and LH in the serum **(Hafez and Hafez, 2000).**

1.6. Ovulation time

The time of ovulation is related to the end of the estrus phase than to its beginning **(McDonald, 1975; Evans and Maxwel, 1987),** the time of ovulation varies is relation to internal and external factors **(Hafez and Hafez, 2000).** Accurate knowledge of the time of ovulation is crucial to the success of insemination **(Evans and Maxwel, 1987; Ritat *et al*,. 1984).** Most goat breeds ovulate between 24-36 hours after the onset of estrus **(Hafez and Hafez, 2000).**

1.7. Ovulation rate

Ovulation rate partly determines the number of offspring that a female will carry **(Evans and Maxwel, 1987).** Ovulation rate is determined by the number of follicles which develop to the graafian stage. There are a number of factors, which influence the ovulation rate; these include genetic factors, live weight, nutritional status, age of animal, and season **(Evans and Maxwel, 1987).** Ovulation rate increases with age and reaches peak at 3-5 years **(Evans and Maxwel, 1987).**

1.8. **Selenium**

Selenium (Se) is a source of microminaral feed supplement. It is an organic substances required by animals in very small amounts for regulating various body processes towards normal health, growth, production and reproduction **(Varly, 1967).**

In the area of human health, knowledge about selenium deficiency symptoms has only been derived from observations of naturally occruing selenium deficiency. This because of ethical reasons. Most emphasis regarding selenium nutrition in humans has been directed to possible anti carcinogenic effects. It should be emphasized, however, that these effects are only observed at supraphysiological doses of selenium, i.e. at levels well above the nutritional requirement. However, it cannot be excluded that a preventive effect of selenium against certain types of cancer may even be present at a nutritionally appropriate selenium intake, although considerable experimental work has been done on the anti carcinogenic functions of selenium in humans during recent years. From a nutritional point of view, the inorganic forms selenate and selenite and organic forms selenomethionine and selenocysteine might be the most important chemical forms of selenium **(Varly, 1967).** In seleno-amino acids, the sulfur of the corresponding sulfur containing amino acids is simply replaced by selenium during synthesis **(Ndibualonji *et al.*, 1997).** Whereas most plant –and animals- derived by feed and food from natural sources contain organic forms of selenium, e.g. selenoamino acids, the inorganic selenium salts (selenite, selenate) are commonly used for supplementation of livestock rations as cheap and efficient means to prevent selenium deficiency **(Varly, 1967).** However, during recent years organic forms of selenium in the form of selenium –enriched yeast have become available to farmers. It has been assumed that in yeasts cultured in high-selenium medium the most abundant chemical form of selenium may be selenomethionine. However, recent findings with improved

analytical methods indicate that a wide variety of seleno compounds, which are unidentified, are present in selenium yeast **(Awadeh *et al.*, 1998).** Thus the selenium yeast actually contains a cocktail of selenium in a variety of chemical forms. It should be further mentioned in this context, that the specific chemical forms of selenium in most food and feed are unknown.

Now adays the use of Se in animal food stuff is well implicated and needs further investigation of its metabolism**.** According to the previous comperative studies, it appears that different livestock spp. show different metabolic profiles of serum Se levels and glutehione perioxide (GSH-px) activity (**Seboussi *et al.*, 2009)**.

1.8.1. Bioavailability of selenium

The term bioavailability has been used to describe different phenomena ranging from the disappearance of a substance from the contents of the gastrointestinal tract, i.e. apparent digestibility, to the accumulation within several organs or the synthesis of a certain enzyme. Bioavailability may be best delined as the part of selenium absorbed from the gastrointestinal tract which is metabolically available for the maintenance of the normal structures and physiological processes of an organism under delined conditions **(Varly, 1967).** The view that measuring tissue accumulation of selenium, e. g., in muscle or liver, without the addition measurement of a functional parameter of selenium, e. g., the activity of glutathione peroxidase, does not sufficiently describe bioavailability of selenium. This dose not means that there is no correlation between tissue accumulation, enzyme activity, and selenium status. It has been repeatedly demonstrated that a good correlation exists between these variables in situations of deficient or marginal selenium supply. **Waghron (1986)** the amount and chemical from of the element ingested with feed or food, solubilization within gastrointestinal contents, the physiological state of organism (e.g., growth, pregnency), the bioavailability and methionince diseases, drugs and age. The fellowing sections will deat with two major aspects

regarding the bioavailability of the trace element selenium, mainly intestinal absorption and post absorptive metabolism **(Butler *et al.* 1989).**

1.8.2. Intestinal absorption of selenium

The disappearance of chemically delined selenium compounds, e.g., selenite or selenomethionine, form the gastrointestinal tract have shown efficient absorption of inorganic (selenate, selenite) as well as organic (selenoamino acid) forms of selenium. **Thomson** and **Stewart.(1973)** investigations on the absorption of selenium from natural animal sources, e. g. kidneys of rabbits or fish muscle from fish fed labeled selenite on selenomethionine, showed that 87% of the selenium from the kidneys of rabbits was absorbed, whereas selenium absorption from fish reached only 72-77% of applied dose (**Richold *et al.*, 1977). Butler, *et al.* (1989)** incorporated into rat diets muscle, liver and hemoglobin from sheep which had been fed high selenium diet (1 mg Se/kg). Selenium as either selenic or L-selenomethionine were used as standards. Tissue selenium levels and GSH-px activities were used to assess bioavailability, selenium was found to be more available from muscle than liver or hemoglobin **(Butler *et al.*, 1989).** Absorption of selenium in ruminants appears to be less efficient compared with monogastric animals. This might be attributed to that selenium can form insoluble metal selenides or ca be reduced to elementary selenium within the content of the forestomachs, thus rendering selenium less available for absorption **(Preston** and **Spedding, 1963** and **Wright and Bell, 1966).** In sheep, 66-69% of an orally applied dose of selenium in the form of the inorganic salts (selenite and selenite) were excreted with the feces **(Wright and Bell, 1960** and **Paulson *et al.*, 1966)**, whereas a somewhat lower value (54%) was found for organic bound selenium (selenium-rich red clover) **(Preston** and **Spedding, 1963)** determined effects of diet composition and chemical form of selenium on intestinal flow, absorption and retention of selenium in sheep. Animals were fed forage (alfalfa hag) based- (0.37 mg se /kg) or concentrate (barley) - based (0.27 mg se /kg) diet at 90% of ad libitum

intake and stable isotopes of selenium (enriched 77se yeast, enriched 82se. selenite) were administered into the rumen. A larger proportion (51-61%) of the selenium tracers flowing to the duodenum was associated with the fluid fraction, mainly as bacteria- associated selenium, than with the fluid fraction. The 82se. selenite was more available for absorption and retention than 77se- yeast, indicating than inorganic chemical forms of selenium are as available to the ruminant as organic forms of selenium commonly found in feedstuffs **(Koenig *et al.*, 1997).**

Selenium absorption and retention were greater in sheep receiving the concentrate –based diet than in sheep receiving the forage based diet. Thus, the availability of selenium from inorganic and organic sources in sheep seems to be influenced by diet composition **(Koenig *et al.*, 1997).**

Taken together, inorganic as well as organic selenium compounds present either in feed and food or used for selenium supplementation seem to be reasonably well absorbed. Thus gastrointestinal absorption does not limit bioavailability of selenium. Furthermore, in contrast to other trace elements like iron or zinc, absorption of selenium appears not to be influenced by selenium status indicating that intestinal absorption does not play a role in whole body selenium homeostasis **(Venderland *et al.*, 1992).**

1.8.3. Selenate

The presence of sodium clearly enhanced mucosal uptake of selenate but not of selenium from selenite, the effect was most pronounced in the ileum **(Ardüser *et al.*, 1986).**

Using isolated brush-border membrane vesicles from different species (rat, sheep, and pig) the nature of active selenate transport was further unraveled **(Wolffrana, *et al.*, 1986 - 1988). Ardüser.(1985-1986**) who found inhibition of sodium- dependent selenate uptake across the intestinal brush-border in rat and sheep by thiosulfate, sulfate, chromate and tungstate as already mentioned above

(Wolffrana, *et al.*, 1986 - 1988). Membrane vesicles that selenate and sulfate share common carrier-mediated mechanisms to cross the intestinal brush-border membrane. It should be mentioned, however, that despite the fact that some competition for common transport mechanisms might exist between selenate and sulfate and some other metal oxides, these findings seem to be of minor practical importance because an influence on the absorption of selenium from selenate might be expected only if toxic level of these substances are ingested.

1.8.4. Selenite

Selenate, intestinal absorption of selenite per se is sodium-independent and seems to occur by simple diffusion **(Wolffrana, *et al.*, 1986).**; **Mykkänen and Wasserman 1989).** Furthermore, absorption of selenium from selenite is not influence by sulfate and other physically and chemically related oxyanions **(Ardüser *et al.*, 1986).** Reactions of selenite with intracellular thiols may even enhance diffusive uptake of selenium from selenite by maintaining a chemical gradient for selenite direction into the cells. The most abundant intracellular thiol in mammalian cells is the tripeptide glutathione which may occur at concentration of 0.5-10 mmol/ 1 **(Meister** and **Anderson, 1983; Bary and Taylor, 1993; Kelly, 1993).** Indeed, **Anundi *et al.* (1984)** described a rapid decrease of intracellular reduced glutathione in rat enterocytes incubated with selenite. Furthermore, oxidation of intracellular glutathione by the oxidant diethyllmaleate prior to incubation with selenite, significantly inhibited uptake of selenium from selenite **(Anundi *et al.*, 1984). Ardüser.(1986)** also indicated a function of intracellular glutathione in the uptake of selenium from selenite. Pre-incubation of mucosal sheets of red ileum in a glutathione-containing medium stimulated selenium uptake from selenite. Not only intracellular thiols, but also extra cellular thiols present in intestinal lumen influence absorption of selenium from selenite.

1.8.5. Seleno amino acids

In feed and food from natural sources, a substantial amount of total selenium is present as a free or peptide-bound seleno amino acids e g. selenomethionine, slenocystine and selenocysteine. **Milford and Minson (1966)** demonstrated active transport of L- selenomethionine but not of selenocysteine across the intestinal wall of the hamster. They described mutual inihibition between methionine and selenomethionine.

1.9. Metabolism of selenium

Organic selenium compounds e.g. selenomethionine or selenium yeast, are more bioavaible than inorganic selenium sources e. g. selenate and selenite, in terms of tissue levels of selenium, including meat and milk (**Yeh *et al.*, 1997). Clariget *et al.* (1998)** furthermore, stated that selenium concentrations in blood and other tissues as well as glutathione peroxidase activity are maintained for a longer period upon selenium depletion if animals had previously been supplied with organic compounds compared with inorganic sources of selenium. On the other hand, the inorganic selenium compounds are equally effective or may be even more effective than organic compounds with respect to elevation or maintenance of the activity of selenoprotins **(Yeh *et al.* 1997; Hogue, 1985; AOAD, 1990; Nolan, 1993; Mahan and Kim, 1996).** However, as already mentioned, measurement of tissue selenium levels without an appropriate measurement of a biologically active principle of selenium, e. g. glutathione peroxidase, is not fully suitable for the evaluation of the bioavailability of selenium under various conditions **(Cantor, 1997).** It should be pointed out that two major pools of selenium appear to exist in the body, which is differentially supplied by inorganic selenium compounds ingested with the diet **(Darrag, 1994: Jagusch, *et al.*, 1983).** One of the pools, termed the exchangeable metabolic pool, incorporates intermediary products that appear during reduction of selenate/selenite, endogenously synthesized selenoproteins as well as compounds

derived from methylatoin of selenide, e.g. methylslenol, dimethylselenide or trimethylselenonium. This exchangeable metabolic pool can also be entered by selenoamino acids. This pool provides metabolism and synthesis of the functionally important seleno- compounds **(Darrag, 1994).** The second pool comprises all selenomethionine- containing protein but has no known function other than perhaps to contribute to selenium stores (**Darrag, 1994**). Because selenomethionine is a ready substrate for the enzymes which use methionine, selenomethionine is a none specific substitute for methionine in a large numer of protein (especially skeletal muscle protein) particularly when large doses of selenomethionine are supplied **(Shelton, 1990; Darrag, 1994; Bello 1985 and Butler *et al.*, 1989).** The extent of unspecific incorporation of Selenomethionine instead of methionine in protein depends on the dietary supply of methionine. The percentage of ingested selenium in the form of selenomethionine immediately available for synthesis of functional selenoprotiens may be reduced **(Butler *et al.*, 1989).** Selenocystiene can also be incorporated into proteins instead of cysteine, but can not be directly incorporated into specific selenoprotiens **(Shelton 1990 and Darrag, 1994).** Selenomethionone can be metabolized to selenocysteine analogous to the metabolism of methionine to cysteine via the methionine tarns sulfuration pathway. Alternatively, methylselenol might be directly released from selennomethionine in animals also by an L-methionine-y- lyase**.** Selenocysteine does not seem to follow the pathway of cysteine metabolism with oxidative release of selenite instead a selenocysteine-specific enzyme, selenocysteine- B-lyase, directly releases selenide in the presence of reducing substances **(Shelton 1990 and Burk, 1991).** Selenate may be reduced to selenite which may be further reduced to selenide by means of glutathione and glutathione reductase **(Graham and Searle 1975 and Green, 1999).** With respect to the incorporation as well as the excretion of selenium, selenide may hold a central role in selenium metabolism. For excretion, selenide is methylated via methylselenol to dimethylselenide and trimethylselenonium **(ILRI, 1996; Shelton,**

1990; Green, 1986 and Lawrence and Pearce, 1964). Demethylation of trimethyl selenium and dimethylselenide appears also to be possible **(ILRI, 1996).** Although organic forms of selenium are absorbed by different mechanisms, intestinal absorption of selenium does not seem to be limiting factor for the bioavailability of selenium, particularly for non ruminants. Furthermore, intestinal absorption of selenium is not involved in the homeostasis of this essential trace element.

Following absorption, both organic and inorganic sources of selenium are utilized for the synthesis of selenoproteins. Whereas selenium from selenate or selenite may be immediately available for these synthetic processes, a varying part of selenium provided as selenomethionine or selenocysteine will be unspecifically deposited in tissue proteins. **Shelton (1990)** on the other hand, stated that this "selenium store" may contribute to the synthesis of specific selenoproteins during insufficient dietary selenium supply. If selenium is supplemented to improve animal health and performance, organic and inorganic sources of selenium seems to be equally well suited. However, if selenium is supplied to livestock as a means to improve selenium nutrition in humans by selenium enriched animal products like meats, eggs or milk, organic selenium sources like selenomethionine or selenized yeast are advantageous **(Barnes *et al.*, 1990).**

1.9.1 Post-absorptive metabolism of selenium

In order to fulfill its metabolic role, selenium absorbed from the gastrointestinal contents must enter a metabolically active pool and be transformed into biologically active principles within the organism, aside from the effects of selenium incorporated into several selenoproteins with specific functions. It is important for an understanding of selenium metabolism to note that all of these seleno proteins contain selenium as one or more selenocystyl residues within the peptide chain. **Shelton (1990)** replacement of selenocysteine by cysteine in these proteins will result in a marked decrease or even complete loss of specific function

(Kaneko, 1980; Lasen, 1980; Shelton, 1990; Wilson, 1991; Burk and Hill, 1992; 1994; Keery and Amos, 1993; Aharoni *et al.*, 1995).

1.10. Selenium digestibility

In ruminants, selenium absorption is around 35% while in pigs, absorption values are 75-85% **(Luginbuhl, 1998). Anundi and Stahl (1984**) measured selenium digestibility in growing pigs weighing around 52 kg. They found that the apparent selenium digestibility of both selenite and organic selenium average about 75% when fed at supplemental levels of 0.3 ppm. Selenium retention was increased significantly when sel-plex was the added dietary selenium source. Most of the difference in selenium retention was the result of increased selenium absorption since there was no difference in average daily urinary selenium exertion between the two supplemental sources. **Mahan and Parrett (1996)** also foud increased selenium retention with sel-plex in growing pigs; but in contrast to the present study the difference was due increased urinary excretion of selenium in selenite-supplemental animals. While average daily urinary selenium excretion was not different between treatments, selenium excretion following exercise test in horses given inorganic selenium was higher than during day 1 or 2 of the collection.

1.11. Whole blood and plasma selenium

Before horse exercise, mean plasma values were slightly higher than reference value reported by **Sanz *et al.* (1995).** but similar blood values were typical for horses receiving selenium supplementation **(Siddon *et al.*, 1985 and Mosoni *et al.*, 1999).** Seymour and Polan (1986) reported increase in serum selenium following training jog in 45 standard bred horses. Red blood cell selenium was similar between treatment groups and there was a trend towards a decrease in

RBC selenium post-exercise. Plasma selenium remained elevated in both treatments post-exercise. The digestibility of selenium in horses appears to be intermediate between ruminants and non ruminants **(Sanz *et al.*, 1995)**. Selenium from yeast was more digestible than from sodium selenite leading to a greater positive selenium balance in those horses. **Sanz *et al.* (1995)** reported that the level of selenium supplementation was probably above requirement since every horse was in positive selenium balance and blood selenium values were on the high side of normal reference ranges established for selenium requirements. Increased urinary selenium excretion following exercise in the inorganic selenium group suggests that requirement for selenium by exercised horses may be dependent on the selenium source and the exercise frequency **(Henry and Ammerman, 1995).** The exercise intensity used in an increase in plasma selenium in both treatment groups. The source of this increased plasma selenium may have been the red blood cells **(Mahan and Parrett, 1996),** since there was a trend towards lower RBC selenium following exercise, plasma selenium in those fed the inorganic source returned to pre-exercise levels while plasma selenium in those given sel-plex remained elevated at 24 hrs post-exercise, perhaps part of the selenium that mobilized from the RBC in the selenite group during exercise was voided in the urine, leading to an increase in urinary selenium excretion during the subsequent 24 hr collection period. After absorption, **Henry and Ammerman (1995)** stated the red blood cells (RBC) take up inorganic selenium and return it to plasma in the reduced (i.e. hydrogen selenide) form, where it binds to plasma proteins and is transported to the liver to become part of the selenium pool for selenoprotein formation **(Combs and Combs, 1985).** Some Se travels to the kidney and is excreted via urine. Organic selenium (selenoamino acids) travels in the blood by amino acid transport mechanisms and is less likely to be lost through urinary excretion.

1.12. Dietary requirement of Selenim

Dietary requirements based solely on the Se content of the diet are difficult to do because many factors affect the bioavailability of Se. **Combs and Combs (1986)** stated that bioavailability varies with the form of the Se compound, feedstuffs vary with respect to bioavailability, and other dietary factors can either enhance or decrease availability of supplemental Se sources. **Henry and Ammerman (1995)** found that the relative bioavailabilities for cattle are sodium selenite 100; cobalt selenite 105; selenomethionine 245 and Se yeast 290 ppm. They also point out that currently only sodium selenite and sodium selenate can be legally used as supplements. Vitamin E is probably the dietary ingredient which has the most profound effect on dietary Se needs. The action of Se and vitamin E are synergistic and many studies have demonstrated a reduction or elimination of deficiency symptoms where either compound is used. **Weiss *et al.* (1997)** showed that dairy cows fed high levels of vitamin E (up to 4000 IU/day) were less likely to have clinical mastitis when fed only 0.1 mg/kg of Se than cows receiving 100 IU/day of supplemental vitamin. **Weiss *et al.* (1997)** stated that cows in Se deficient areas require between 5 and 1 mg/day of supplemental Se (approximately 0.3ppm for the average lactating cow or 0.6ppm for the average dry cow) to maintain blood and plasma concentrations in the optimal range. **Weiss *et al.* (1997)** stated that most of the use is of sodium selenite as the source of Se. Because of the possible effects of chronic Se toxicity, may diets are not supplemented to an optimal level. Acute selenosis is monitored in diarrhea, respiratory distress and neurologic impairment **(Blood *et al.*, 1983)** and chronic selenosis can result in illthrift and lameness, hence over supplementation must be avoided. **Dargatz and Ross (1996)** reported in cattle blood Se concentrations, on supplemental Se that it is not adequate for many herds. Based on his survey, the relatively low level of supplementation recommended (0.1 to 0.2 ppm) for beef cattle **(NRC, 1996)** may not be adequate, and this recommendation is then for total dietary Se and not supplemental Se.

1.13. Role of Selenium yeast

In dairy herds, where blood levels of Se do not reach optimal levels even when 0.3mg/kg of Se is supplied, either enhancing factors such as vitamin E or a more bioavailable source of Se such as Se yeast might be recommended. In many diary herds, this may be most critical during the dry period or the first few weeks of lactation when feed intake (dry matter intake as % of body weight) is lowest. **Mahan and Parrett (1996)** compared sodium selenite with Se-enriched yeast (sel-pelx 50, Alltech Inc) as dietary Se sources for grower and finisher swine. They demonstrated higher retention and lower excretion for the Se-enriched yeast than for sodium selenite. In a similar study with dairy cattle, **Fisher *et al*. (1995**) compared sodium selenite with Se yeast. Each source was fed at a rate of 2.2mg Se/head/day. It was some what similar to the studies with swine. The Se yeast gave higher serum Se concentrations and milk Se was also higher for cows. Se levels in natural feedstuffs are not adequate for optimum performance of dairy cattle. **Forbes (1995)**, found impaired immune response (susceptibility to mastitis) and reproductive problems are most frequently associated with Se deficiency, and other problems can also occur. The dry and early lactation periods are the most likely to be deficient when supplemented with dietary Se because of the relatively low intake of feed during these periods **(Mahan and Parrett, 1996).** Either the use of enhancing factors such as vitamin E or the use of more bioavailable source of Se, yeast may be needed when limits are set on the amount of Se which can be added to the diet **(Gall, 1996).**

1.14. Organic selenium and health:-

The amount of demonstrating the selenium is vital for animal performance and health is increasing but current investigation are also beginning to show it may be helpful is curbing one of the more dreaded human health problem is-cancer **(Clark *et al.*, 1997).** The incorporation of the element into the organic structure of

amino acids or selenoprotiens is now showing evidence that its role in several biological mechanisms may surpass that of the inorganic from of the element **(Grimaud, *et al.*, 1998).** Organic selenium may thus be one of the important nutritional keys to improved human and animal health of the 21st century because less research has been conducted with organic selenium than the inorganic form, many questions have yet to be asked and answered before we completely understand its role in animal and human nutrition **(Grimaud *et al*., 1999).** It is becoming clear from the existing research that the biological function of organic selenium is basically the same for all species and that form of selenium may be superior to the inorganic form of the element.

1.15. Organic selenium: nature's source of animal and humans

Selenium found in grains produced in nature is organic. Consequently its initial discovery as a dietary essential nutrient was established form it's use in the organic form. **Schwartz** and **Foltz (1957)** discovered by feeding brewer's yeast that a liver abnormality could be corrected. The brewer's yeast that was fed, later found to contain a vital component that initially established selenium as a nutritional essential. **Olson *et al.* (1970)** later demonstrated that selenomethionine was the principle form of selenium in wheat and thus selenomethionine is considered the major from of selenium in most non-accumulator plants. Because it was common practice prior to house animals outdoors, particularly reproducing animals, and to formulate diets using forage products (unknowingly with and indigenous source of organic selenium) the deficiency occurrence of this element was not recognized until livestock confinement began. Upon the discovery that liver and white muscle problems encountered with livestock where of selenium deficiencies, it was the inorganic form of the element, notably sodium selenite, that became the commonly used form of supplemental from yeast containing selenium in the protein component of the yeast cell and a similar selenoamino acid profile to grains **(Kelly**

and Power, 1995), an organic source of supplemental selenium is now an option. Although the supplementation of inorganic selenium to live stock feeds was initially of tremendous benefit to the livestock industry (**Hichs *et al.*1990)**, evidence is now emerging that the organic form of the element may have additional benefits that surpass that of organic form, the roles that organic selenium plays while performing many of the same functions as inorganic selenium. One of recognized biological needs for selenium in all animal species is for the production of glutathione peroxidsae (GSH-Px). This enzyme contains selenium and has active antioxidant role in the cytosol of the cell where it prevents the accumulation of superoxide molecules from forming toxic free radicals. The incorporation of selenium into muscle tissue is largely dependent upon the form of selenium provided and secondarily by the level of selenium in the diet. **Goetsch et *al.* (1998)** demonstrates that only a small increase in muscle selenium concentration occurred when inorganic selenium was fed, whereas **Sahlu *et al.* (2004)** proved that when the organic source was provided, there was a linear increase in the selenium content of muscle tissue. This could be important in improving the nutritional quality of pork as well as providing a product with an excellent source and quantity of selenium for humans. Inorganic and organic selenium have been found to differ with regard to placental transfer. An experiment evaluated the efficacy of both sources of the element at 0.1 or 0.3ppm Se **(Mahan and Kim, 1996).** A higher concentration of selenium was transferred when 0.3ppm Se level was provided from either selenium source, but the magnitude of increase was higher when the organic source was provided. However, at low dietary levels some of the organic selenium may be diverted to and incorporated initially into muscle tissue with less being available for incorporation into liver tissue. When the inorganic form of the element was provided there was smaller linear increase in milk selenium, whereas the magnitude of the increase when sel-pelx was provided was much greater. The combination of feeding the sows both organic and inorganic selenium sources resulted in a milk

selenium content that was intermediate to that of both groups fed the 0.3ppm Se. Interestingly, **Patteson *et al.* (1957)** stated that the value of the latter group was almost identical to the milk selenium content when sows had been fed 0.15 ppm in the organic form. This suggests that the organic form was readily transferred across the mammary tissue while the inorganic form was not. There is little benefit to increase the dietary inorganic selenium level above 3ppm **(Edens, 1996).**

1.15.1. Ruminants

Castro *et al.* (1997) cited inorganic selenium to have a reduced bioavailability in the ruminant because of the anaerobic conditions in the rumen. Although some of the oxidized form of selenium (sodium selenite) is reduced in the rumen to the selenide form which is not absorbed through the rumen or the intestinal tract, some of the consumed inorganic selenium is used by rumen microbes for their metabolism. Further, **Edens (1996)** suggests microbial protein thus formed with selenium can pass into the small intestine and serve as a source of dietary selenium for the ruminant, but overall the bioavailability of inorganic selenium for ruminants is poor. In contrast, organic selenium is in the form of selenoamino acids and generally by- passes the rumen. The selenium –enriched yeast protein is hydrolyzed in the rumen and small intestine to the respective amino acid **(Carnier *et al.,* 1984)**.These selenoamino acids are absorbed with selenium being retained and effectively utilized. Subsequent research has demonstrated that blood GSH-Px activity in ruminants is lower when the inorganic form of the element is fed to dairy cows **(Pehrson *et al.,* 1989).** Other research has demonstrated that milk selenium can be increased 4-5 fold by feeding organic selenium; it would appear that however much conducted with supplement selenium as sodium selenite, may need to be replenished using an organic selenium source **(Clark *et al.,* 1997).**

1.16. Effect of organic Selenium on blood

Low Se in soils and forage combined with difficulty in supplementing growing heifer approaches calving. Selenium fertilization is effective during the grazing season; however change in availability occur due to factors such as time post application and the change from pasture to hay and silage as feed sources **(Gulteridge** and **Shelton, 1994).** An additional disadvantage of this method is the toxicity of the material and danger of over-application. The purpose of this field investigation was to compare blood Se levels in animals on Se-fertilized pasture with those receiving either injected Se or Se in the form of selenium yeast. Animals of 6-12 months of age were fed Se yeast in concentrate mix. **Goetsch, *et al.*, (1998)** these animals consumed 3 kg of concentrate per day. Sel-plex 50 was added to this; a premix containing 4 gms of sodium selenite per ton of feed was added. The heifers run on kukuyu pastures during summer and receive hay and millet silage in winter. Lactating cows fed concentrate prior to calving; they graze pasture during summer and receive silage and hay on pastures during winter.

Varly (1967) reported the feeding of selenium yeast has the effect of rapidly raising blood Se levels in the initial stages of lactation which in turn will benefit in rebreeding if cows are fed a dry cow ration through the dry period as well. **Toussaint (1984)** added an advantage of sel-plex50, that it can be withdrawn and included according to blood levels analyzed. It should be noted that the trail was not conducted on the same animals. The reason being that it would take 3years to reach a conclusion; however, these fields illustrate the success of this approach **(Van Eenaeme *et al.*, 1998).**

1.17. Selenium supplementation for lactating dairy cows

Some soils are poor in minerals essential for growth of plants and animals. **Gulteridge and Shelton (1994)** reported difficient trace element Se in dairy cows

because one third of grazing pastures land is low in Se, a deficiency that can impair immune function and cause reduced reproductive performance. Beyond simply meeting cow dietary requirements, trace element management can also benefit consumers of milk and dairy products. Supplement technologies that substantially increase Se concentration in whole milk and casein will provide Se in a natural biochemical form that is readily absorbable by infants and children, the populations most at risk of Se insufficiency **(Gulteridge** and **Shelton, 1994).**

1.18. Effect of organic selenium on production and reproduction

Selenium for optimal production and reproductive efficiency has created numerous dilemmas for nutritionists and regulatory officials. The mineral is typically absent, or at lower than adequate levels in available feedstuffs. This is due to crops being produced in areas characterized by selenium deficient soils- a widespread problem throughout the world. This creates the necessity to provide selenium via the ration and creates a second area of concern for nutritionists **(Devendra** and **Burns, 1983).** The regulatory have placed a maximum allowable level of selenium inclusion in the total diet of 0.3ppm. This level has been determined due to toxic properties of selenium in the environment and the desire to keep selenium intake well below toxic levels. In certain cases the 0.3ppm level is not sufficient to adequately meet the nutritional needs of the animal due to severe selenium deficiencies in available feed ingredients **(Casey, 1992).**

1.18.1 Effect on lactating dairy cows

Selenium has been recognized as essential trace element in lives-enzyme glutathione peroxidase make selenium, along with vitamin E, important in reproduction and immunity along with a wide range of metabolic and digestive roles **(Casey, 1992).** Selenium deficiency in the dairy cow is often seen as retained placenta and reproductive failures of serious kinds. Calves born to deficient dams

are weak at birth or even stillborn. Often Se deficiency has an effect on milk production, but this effect may be blamed on other nutritional or environmental problems **(Casey, 1992).**

Flavophospholipol (flavomycin) is a feed additive to be used exclusively as growth stimulant, and for the improvement of the feed conversion rate in farm animals. Se is also involved in the immune function of animals. **Larson (1988)** observed a trend for increase 1g concentration in Se supplemented ewes and lambs. Calves farling to absorb enough l g have higher risk of morbidity **(Me Guire *et al.*, 1976).** In addition. **Larson (1988)** reported significant increased titers to telanus toxioid in Se supplemented lambs. **Toukourou** and **Peters (1999)** reported a decreased lymphocyte response in lambs deficient in vitamin E and selenium. At lambing rectal temperature and body weight of each new born lamb were recorded and colostrums sample was collected from the ewe on twin lambwas removed before suckling the ewe for eventual tissue collection **(Larson, 1988).** The lamb was fed pooled bovine colostrums (25 ml colcstrum/kg of bodyweight) initially and again 6 hrs late. At 12 hrs blood sample were taken and lambs were euthanized with sodium pento barbilol. Liver, kidney, heart, gastrointestinal tract and brown adipose tissues form the perirenal and pericardial regions were removed, weighed and frozen. Intake of Se supplementation and was similar between the two supplement group. **Toukourou** and **Peters (1999)** feed intake and body weights were similar among treatment groups. Overall there was a linear decrease in concentrations of Se in serum for ewes with length of gestation. **Van Eenaeme *et al.* (1998)** reported that while Se from organic sources increased concentrations of Se in whole blood of sheep, chemical form of selenium did not significantly affects plasma. Se accordingly, the concentration of selenium in whole blood were increased ($\leq$ 0.0001) by Se supplements indicated greater uptake by the red blood cell of Se from sel-plex than selenite. **Seymour** and **Polan (1986)** providing further evidence that organic forms of Se are more readily transported across the placenta.

Concentrations of Se in the liver of lambs were also significantly increase by Se supplementation in addition ewes given the sel-plex had lambs with significantly higher GSpx activity than the lambs of ewes given selenium.

Thus maternal Se supplementation improved some of the measures predictive of susceptibility of new born ruminants of disease **(Larson, 1988).** Se in sel-plex was transferred more readily to ovine fetus and colostrums than selenium selenite thus; supplements containing sel-plex may be particularly beneficial when total intake of Se are limited- the finding that low but not deficient intake of Se by pregnant cows and ewes effects levels has large significance for health and survival of newborn passive immunity **(Varly, 1967)** and heat production from brown adipose are required for newborn survival and events may be influence by maternal Se intake. Se in forage and seed grains is normally present as organic Se in the form of selenocysteine, selenocysteine and selenomethionine. Inorganic selenium Se sources such as sodium-selenite are less available to ruminants due to reduction of selenium in selenite to an unavailable from by the rumen bacteria **(Seymour** and **Polan, 1986).**

Addition of l mg Se from sel-plex Se resulted in average increase in GSH-px activity of 2.28 times after five month demonstrate effectiveness of the organically-bound Se in sel-plex in improving herd Se status **(Combs** and **Combs, 1986).**

Selenium has long been recognized as an essential trace element in livestock nutrition its function as a part of the chief intracellular antioxidant enzyme glutathione peroxidase make selenium along with vitamin E important in reproductive and immunity along with a wide range of metabolic and digestive roles **(Seymour** and **Polan, 1986).**

1.19. The vitamins

Vitamins are organic substances required by animals in very small amounts for regulating various body processes toward normal health, growth, production and reproduction. But this definition ignores the importance part that these chemical substances play in plants and their importance generally in the metabolism of all living organisms. The term vitamin was used by funk in 1912 for an amine, the active factor from rice polishing which are necessary for existence of life (vital+amine). Later on the terminal "e" was omitted, leaving vitamin. **Combs** and **Combs (1986)** the existence of vitamin like factors was first recognized in the orient when prisoners fed on unpolished rice seemed to be suffering with beriberi disease than were non-prisoners consuming polished rice. Soon thereafter, workers in the United States recognized the presence in the butter fat of milk of a factor which prevented night blindness in calves. However, **(Seymour and Polan, 1986)** the factors seemed to be different from that in rice polishing since the milk fat soluble rather than water soluble and also did not contain nitrogen.

Vitamin E also involved in the synthesis of ascorbic acid uniquinine (co-enzyme) and the metabolism of nucleic acid. Vitamin E deficiency cause embryonic degeneration of female hen, turkey, cow and ewe. **Casey (1992)** it leads to sterility in male guinea pig, cod and cock. Vitamin E is equally effective in preventing nutritional encephalomalacia, while selenium is not important. In other two diseases affecting chicks, both vitamin E and selenium appear to be involved. It is widely distributed in foods **(Goetsch *et al.*, 1998)**, green fodder is good source of α-tocopherol young grass being a better source than mature herbage. Animal products are relatively poor source of the vitamin. Vitamin E values of food are often stated in term of international units. One i.u. of vitamin E being defined as the specific activity of synthetic α-tocopherd acetate.

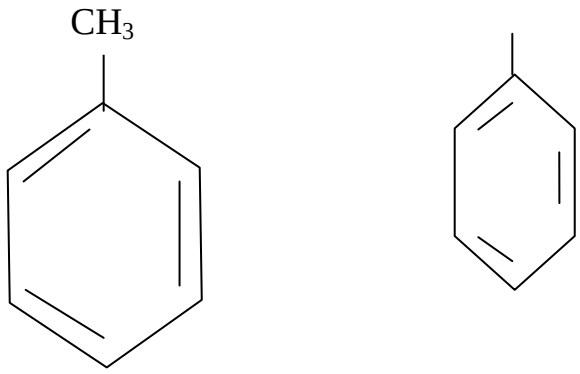


Figure 1. Vitamin E

Chemically divided into two groups:

i. Saturated (0,13y and 8 tocopherols)
ii. Unsaturated (8, 13, y and tocopherpls).

1.19.1. Vitamin E and Selenium:-

New studies have been referred to use one of the metabolic signals to stimulate gonadotrophic hormonal release **(Hall *et al.*, 1992)** specific nutrients that are involved in reproduction **(Martin** and **Walked–brown, 1995)**, growth and development **(Lindsay *et al.*, 1993 and Foster, 1994)** may be used for the same goal.

Preston (2000) reports have suggested that vitamin E and selenium (Se) are one of the important nutrients that can affect many biological processes in the body such as immunity **(Hemken *et al.*, 1998).** Metabolism **(Awadeh *et al.*, 1998)** and reproduction **(Jarry, 1996)** in addition vit. E and Se improved spermatogenesis and semen quality **(Marin-Guzman 1990, Brzezinsks- Slebodzinsha *et al.*, 1995 and Marin-Guzman *et al.*, 1996).**

1.19.2. Vitamin E requirement

The vitamin E requirement may therefore be defined as the amount required preventing peroxidation in the particular subcellular membrane which is most susceptible to peroxidation. Moreover, Se is a component of selenoproteins and is involved in immune and neuropsychological function in the nutrition of animals

(Meschy, 2000). Most nutritionists assume that reproductive performance will not be limited when animals are fed diets that meet the NRC levels. However, little is known about the effects of vitamin E supplementation on specific reproductive events in sheep. Because fertilization in sheep is an all-or-none phenomenon (i.e., either all ovulated eggs are fertilized or none are fertilized), the three major variables that contribute to litter size are ovulation rates, embryonic survival and fetal survival **(Koyuncu *et al.*, 2006).**

Vitamin E and Selenium are essential nutrients that complementary biological functions as antioxidants to minimize cellular damage caused by endogenous peroxides **(Kolb *et al.*, 1997).** Selenium (Se) has a biological function related to vitamin E in that Se is an essential component of glutathione peroxidase, an enzyme involved in detoxifation of hydrogen peroxide and lipid hydroperoxides. The vitamin E requirement may therefore be defined as the amount required preventing peroxidation in the particular sub cellular membrane which is most susceptible to perxidation **(Koyuncu** and **yerlikaya 2007).** Vitamin E prevents oxidative damage to sensitive membrane lipids by suppressing hydroperoxide formation **(Chow, 2001)** and protects cellular membranes thus maintaining membrane integrity and reducing oxidative stress **(Hogan, 1993).**

Vitamin E displays a wide variety of deficiency signs, more than any other vitamin. Deficiency signs differ among species and even within species. **Blaxter (1962)** reported that muscle degeneration and muscular dystrophy appear to be the one vitamin E deficiency syndrome common to all species. White muscle disease (WMD), also known as nutritional muscular dystrophy or nutritional myodegeneration (NMD), is the major clinical manifestation of a vitamin E or selenium deficiency in newborn calves, lambs and kids.

Likewise, **Walsh *et al.* (1993a)** reported that non-ruminating calves fed diets deficient in either vitamin E or both vitamin E and selenium had increased lipid peroxidation products in muscle tissue. Feeding rumen-protected linseed oil to

vitamin E-selenium deficient calves further increased the level of lipid peroxidation in muscle.

1.19.3. Vitamin E deficiency in sheep and goats

In lambs, white muscle disease (WMD, also known as stiff-lamb disease) takes a course similar to that observed in calves. Motor disturbances such as unsteady gait, stiffness of the muscles of the hindquarters, neck and forelimbs, arched back, muscle tremors and perspiration are encountered in the acute form. On necropsy, white striations in cardiac muscle and bilateral lesions in skeletal muscles characterize the disease. A gradual but progressive swelling of the muscles, particularly in the lumbar region and rear legs, gives the erroneous impression of muscular development. In similarity to the peracute deficiency encountered in calves (changes occur primarily in the myocardium), chronic cardiac muscle degeneration also occurs in the lamb. Affected lambs appear normal at birth, but quickly lose weight after the third week of life. They also show aversion to social stress and may stand apart from the flock. Cardiac arrhythmia and increased heart rate can result even after slight exercise. In the advanced stage, animals consume little if any feed and rapid wasting occurs. Symptoms can be reversed by prompt administration of vitamin E and selenium.

For dystrophic lambs, an oral therapeutic dose of 500 IU dl-alpha-tocopheryl acetate followed by 100 IU on alternate days until recovery is successful **(Rumsey, 1975).** Vitamin E-selenium responsive conditions are not restricted to young animals and are manifested as lack of thrift, occurring in lambs at pasture **(Underwood, 1981).** Marginal vitamin E deficiency of yearling sheep can progress to WMD. In sheep of 9 to 12 months of age, the disease is frequently observed following driving of the flock with the rapid onset of listlessness, muscle stiffness, inability to stand, prostration and, in severe acute cases, death within 24 hours **(Andrews *et al.*,1968). Hartley** and **Grant (1961)** reported the incidence of WMD

in barren ewes was reduced from over 30% to 5% with selenium administration. Farms in New Zealand have had lamb losses as high as 40% to 50%. In these regions, the syndrome may respond to vitamin E, selenium or both. **Maas *et al.*, (1984)** described nutritional myodegeneration in lambs and yearling ewes with normal selenium status but deficiency of vitamin E.

Deficiency of vitamin E and (or) selenium in the goat, as in other ruminants, results mainly in WMD. Goat kids are born with little or no reserves of the fat-soluble vitamins A, D, and E. Sudden death of young kids fewer than two weeks of age may reveal postmortem evidence of muscle disease and degeneration in the heart muscle or the diaphragm. In older kids and mature animals, deficiency can occur after sudden exertion and stress. Affected animals exhibit bilateral stiffness, usually in the hind legs. In high-producing dairy goats, deficiency manifests itself in poor involution of the uterus, accompanied by retained placenta and metritis following kidding **(Guss, 1977).** Goat kids four to five weeks of age that were diagnosed with nutritional muscular dystrophy (myodegeneration, NMD) had lower concentrations of both vitamin E and selenium in the liver, skeletal muscle and myocardium **(Rammell *et al.*, 1989).** Vitamin E concentrations in the liver, skeletal muscle and myocardium in NMD cases averaged 40%, 43% and 30% respectively of those in healthy goat kids, clearly indicating vitamin E depletion

Selenium is an essential trace mineral present in the soil is absorbed by growing plants that are eaten by foraging/browsing goats. Proper selenium levels are necessary for goats to reproduce, lactate, give birth, urinate, and have properly functioning muscles. Selenium working with vitamin E helps develop and protect healthy brain cells, assists in thyroid function, helps the immune system function properly, and prevents cell wall damage. Symptoms of selenium deficiency are similar to those of Vitamin E deficiency. White Muscle Disease, also known as Nutritional Muscular Dystrophy, is a condition in which kids are too weak to stand or suckle at birth, they consistently cough, milk sometimes runs out of their nose

after nursing, and they develop pneumonia because of muscle weakness in their lungs. In adults, abortions, stillbirths, retained placenta, or inability to conceive can be indicative of selenium deficiency.

Pen-fed goats can be more susceptible to selenium and vitamin E deficiency since they don't have access to forage plants containing them. High levels of sulphur in feed prevent selenium absorption. Proper levels of calcium in feed can help in selenium and vitamin E uptake. Selenium is routinely added to processed grain by feed mills, but the amount permitted by US law may be insufficient for some areas.

Oral supplementation of vitamin E should be given in conjunction with Mineral Max injections. Mineral Max/Multi Min is another product that is not labeled for goats and must be used under a qualified vet's prescription and supervision.

CHAPTER TWO

MATERIALS AND METHODS

In this study, two experiments were conducted to evaluate the effect of selenium on the reproductive performance of Nubian goats, alone or synergized with ά-tocopherols (Vitamin E).

2.1 Effect of selenium on the reproductive performance of Nubian goats

2.1.1 Experimental animals and housing

Eleven mature Nubian goats, of weight range 20-27 kg, were selected from a goat flock affiliated to the Faculty of Agricultural Science, Sudan University of Science and Technology, Shambat. They were housed in three separate pens holding 5, 3 and 3 heads each. They were pregnancy diagnosed by abdominal palpation to be non-pregnant.

Goats were ear tagged and prepared by washing with acaricide Hexachlorocyclohexane (Gamatox, Cooper, UK) for the control of external parasites and Avermectin (Ivomec, Coopers, UK) for the control of internal parasites. Three mature Nubian bucks of which one was castrated were added to the flock each in a pen.

2.1.2 Feeding program

The experimental animals were kept under indoor forageing system where Abu 70 forage (*Sorghum bicolor*, **Table 1**) was cut and supplied to all experimental groups daily at the rate of 1.5kg per head supplied twice daily at 10:00 morning and 5:00 evening.

A concentrate (**Table 2**) was offered also on daily basis at the rate of 1 kg per group, supplied once at noon (12:00) time. Water was amply provided during day time.

Table 1. Percent analyzed chemical composition (dry-matter basis) of *Sorghum bicolor* forage fed to experimental goats for 6 months.

Components	%
Dry matter	28.00
Crude protein	01.92
Ether extract	00.84
Crude fiber	10.81
Nitrogen-free extract	12.04
Ash	02.39

Table 2.Percent composition (as-fed basis) and calculated chemical composition (dry-matter basis) of the concentrate fed to experimental goats for 6 months.

Ingredients	%
Dura	38
Cottonseed cake	20
Molasses	16
Groundnut hulls	24
Limestone	01
NaCl	01
Total	**100**

Components	%
Dry matter	93.13
Crude protein	7.88
Ether extract	1.85
Curde fibre	28.75
Nitrogen-free extract	46.97
Ash	7.68
Energy (Mcal ME/kg)	1.40

2.1.3 Experimental design and dosages

The goats were divided to three groups according to similar body weight basis. Each of groups A, B and C constituted 5, 3 and 3 heads respectively. Group A was designated as control goats (having the castrated buck). Selenium premix (Alltech, Kentucky, USA ; **Appendix 1**) at dose rates 0 (devoid), 5(low) and 10 (high) mg/kg was fed **(Appendix 2**) twice weekly, mixed in concentrate by dilution, to groups A, B and C respectively.

2.1.4 Data collected

2.1.4.1 Health

Goats were observed throughout the experimental period for health remarks during gestation and delivery.

2.1.4.2 Blood

Heparinized blood samples were withdrawn from the jugular vein every three weeks at about 9.00 a.m., and were analyzed on the same day for RBCs count, Hb concentration, PCV, WBCs count and their cell differential lymphocytes.

2.1.4.3 Serum

Sera were prepared and analyzed for metabolic indicators glucose, total protein, albumen, globulin and cholesterol. Electrolytes Ca, P and Se were determined. Serum enzymes ALT and ALP activities were also measured.

2.1.4.4 Gestation and deliveries

Gestation start was assessed by palpation. On delivery, neonate sex and birth weight (kg) were recorded and then body weight four weeks later.

2.2 Effect of selenium synergized with vitamin E on the reproductive performance of Nubian goats

2.2.1 Experimental animals, accomodation and feeding

Eleven mature Nubian goats, weighing 20-27 kg, were selected from a goat flock of the Faculty of Agricultural Science, Sudan University of Science and Technology, Shambat. They were housed, pregnancy diagnosed, prepared, fed and watered as was done in experiment one. Three mature Nubian bucks of which one was castrated were added to the flock each in a pen.

2.2.2 Experimental design and dosages

The experimental goats were divided to three groups on similar body weight basis. Groups A, B and C constituted 5, 3 and 3 heads respectively with group A designated as control goats (having the castrated buck). Selenium Premix (Alltech, Kentucky, USA ; **Appendix 1**) and vitamin E (ά-tocopherols acetate, Avico, Jordan) at 50% concentration (500 I.U./g) in powdered form were fed added to the

concentrate by dilution, at dose rates 0+0 (devoid), 5+250 (low) and 10+500 (high) mg/kg + I.U./g **(Appendix 2**) twice weeky to groups A, B and C respectively.

2.2.3 Data collected

Health observation throughout the experimental period; blood withdrawal and analyzed parameters; sera preparation and analyzed indicators; assessment of gestation beside neonate weighings, all were monitored as outlined in experiment one.

2.3 Methods

2.3.1 Hormonal protocols

One hormonal protocol was chosen to assess its effectiveness in synchronizing estrus in two groups of experimental animals. In the third group a castrated buck was introduced and remained with the females for consecutive 15 days to synchronize estrus (buck effect). The synchronization was run in two phases as follows:

- Group B goats (3 heads) were injected with a dose of prostaglandin PGF2a (Cloprostenol Estrumate, Schering Plough Anumal Health, Germany) on day 1 and another dose 11 days later i.e. on day 12 (each dose contained 7.5 mg PGF2a per ml). Signs of heat were observed and recorded (teaser buck was used to detect heat).
- In group C goats (3 heads), the castrated buck was introduced and kept continuously with the goats for 15 days. Signs of estrus, behavioral signs, estrus duration were recorded for each high and low level group in Se and Se / Vit. E.

2.3.2. Haematological methods

All of the eleven goats were subjected to drain of blood samples every three weeksat about 9.00 am from the jugular vein, using a 10 ml plastic disposable syringe. Ten ml of blood were obtained from each animal into clean dry heparinized vacotainers for haematological parameters which were evaluated on the same day of collection (**Schalm, 1965**). During the collection of samples the animals were kept quiet, as the excitation may lead to significant change in the composition of the blood (**Kelly, 1984**). The area from which the blood was drawn was cleaned with alcohol.

2.3.2.1 Red blood cell count (RBC)

Red blood cells were counted with an improved Neubauer haemocytometer (Hawksly and Sons Ltd. England). Formal citrate (3 gm sodium citrate, 1 ml conc. formaldehyde, 100 ml distilled water) was used as a diluent. The red blood cells diluting pipette was filled according to the procedure described by **Schalm (1965)**. All erythrocytes in the 4 corners of the chamber and the center squares were enumerated; the total was multiplied by 10^4 and expressed in millions per cubic mm of blood.

2.3.2.2 White blood cell count (WBC)

White blood cells were counted with an improved Neubauer haemoycytometer (Hawksley and Sons Ltd. , England). Turk's solution (1 ml glacial acetic acid, tinged with gentian violet, 100 ml distilled water) was employed as a diluent. The white cell diluting pipette was filled according to the procedure described by **Schalm (1965)**. Enumeration was of the white cells was achieved by counting all leucocytes in the 4 leucocytes counting squares, the total was multiplied by 50 and expressed in thousands per cubic mm of blood.

2.3.2.3. Differential leukocyte count

The percentage of neutrophils and lymphoctes were determined microscopically from a count of 100 leukocytes in thin, Giemsa stained blood smear (**Kelly, 1984**). Giemsa stain was prepared by transferring 3.8g of Giemsa powder to a dry bottle, 250 ml of methanol were added to the stain and mixed well, and then 250 ml of glycerol were added and mixed well. For use, the prepared Giemsa stain was diluted 1:10 with distilled water.

The blood smear was prepared freshly. The blood was spread using the spreader slide, then the smear was dried at room temperature and fixed by placing in absolute methyl alcohol for 5 minute. The smear was covered by the stain for 15 minutes, and then the smear was rinsed well in distilled water and allowed to dry. The blood film was examined using the oil immersion lens (X 100).

2.3.2.4. Packed cell volume (PCV)

Fresh blood samples were centrifuged in a microhaematocrit centrifuge (Hawksley and Sons Ltd., England) for 5 minutes and PCV percentage was read off on the microhaematocrit reader provided with the centrifuge.

2.3.2.5. Haemoglobin concentration (Hb)

Haemoglobin concentration was determined by the cyanmethaemocytometer technique using a haemoglobinometer. The method is based on the conversion of haemoglobin by means of Drabkin's solution (0.2 gm potassium cyanide, 0.2 gm potassium ferricyanide and 1 gm sodium bicarbonate per liter distilled water) to cyanmethaemoglobin. The concentration was measured in gm/100 ml of blood.

2.3.2.6. Red cell indices

2.3.2.6.1. Mean corpuscular volume (MCV)

MCV was calculated from RBC number and PCV values as follows:

$$MCV\ (m^3) = \frac{PCV \times 10}{RBC \text{ in million/mm}^3}$$

2.3.2.6.2 Mean corpuscular haemoglobin (MCH)

MCH was calculated from RBC count and Hb values as follows:

$$MCH = \frac{Hb\ (g/dl)}{RBC \text{ in million/mm}^3}$$

2.3.2.6.3 Mean corpuscular haemoglobin concentration (MCHC)

MCHC was calculated from PCV and Hb values as follows:

$$MCHC\ \% = \frac{Hb\ (g/dl) \times 100}{PCV\ \%}$$

2.3.3 Chemical methods

2.3.3.1 Serum profiles

Non-heparinized blood samples were withdrawn from the jugular vein fortnightly at 7.00 a.m. into clean dry vials, allowed to clot overnight and serum was separated by centrifugation at 3000 r.p.m for 10 minutes and then stored at –20°C until analyzed for concentrations of metabolites glucose, total protein, albumin, globulin and cholesterol; and ALT and ALP enzyme activities; and minerals P and Ca using Spectrophotometer, Merck Mega, Version 0.6, 1995 (E. Merck, Darmstadt, Germany). Serum Se was analysed using Atomic Absorption Spectrometer.

2.3.3.1.1. Serum enzyme activities

2.3.3.1.1.1. Alanine amino transferase, ALT (Glutamic pyruvic transaminase, L-aspartate,2-oxoglutamate ,GPT)

It is an enzymatic method, which measures glutamic pyruvic transaminase in sera by monitoring the concentration of pyruvate hydrazone formed with 2-4dinitrophenyl hydrazine.

Test principal:

Oxoglutarate + L-alanine $\xleftrightarrow{GPT}$ L-glutarate + pyruvate

The absorbance of samples were read against the reagent blank after 5 mintues at wavelength of 630 nm UV/VIS Spectrophotometer. GPT was measured in U/L.

2.3.3.1.1.2Alkaline phosphatase (orthophosphoric mono-ester phosphohydrolase, E.C.3.1.3.1., ALP)

The serum ALP activity was measured photometrically using commercial kits (Randox Laboratories Ltd, U.K.).

Test principal

ALP catalyses the hydrolysis of *p*-nitrophenyl phosphate liberating *p*-nitrophenol and inorganic phosphate. The rate of *p*-nitrophenol formation is proportional to the ALP activity present in the serum:

p-nitrophenyl phosphate + H_2O $\xleftrightarrow{ALP}$ *p*-nitropheyl + inorganic phosphate.

The substrate (Nitro-4-phenylphosphate) was added to a buffered amino-2methyl-2propanol-1 and the serum was mixed with the buffered substrate. The mean absorbence change per minute (A_{405} nm/minute) was determined for calculation of enzyme activity as follows:

$$I.U. = A_{405}/min.x\ 2575$$

2.3.3.1.2 Serum metabolites

2.3.3.1.2.1 Blood sugar level

Serum glucose values were determined using enzymatic kits provided by Plasmatic Laboratory Product, Ltd.

Test principle

Glucose level was measured according to the method described by **Giterson, *et al*., (1971).**

$$\text{Glucose} + O_2 + H_2O \longrightarrow \text{Gluconate} + H_2O$$
$$2H_2O_2 + \text{phenol} + \text{4-amino-antipyrine} \longrightarrow \text{red chinonimin} + H_2O$$

Protocol

Dilute enzyme reagent (GOD + POD + 4-amino-antipyrine) was mixed with buffer (phosphate buffer, *p*H 7.5 + phenol). None-haemolytic serum was mixed with the reagent solution and the mixture was incubated at 37ºC for 15 min. The absorbance was read at wavelength 500 nm against the reagent blank and calculated as follows:

$$U/L = \Delta A \text{ sample} / \Delta A \text{ standard} \times \text{Standard concentration (100 ml/dl)}$$

(Where A= absorbance)

2.3.3.1.2.2 Total protein

Total serum protein was measured by a colorimetric method using a commercial kit (Randox Laboratories Ltd., U.K.)

Test principle

Colorimetric determination of total protein in serum is based on the biuret reaction. The serum protein reacts with copper sulphate in the presence of sodium hydroxide. The Rochelle salt (K-Na-tartarate) contained in the biuret reagent is utilized to keep the formed cupric hydroxide in solution which gives the blue colour. The intensity of the colour produced is proportional to the amount of protein in the sample. The absorbencies of the sample (A-sample) and of the standard (A-standard) were read against the reagent blank in the spectrophotometer at a

wavelength of 545 nm. The total serum protein concentration (C) was calculated as follows:

$$C\ (mg/dl) = A\frac{\text{sample X concentration of the standard}}{\text{A Standard}}$$

2.3.3.1.2.3 Albumin

Serum albumin was estimated quantitatively by the bromocresol green indicator method (**Doumas, *et al*., 1971**).

Test principle

Measurement of serum albumin is based on its quantitative binding to the indicator 5,5-dicromo-o-cresolsulphonaphthaline (bromocresol green, BCG).

Protocol

None-haemolysed serum was mixed with a buffered BCG reagent and incubated at 20-25ºC for 5 minutes. The absorbance of the sample and that of the standard were measured against the reagent blank at a wavelength of 630 nm and albumin concentration was calculated as follows:

$$g/L = \Delta A \text{ sample} / \Delta A \text{ standard} \times \text{standard concentration (6 g\%)}$$

(Where A= absorbance)

2.3.3.1.2.4 Cholesterol

Total serum cholesterol concentration was measured by an enzymatic colorimetric method using a kit (Randox Laboratories Ltd., U.K.).

Test principle:

Free cholesterol and cholesterol released from ester after enzymatic hydrolysis are oxidized enzymatically. The indicator quinoneimine is formed from hydrogen peroxide and 4-amino anti-pyrine in the presence of phenol and peroxidase:

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol Esterase}} \text{Cholesterol} + \text{fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol Oxidase}} \text{Cholestenone} + H_2O_2$$

$$2H_2O_2 + \text{4amino antipyrine} + \text{phenol} \xrightarrow{\text{Peroxidase}} \text{quinoneimine} + H_2O$$

The intensity of the coloured quinoneinine formed is proportional to the amount of cholesterol present in the sample. The absorbance of the sample (A-sample) and of the standard (A-standard) was read against a blank in the spectrophotometer at a wavelength of 500 nm and cholesterol concentration C was estimated as follows:

$$C\ (mg/dl) = \frac{\text{A sample X concentration of standard}}{\text{A standard}}$$

2.3.3.1.3 Determination of serum electrolytes

2.3.3.1.3.1 Calcium

Serum calcium concentration was determined by a colorimetric method using a commercial kit (Randox Laboratories Ltd., U.K.).

Test principle:

Calcium ions form a violet complex with chromogen (-O-cretholphthalein complex one-8- hydroxyquinoline hydrochloric acid) in an alkaline medium (2-amino-2-methyl-propan-1-01).

Serum was mixed with a buffered reagent and the absorbance of the sample (A-sample) and of the standard (A-standard) were measured against a reagent blank at a wave length of 578 nm and calcium concentration (C) was calculated as follows:

$$C\ (mg/dl) = \frac{\text{A sample X concentration of the standard}}{\text{A standard}}$$

2.3.3.1.3.2 Phosphorus

Determined by a colorimetric method using a commercial kit (Randox Laboratories Ltd, U.K.).

Test principle:

In nitric acid phosphate forms a coloured complex with molybdate and a reductant.

Serum was added to trichloroacetic acid for deproteinization, then the supernatant was added to the reductant and molybdate and the absorbance of the sample was read against a blank at a wavelength of 405 nm. Phosphorus concentration (C) was calculated as follows:

$$C\ (mg/dl) = 42.2 \times A\ sample.$$

2.3.3.1.3.3 Selenium

Selenium was detected in the sera of experimental animals electrochemically by atominc absorption according to **Price (1979)**.

Sample preparation

Sample was first diluted (0.5 sample with 4.5 distil. water) and then digested in 7ml of nitric/perchloric acid mixture (2:5) before temperature is raised to 210^{o}C and fumes of the perchlorate evolve. Two ml of HCl were added before transferring to the hydride generater using Na bromohydride (5g in 100ml of 0.1% mass/vol. caustic soda solution).

Detection

Sample concentrations (ppb) were determined by atomic absorption spectrometer (wavelength 196 μm) using standard (sample salt) and special lamp at 23mA.

2.3.4. Proximate analyses of experimental diets

Samples of basal experimental diets (forage or concentrate) were proximately analyzed, on dry-matter basis, for moisture, ash, crude protein, crude fiber, ether extract (fat) and nitrogen–free extract by the procedure described by the Association of Official Agricultural Chemists (**AOAC, 1980**). Results were expressed as percentage composition.

2.3.5. Statistical analyses

Mean test values in blood and serum, neonate weights and milk yield were compared to the control using the unpaired students't-test (**Snedecor and Chochran, 1967**).

CHAPTER THREE

RESULTS

3.1 Effect of selenium on the reproductive performance of Nubian goats

3.1.1 Health observations

All experimental goats showed the basic health signs althrough the experimental period. No ill signs were observed. Voluntary feed intake was at normal rates. Daily behavioural patterns were also normal. All deliveries were normal and no distokias experienced.

3.1.2 Blood parameters

Table 3 shows average hematological values of experimental goats fed selenium at low and high levels during the gestation period. All hematological values were not significant (P>0.05) compared to the control. Generally for all hematological parameters observed, values of LS were higher than those of HS, but always higher (P> 0.05) than the control except for lymphocytes of HS (4.90±2.99).

Table 3. Average (mean ± s.d.) hematological values of experimental adult goats fed selenium at low and high levels during the gestation period.

Groups	Parameters							
	RBC $\times 10^6$ mm	**WBC** $\times 10^3$ mm	**Lympho-cytes**	**Lympho-cytes %**	**MCV** %	**MCH** %	**MCHC** %	**Hb** g/dl
A-Control **(0 mg/kg)**	**2.06 ±0.72**	**13.22 ±4.93**	**5.58 ±4.22**	**40.01 ±27.10**	**98.75 ±19.99**	**36.60 ±7.42**	**36.77 ±9.14**	**7.50 ±1.98**
B - LS **(5 mg/kg)**	**2.39 ±0.39**	**14.48 ±3.45**	**7.42 ±3.59**	**51.73 ±19.76**	**102.36 ±12.81**	**36.73 ±2.53**	**36.38 ±5.24**	**8.80 ±1.22**
C- HS **(10 mg/kg)**	**2.25 ±0.43**	**13.42 ±3.52**	**4.90 ±2.99**	**39.03 ±23.87**	**102.50 ±10.09**	**34.91 ±5.07**	**34.75 ±7.33**	**7.65 ±0.86**

HS: High selenium.
LS: Low selenium.
All test groups means were not significant (p > 0.05)

3.1.3 Serum metabolites

Table 4 shows average serum metabolites values of experimental goats fed selenium at low and high levels during the gestation period. All serum metabolites values were not significant (P>0.05) compared to the control. Serum metabolites glucose, total protein and globulin have higher (P>0.05) values with HS when compared to the control, whereas albumin (3.57±0.51) and cholesterol (69.25±16.87) values with LS were higher (P> 0.05) than the control.

Table 4. Average (mean ± s.d.) serum metabolites values of experimental adult goats fed selenium at low and high levels during the gestation period.

Groups	Parameters				
	Glucose	Total protein	Albumin	Globulin	Cholesterol
A-Control **(0 mg/kg)**	**7.25 ±4.43**	**6.65 ±1.18**	**3.46 ±0.76**	**3.19 ±0.85**	**58.95 ±14.62**
B - LS **(5 mg/kg)**	**7.08 ±4.40**	**6.47 ±0.90**	**3.57 ±0.51**	**2.90 ±1.00**	**69.25 ±16.87**
C- HS **(10 mg/kg)**	**9.25 ±5.02**	**7.26 ±0.43**	**3.43 ±0.47**	**3.81 ±0.69**	**55.50 ±9.35**

HS: High selenium.
LS: Low selenium.
All test groups means were not significant (p > 0.05)

Table 5 shows estimated correlation values of selenium concentration with, total protein and cholesterol concentrations of experimental goats fed selenium at low and high levels during the gestation period. The control group correlations with either total protein (0.06) or cholesterol (0.10) are not significant (p > 0.05). High (0.03) and low (0.17) selenium correlations are not significant (P>0.05) with total protein, but correlation values of high (0.90) and low (0.56) selenium are significant at (p < 0.01) and (P>0.05) respectively with cholesterol.

Table 5. Estimated correlation(r) values of selenium concentration with, total protein and cholesterol concentrations of experimental goats fed selenium at at low and high levels during the gestation period.

Groups	Metabolites	
	Total protein	Cholesterol
A-Control **(0 mg/kg)**	**0.06 NS**	**0.10 NS**
B - LS **(5 mg/kg)**	**0.17 NS**	**0.56***
C- HS **(10 mg/kg)**	**0.03 NS**	**0.90****

LS: Low selenium..
HS: High selenium
*Denotes r-value significant (p <0.05)
** Denotes r-value significant (p <0.01)
NS = not significant (p > 0.05)

Table 6 shows regression equations of selenium, total protein and cholesterol of experimental adult goats fed selenium at low and high levels during the gestation period**.** Neither metabolite total protein or cholesterol in test groups is showing significance (P>0.05) for the regression coefficient. Only test groups Se is showing significance (P<0.05) for the regression coefficient.

3.1.4 Serum enzymes and minerals

Table 7 shows serum enzymes and minerals values of experimental goats fed selenium at low and high levels during the gestation period. All parameters values were insignificant (P > 0.05) compared to the control. Calcium was highest for HS (9.15±0.86) followed by LS and least is the control. For the P, the control value (6.96±2.24) was higher that of the test groups.

Table 6. Regression equation▪ of selenium, total protein and cholesterol of experimental goats fed selenium at at low and high levels during the gestation period.

Groups	Metabolite	Regression coefficient (b)	Constant (c)	S E	Significance
A-Control (0 mg/kg)	**Se**	**0.23**	**42.92**	**0.52**	**NS**
	Total protein	**0.24**	**6.65**	**0.01**	**NS**
	Cholesterol	**0.12**	**62.28**	**0.26**	**NS**
B - LS (5 mg/kg)	**Se**	**0.65**	**14.86**	**0.31**	*****
	Total protein	**0.27**	**6.11**	**0.03**	**NS**
	Cholesterol	**0.10**	**66.75**	**0.51**	**NS**
C- HS **(10 mg/kg)**	**Se**	**0.65**	**15.70**	**0.32**	*****
	Total protein	**0.46**	**7.55**	**0.01**	**NS**
	Cholesterol	**0.10**	**56.94**	**0.28**	**NS**

HS: High selenium.
LS: Low selenium.
All test groups means were not significant ($p > 0.05$)

Table 7. Average (mean ± s.d.) serum enzymes and minerals values of experimental goats fed selenium at low and high levels during the gestation period.

Groups	Parameters				
	Ca	**P**	**Se**	**ALP**	**ALT**
A-Control **(0 mg/kg)**	**8.53 ±1.55**	**6.96 ±2.24**	**14.59 ±3.26**	**84.40 ±61.95**	**33.98 ±29.43**
B - LS **(5 mg/kg)**	**8.72± 0.72**	**6.72± 1.90**	**22.79± 12.94**	**124.79± 83.25**	**35.30± 28.20**
C- HS **(10 mg/kg)**	**9.15± 0.86**	**6.48± 1.94**	**27.79± 14.21**	**159.71± 89.60**	**40.48± 38.58**

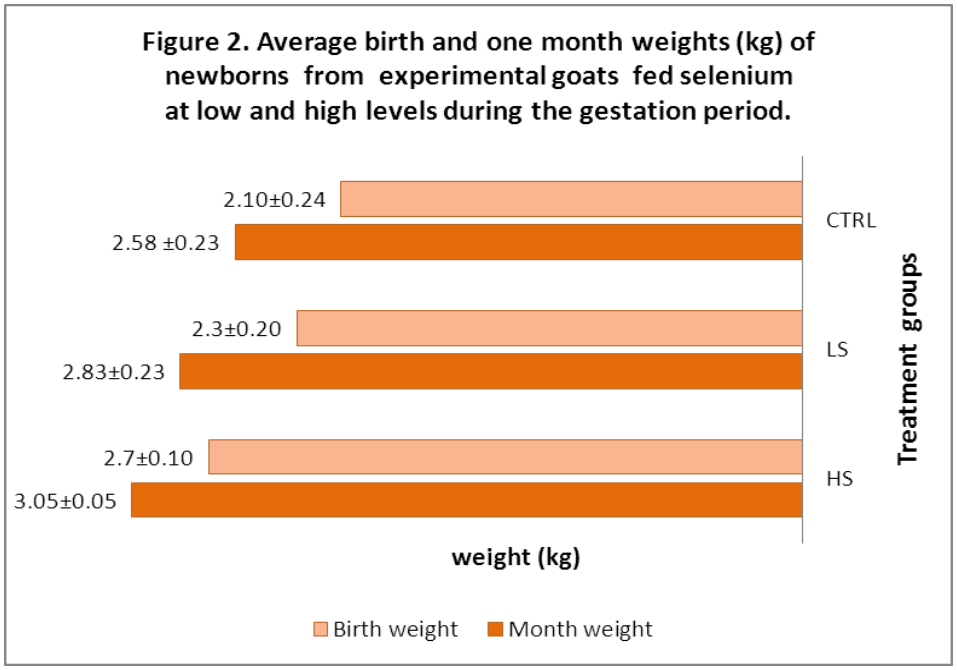
HS: High selenium.
LS: Low selenium.
All test groups means were not significant ($p > 0.05$)

The Se selenium level was highest (P>0.05) for the HS (27.79 ±14.21) followed by LS and the control is least.

Both ALP and ALT enzymes were highest (P>0.05) for the HS (159.71 ±89.60 and 40.48 ±38.58 respectively) followed by LS and the control is least.

3.1.5 Birth weights

Figure 2 shows average birth and one month weights (kg) of newborns from experimental goats fed selenium at low and high levels during the gestation period. Birth and month weights of newborns increased progressively over the control.



When birth to month weights of treatment groups were compared, they were not significant (P>0.05) for the control and significant (P<0.05) for LS and HS. Comparison of birth and month weights of test groups to the control revealed LS (2.30±0.20 and 2.83±0.23 respectively) to be not significant (P>0.05), and likewise HS (2.70±0.10 and 3.05±0.00 respectively) to be significant (P<0.05).

3.2 Effect of selenium synergized with vitamin E on the reproductive performance of Nubian goats

3.2.1 Health record

All experimental goats were healthy althrough the experimental period. Voluntary feed intake was at normal. Behavioural patterns were also normal.

3.2.2 Blood parameters

Table 8 shows average hematological values of experimental goats fed selenium and vitamin E at low and high levels during the gestation period. All hematological values were not significant (P>0.05) compared to the control. Generally for all hematological parameters observed, values of LSLV and HSHV were higher (P> 0.05) than those of the control except for MCH% and MCHC%. The values of the two vitamin synergized selenium levels were closely similar (P>0.05) in values.

Table 8. Average (mean ± s.d.) hematological values of experimental goats fed selenium and vitamin E at at low and high levels during the gestation period.

Groups	Parameters							
	RBC $\times 10^6$ mm	**WBC** $\times 10^3$ mm	**Lympho-cytes**	**Lympho-cytes %**	**MCV** %	**MCH** %	**MCHC** %	**Hb** g/dl
A-Control **(0 mg/kg + 0 IU/g)**	**2.06 ±0.72**	**13.22 ±4.93**	**5.58 ±4.22**	**40.01 ±27.10**	**98.75 ±19.99**	**36.60 ±7.42**	**36.77 ±9.14**	**7.50 ±1.98**
B - LSLV **(5 mg/kg + 250 IU/g)**	**2.47 ±0.26**	**17.74 ±5.05**	**7.32 ±3.83**	**40.02 ±22.25**	**102.64 ±10.22**	**35.09 ±7.13**	**35.87 ±4.05**	**9.00 ±0.89**
C - HSHV **(10 mg/kg + 500 IU/g)**	**2.51 ±0.34**	**17.17 ±5.51**	**6.88 ±3.83**	**45.74 ±26.57**	**105.41 ±8.97**	**35.63 ±1.88**	**34.03 ±3.35**	**8.89 ±0.92**

LSLV: Low selenium low vitamin.
HSHV: High selenium high vitamin.
All test groups means were not significant (p > 0.05)

3.2.3 Serum metabolites

Table 9 shows average serum metabolites values of experimental goats fed selenium and vitamin E at low and high levels during the gestation period. All serum

metabolites values were not significant (P>0.05) compared to the control. Serum metabolites glucose, albumin and cholesterol have higher (P>0.05) test groups values when compared to the control, whereas total protein and globulin test groups values were higher (P> 0.05) than the control.

Table 9 . Average (mean ± s.d.) serum metabolites values of experimental goats fed selenium and vitamin E at at low and high levels during the gestation period.

Groups	Parameters				
	Glucose	Total protein	Albumin	Globulin	Cholesterol
A-Control **(0 mg/kg + 0 IU/g)**	**7.25 ±4.43**	**6.65 ±1.18**	**3.46 ±0.76**	**3.19 ±0.85**	**58.95 ±14.62**
B - LSLV **(5 mg/kg + 250 IU/g)**	**8.00 ±4.98**	**6.55 ±0.36**	**3.83 ±0.49**	**2.72 ±0.60**	**62.81 ±7.56**
C - HSHV **(10 mg/kg + 500 IU/g)**	**7.96 ±4.68**	**6.38 ±0.41**	**3.65 ±0.40**	**2.73 ±0.55**	**69.42 ±11.47**

LSLV: Low selenium low vitamin.
HSHV: High selenium high vitamin.
All test groups means were not significant (p > 0.05)

Table 10 shows estimated correlation values of selenium concentration with, total protein and cholesterol concentrations of experimental goats fed selenium and vitamin E at low and high levels during the gestation period. The control group correlations with either total protein (0.06) or cholesterol (0.10) are not significant (p > 0.05). HSHV (0.51) and LSLV (0.97) selenium correlations are significant at (P>0.05) and (p < 0.01) respectively with total protein, but selenium correlation values of HSHV (0.85) and LSLV (0.82) are both significant (p < 0.01) with cholesterol.

Table 10. Estimated correlation(r) values of selenium concentration with, total protein and cholesterol concentrations of experimental goats fed selenium and vitamin E at at low and high levels during the gestation period.

Groups	Metabolites	
	Total protein	Cholesterol
A-Control **(0 mg/kg + 0 IU/g)**	**0.06 NS**	**0.10 NS**
B - LSLV **(5 mg/kg + 250 IU/g)**	**0.97****	**0.82****
C - HSHV **(10 mg/kg + 500 IU/g)**	**0.51***	**0.85****

LS: Low selenium..
HS: High selenium
*Denotes r-value significant ($p < 0.05$)
** Denotes r-value significant ($p < 0.01$)
NS = not significant ($p > 0.05$)

Table 11 shows regression equations of selenium, total protein and cholesterol of experimental adult goats fed selenium and vitamin E at different concentrations during the gestation period. All regression coefficients of the treatment groups in Se, total protein and cholesterol were not significant ($p > 0.05$).

3.2.4 Serum enzymes and minerals

Table 12 shows average serum enzymes and minerals values of experimental goats fed selenium and vitamin E at low and high levels during the gestation period. All test groups values were insignificant ($P > 0.05$) when compared to the control except for Se ($P< 0.05$). There is a better ($P> 0.05$) response of Ca in the treatment HSHV (9.23±0.97). For the P, ($P> 0.05$), the control (6.96±2.24) was higher when compared to the other test groups with the least value (5.96±1.15) recorded by the HSHV.

Table 11. Regression equation■ of selenium, total protein and cholesterol of experimental goats fed selenium and vitamin E at low and high levels during the gestation period.

Groups	Metabolite	Regression coefficient (b)	Constant (c)	S E	Significance
A-Control **(0 mg/kg** + 0 IU/g)	**Se**	**0.23**	**42.92**	**0.52**	**NS**
	Total protein	**0.24**	**6.65**	**0.01**	**NS**
	Cholesterol	**0.12**	**62.28**	**0.26**	**NS**
B - LSLV **(5 mg/kg** + 250 IU/g)	**Se**	**0.15**	**34.08**	**0.48**	**NS**
	Total protein	**0.31**	**6.72**	**0.01**	**NS**
	Cholesterol	**0.05**	**63.33**	**0.28**	**NS**
C - HSHV **(10 mg/kg + 500 IU/g)**	**Se**	**0.17**	**28.63**	**0.92**	**NS**
	Total protein	**0.21**	**6.50**	**0.01**	**NS**
	Cholesterol	**0.04**	**7 0.11**	**0.35**	**NS**

LSLV: Low selenium low vitamin.
HSHV: High selenium high vitamin.
■ Lianer regression equation y = bx+c.
NS = not significant (p >0.05).

Table 12. Average (mean ± s.d.) serum enzymes and minerals values of experimental goats fed selenium and vitamin E at low and high levels during the gestation period.

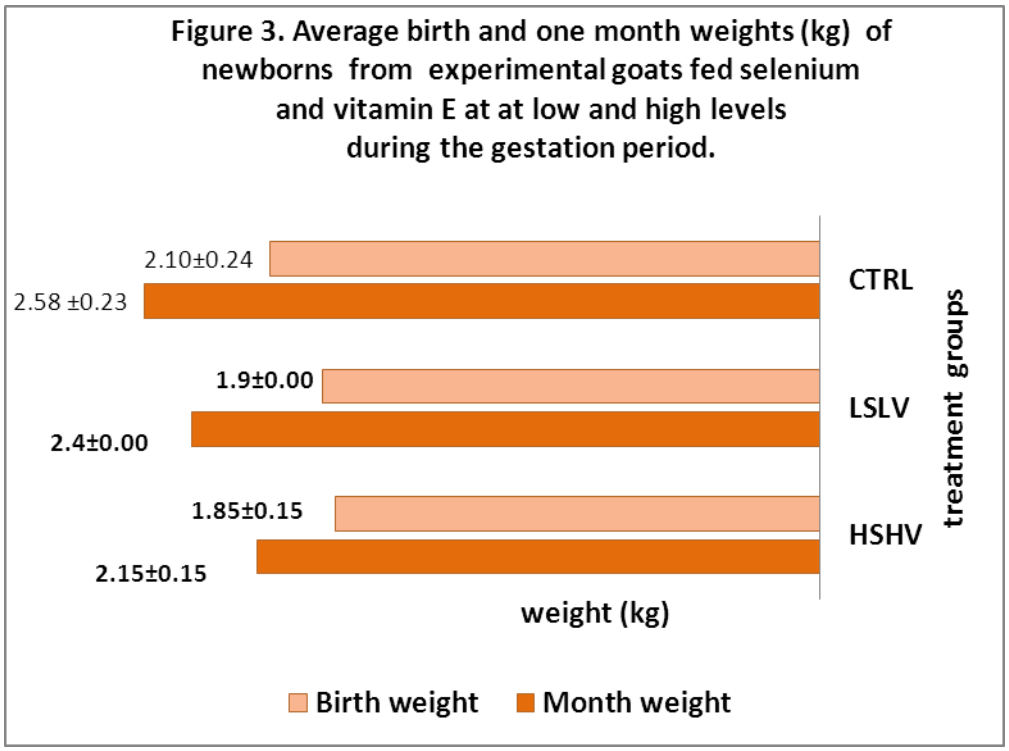
Groups	Parameters				
	Ca	**P**	**Se**	**ALP**	**ALT**
A-Control **(0 mg/kg + 0 IU/g)**	**8.53 ±1.55**	**6.96 ±2.24**	**14.59 ±3.26**	**84.40 ±61.95**	**33.98 ±29.43**
B - LSLV **(5 mg/kg + 250 IU/g)**	**9.09 ±0.77**	**6.71 ±1.79**	**36.47* ±13.73**	**170.63 ±62.31**	**33.43 ±33.75**
C - HSHV **(10 mg/kg + 500 IU/g)**	**9.23 ±0.97**	**5.96 ±1.15**	**36.67* ±12.62**	**183.67 ±99.37**	**35.32 ±31.53**

LSLV: Low selenium low vitamin.
HSHV: High selenium high vitamin.

Test groups values for Se were significant (P<0.05) when compared to the control. Both test groups values for enzymes ALP and ALT were not significant (P>0.05) when compared to the control, with HSHV having higher responses for ALP (183.67 ±99.37) and ALT (35.32±31.53)

3.2.5 Birth weights

Figure 3 shows average birth and one month weights (kg) of newborns from experimental goats fed selenium and vitamin E at low and high levels during the gestation period. Birth and month weights of newborns decreased retrogressively under the control.



When birth to month weights of treatment groups were compared, they were not significant (P>0.05) for the control and HSHV, but significant (P<0.05) for the LSLV. Comparison of birth and month weights of test groups to the control revealed both LSLV and HSHV to be not significant (P>0.05).

CHAPTER FOUR

DISSCUSION

The results of this study showed for the serum metabolites. The value of the experimental adult goats fed selenium and vitamin E at different levels during the gestation period although the mean of test groups expressed a non significant ($p>0.05$) except of ALT when compared with the control, there were some responses of those metabolites to different treatments. The glucose level expressed some response for the treatments with Hs (9.25±5.02) alone and Ls Lv (8.00±4.98) treatments when compared to the control.

For total protein with the Hs (7.26±0.43) better response compared to all other treatments. This might be due to bioavailability was observed effect of this element. Such results might agree with the report of **Yeh, *et al.* (1997) Hakarainen (1993) and Asplia (1991).** As for the Albumin level the combination of Ls Lv gave better results (3.83±0.49) compared to the other treatment groups and the control this might indicate the necessity of symergism of those two elements at a certain level **(Awadeh *et al.*, 1998).** For the Globulin, its level in this experiment indicated a better response to the treatment with Hs alone (3.81±0.69) compared to all other treatments. Concerning the cholesterol level, the effect of Hs Hv treatments is more (69.42±11.47) compared to all other treatments. Such results confirm those findings of **Awadeh *et al.* (1998),** who reported the effective role of Se + vit E in the metabolism and cell physiology and this might illustrate the synergistic action of vit. E in the bioavailability of the Se element and support the findings of **Varly (1967) and Weiss *et al.*(1997)** who reported the synergistic action of vit. E.

The action of Se and vitamin E are synergistic and many studies have demonstrated a reduction or elimination of deficiency symptoms where either component is used **(Weiss, *et al.*, 1997).** The view that measuring accumulation of selenium in muscle without the addition measurement of functional parameter of selenium, the activity of glutathione perexidase, does not sufficiently describe

bioavailability of selenium. This does not mean that there is no correlation between tissue accumulation, enzyme activity and selenium status. It has been repeatedly demonstrated that a good correlation exists between these variables in situations of deficient or margined selenium supply **(waghorn, 1986).**The results of this research supported the above reports. For the mineral metabolites, the result of study showed the effect of the different treatments is insignificant ($p>0.05$) compared to the control. But there is a better response of Ca to the treatments Hs Hv (9.23±0.97) followed by Hs (9.15±0.86), Ls Lv (9.09±0.77) level. For the P, Although non significant ($p>0.05$) between the different treatment was observed, the control (6.96±2.24) gave better response to that element, when compared with the different treatment group with the least effect in the treatment group having Hs Hv (5.96±1.15).

The (Se) selenium level in the experimental groups showed controversial result of this element, since indicated a higher level in Hs Hv (36.67±32.62) then followed by Ls Lv (36.47±13.73) when compared to all other tested group. This might illustrate the synergistic action of vit. E and Se elements, **(Varly, 1967).** From another point this controversial view disagreed with the report of **Weiss *et al.* (1997)** who stated that this action of Se and vit.E are synergistic and many studies have demonstrated a reduction or domination deficiency symptoms where either component is used. Concerning the level of enzymes in the serum of the experimental groups, the results of this study showed significant ($P<0.05$) response of the ALP enzymes in all treatment groups compared to the control (84.40±61.95) (table 3), with a noticable effect of Hs Hv (183.67±99.37) treatment followed by Ls Lv (170.63±62.31) then, Hs (159.71±89.60) and Ls (124.79±83.25) treatment, compared to the control (84.40±61.95) group. This indicated clearly the effect, of Se (selenium) and the synergistic role of vit. E. These results might agree with Conclusion of **Hanten *et al.* (1998)** who report the synergistic role of Se and vit. E affects the biological process on the body. The same effect was obtained concerning

the effect the level of Se (selenium) on ALT enzyme as Hs (40.48±38.58) followed by Ls (35.30±28.20) gave better results then followed by Hs Hv (35.32±31.53) and Ls Lv (33.43±33.75). Aside from the effects of selenium in incorporated into several selenoprotiens with specific functions, it is important for an understanding of selenium metabolism to note that all of these selenoprotiens contain selenium as one or more selenocystyl residues within the peptide chain. **Shelton (1990)** replacement of selenocysteine by cystiene in these proteins will result in a marked decrease or even complete loss of specific function. The exercise intensity used in increased plasma selenium in both treatment groups. The source of this increased plasma selenium may have been the red cells **(Mahan** and **Parrett, 1996).** Since there was a trend towards lower RBC selenium following exercise, plasma selenium in those fed the inorganic source returned to pre-exercise levels while plasma selenium in those given sel-plex remained elevated at 24 hrs post-exercise. Perhaps part of the selenium that mobilized from RBC in the slenite group during exercise was voided in the urine. **Dargtz** and **Ross (1996)** reported that overall, based on blood Se concentrations, 7.8% of the samples were severely deficient and another 10.4% were considered marginally deficient in 40% of the herds that supplemented with Se, this might reflect that supplementation was not adequate for the herds. Based on this survey, the relatively low level of supplementation recommended for beef cattle (**NRC, 1996)** of 0.1 to 0.2 ppm may not be adequate and its recommendation is for total dietary Se and not supplemental Se.

For the result, of hematological study the experimental goats fed Se (selenium) and vitamin E at different concentrations and combination rates during the gestation period, in this study, although reflecting, non significant ($p>0.05$) among all the treatment groups specially RBC when compared with the control, but there are some responses of the blood component WBC to the different treatment, since Hs Hv gave better results followed by Ls Lv followed by Ls then Hs when compared with the control. Those results deem the importance of those two

elements in the cell immnulogy. The feeding of selenium yeast has the effect of rapidly raising blood Se levels in the initial stages of lactation which in turn will benefit in rebreeding if cows are fed a dry cow ration through the dry period as well. Selenium has been recognized as essential trace element in lives-enzyme and hence glutathione peroxidase makes selenium along with vitamin E, important in reproduction and immunity along with a wide range of metabolic and digestive roles according to **Casey, (1992). Larson (1988)** who observed a trend for increase 1g concentration in Se supplemented ewes and lambs. Calves failing to absorb enough 1g have higher risk of morbidity **(McGuire *et al.*, 1976)** in addition; **Larson (1988)** reported significant increased titers to telanus toxioid in Se supplemented lambs. **Turner** and **Finch (1990)** reported a decreased lymphocyte response in lambs deficient in vitamin E and selenium.

Average birth, one month and weight gain (kg) of newborns from goats treated with dietary (Se) selenium, vitamin E during the gestation period Ls gave better result for body weight compared with other treatment then followed the control group this might reflect the dose effect of those level of this element. Results of this study confirmed the finding of **Larson (1988).**

Preston, (2000) reports have suggested that vitamin E and selenium (Se) are one of the important nutrients that can affect biological processes in the body such as immunity **(Hemken *et al*., 1998).** Metabolism **(Awadeh *et al*., 1998)** and reproduction **(Jarry, 1996)** in addition vit. E and Se improved spermatogenesis and semen quality **(Marin-Guzman, 1990; Bezezinsks- Slebodzinska *et al*., 1995 and Marin-Guzan *et al*., 1996).**

The results of this study agree with almost all of the above researchers abide the controversial views created by the scarce information on the actual role of (Se) selenium elements in the cell physiology and blood constitment. Despite the effective role in improving the fertility and reproduction, as stated by **Jarry (1996)**; and also the nature of bioavailability of these elements, are beyond the scope of this

study. The results of reproduction in this experiment illustrated a non significant difference among all the experimental animals. The high birth weight in the group treated with Hs level might be attributed to the sex effect of the birth, since males are born heavier than female kids.

Despite the non significant results among all the treatment, still there were some insignificant effect of adding Se and vit. E to treatment groups. Such results did not consent with most of the results of many other workers, stated in this study. The discrepancy of the results in this study might seek the support of **Combs** and **Combs (1986)** who stated that dietary requirements based solely on the Se content of the diet are difficult to do because many factors, such as feed stuff and the form of the Se, vary with respect to bioavailability and other dietary factors and can either enhance or decrease, its availability. The quality and quantity supply of feed stuff (roughages) offered during this experiment, might be the reason affecting the bioavailability of the selenium elements and hence reflecting a non-significant effect in all treatment animals.

CONCLUSION AND RECOMMENDATIONS

Although there is general consensus by most of researchers about the significant effect of Se on reproduction, the results of this study showed a non-significant effect in treatments groups. It has been concluded from those results that the quality and quantity of feed supplied to the experimental animals might have affected the bioavailability and the absorption of Se and hence gave conflicting results.

So this work may suggest the following recommendations:-

- Soil analysis for all micro elements should be carried out to help in vision of the availability of these elements in the grazing areas.
- Chemical analysis of the feed stuff should be done before suggesting the levels of supplementation to any animal.
- Further studies are needed to investigate on the different forms of selenium bioavailability and absorption in animal cells, and hence ;
- More studies are needed to determine in the effective dose of Se and the form of its availability and the actual role of vit. E as a synergistic factor.

REFERENCES

Aharoni, Y.; Nachtomi, E.; Holstein, P.; Brosh, A.; Holzer, Z., andNitsan, Z. (1995). Dietary effects on fat deposition and fatty acid profles in muscle and fat depots of Friesian bull calves. J. Anim. Sci., 73:2712-2720.

Anundi, I., J. Högberg and A. Stahl. 1984. Absorption of selenite in the rat small intestine: interactions with glutathione. Acta pharmacol. Toxicol. 54:273-277.

AOAD (1990). Goat Resources in Arab States. 11. Sudan. Arabic edit., Arab Organization for Agricultural Development. AOAD Printing press, Khartoum, Sudan.

AOAC (1980). Official Methods of Analysis. Association of Official Analytical Chemists. 12th ed., Washington, USA.

Andrews, E.D., W.J. Hartley, and A.B. Grunt. (1968). Selenium-responsive diseases of animals in New Zealand. N.Z. Vet. J. 16:3.

Ardüser. F., S. Wolffram and E. Scharrer. (1985). Active absorption of selenate by rat ileium, J. Nutr. 115:1203-1208.

Asplia, P. 1991. Metabolism of Selenite, Selenomethionine and Feeding Corporated Selenium in Lactating Goats and Dairy Cows. J. Agric. Sci. Finland, 63: 1-74.

Ardüser. F., S. Wolffram, E. Scharrer and B. Schneider. 1986. Transport of selenate and selenite across the brush border membrance of rat and sheep small intestine. Biol trace Elem. Res. 9:281-290.

Awadeh, F.T., Kincaid, R.L. and Johnson, K.A. (1998). Effect of level and source of dietary selenium on concentration of thyroid hormones and immunoglobulin in beef cows and calves. J. Anim. Sci. 76:1204-1215.

Barnes, T.G.; Varner, L.W.; Blankenship, L.H.; Fillinger, T.J. and Heineman, S.C. (1990). Macro and trace mineral content of selected south Texas deer forages. J. Range Managmt., **43**: 220 –223.

Bearden, H.J. and Fuquay, J.W. (1984).Applied animal Reproduction 2nded. Reston Publishing Co. Verginia, U.S.A.

Bello, A. N. (1985). Performance and Carcass Characteristics of Sudan Desert Kids Compared with their Temperate Crosses. M.V.Sc. Thesis, University of Khartoum, Sudan.

Blood, D.C; Radostets, O. M. and J.A. Henderson. (1983). Veterinary Medicine Bailliere Tindall, London.

Bray, T.M., and Taylor, C. G.1993. Tissue Glutathione Nutrition and Oxidative Stress. Cam. J. Physiol. Pharmacology. 71:746-751.

Brzezinsks-Slebodzinsha. E.; Sledodzinsk. A.B.; Pietras.B. and **Wieczorek. G. (1995).** Antioxidant effect of vitamin E and glutathione on lipid peroxidation in boar semen plasma. Boil trace Elem. Res. 47: 69-74.

Burk, R.F. and K.E. Hill. 1992. Some properties of selenoprotein. P. Biol. Trace Elem. Res. 33:151-153.

Burk, R.F. and **K.E. Hill. 1994**. Selenoprotein P.A selenium-rich extracellular glycoprotein, J. Nutri. 124:1891-1897.

Burk, R.F. and K.E. Hill. M.E; Boeglin, F.F. Ebner and H.S. Chittum. (1997). selenoprotein passociates with endothelial cells in rat tissues. Histochem. Cell Biol. 108:11-15.

Burk, R.F. (1991). Molecular biology of selenium with implications for its metabolism. FASEBJ. 5:2274-2279.

Blaxter, K.L. (1962). Muscular dystrophy in farm animals: Its cause and prevention. Proc. Nutr. Soc. 21:211.

Butler, J.A., M.A. Beilstein and **P.D. Whanger, (1989).** Influence of dietary methionine on the metabolism of selenomethionine in rates, J. Nutr. 119: 1001-1004.

Cantor, A.H. 1997. The role of selenium in poultry nutrition. In: Biotechnology in the feed industry, proceedings of the 13th Annual symposium. (T.P. Lyons, and K.A. Jacques, eda.) Nottingham University Press. U K. P.199.

Carnier, F.; Benoit, E.; Jacquet, J. P. and Telatour, P. (1984). Blood enzymology in the goat: Usual values of CPK, LDH, ICDH and SDH. Ann. Res. Vet., 15: 55-58.

Casey, N. **H. (1992).** Goat meat in human nutrition. International Conference on Goat, University of Pretoria, South Africa.

Castro, A.; Dhinsa, D. S.; Hoversland, A. S.; Malkus, H.; Rosenthiel, C. and Metcalf, J. (1977). Serum biochemistry values in normal pygmy goats. Amer. J. Vet. Res., 38: 2085 – 2087.

Clariget, R. P.; Forsberg, M. and **Rodriguez-Martinez, H. (1998)**. Seasonal variation in live weight, testes size, testosterone, LH secretion, melatonin and thyroxine in Merino and Corriedale rams in subtropical climate. Acta Vet. Scand., 39: 35–47.

Clark, L.C., G.F.; Combs, Jr.; B.W.Turnbull; E.Slate; D.Chalker; J.Chow; L.Davis; R.Glover; G.Graham; E. Gross; A.Kongrad;J.Lesher; K.Park;B. Sander; C.Smith and **J.Taylor. (1997)**. Effect of selenium supplementation for cancer prevention in patient with carcinoma of the skin: a randomized clinical traial.J. Am.Med.Assoc.(Cited by Combs. 1997).

Chow, C.K., 2001. In: R. Rucker, D.B. Suttle, McComick, L. Machlin and J. Editors, 2001. Handbook of vitamins. Marceldekker, New York, pp: 165-196.

Combs, G.F. and S.B. Combs, (1985). Absorption and transfer. In: the role of selenium in Nutrition. Academic press. Newyork.

Combs, G.F., and S. B. Combs. **(1986).** the role of selenium in nutrition.

Academic Press, Inc. Orlando. Florida.

Dargatz, D.A. and Ross P.F. (1996). Blood selenium concentrations in cows and heifers on 253 cow- calf operations in 18 states. J. Anim.Sci. 74:2891.

Darrag, A. A. (1994). The range management to control desertification. Workshop, Ministry of Agric. and Natural Resources, Khartoum, Sudan.

Devendra, C. and **Burns, M. (1983).** Goat Production in the Tropics. Commonwealth Agricultural Bureau, Faraham Royal, U.K., 182pp.

Devendra, C. and **Meleroy, G.B. (1982).** Goat and sheep production inthe tropies. 1st ed., Intermediate tropical Agriculture Series, Lonman Co.Ltd., London.

Doumas, B. T.; Watson, W. A. R. and Biggs, H. G. C. (1971). Albumin standard and the measurement of serum albumin with bromocersol green. Clin. Chem. Acta, 31:87-96.

Edens, F.W. (1996). Organic selenium: from feathers to muscle integrity to drip loss. Five years onward: on more selenite! In: Biotechnology in the feed industry. Proc. of the 12th Annual symposium.

El Naim, Y. A. (1979). Some Productive Traits of Sudan Nubian Goat. M.V.Sc. Thesis, University of Khartoum, Sudan.

El Naim, Y.A. (1979). Some reproductive traits of Sudanese Nubian goats. MVSc. Thesis. Uof K Sudan.

Evans, G and Maxwel.W.M.C. (1987). Salamons artificial insemination of sheep and gaots. 2nd ed., Sydney, Australia, Butlerworth.

Fisher, D. D., R. D. Elliott, S. W. Saxton and Beatty. (**1995).** Effects of selenium source selenium status of lactation cows. Vet. Clin. Nutr. 2:68.

Forbes, J. M. (1995). Voluntary Food Intake and Diet Selection in Farm Animals. Commonwealth Agricultural Bureau (CAB), Farnham, Royal Bucks, U.K.

Foster, D.L. (1994). Puberty in the female sheep. Physiology of Report. (2nd ed) 2: 411-452.

Frape, D. (1998). Equine Nutrition and feeding. (2nd Ed.). Blakwell Science Ltd UK.

Gall, C. (1996). Goat Breeds of the World. CTA, Akershiem Margraf Institute for Anim. Prod., Germany.

Godwin, K.O.(1975). The role and te metabolism of Selenuim in the animal. In "trace element in soil-plant-Animal Systems"(D.J.D. Nicholas and A.R.Egan,eds.), p.259. Academic Press, New York.

Goetsch, A. L.; Galloway, D. L. and Patil, A. R. (1998). Arterial amino acid concentrations in sheep consuming forage diets. Small Rumin. Res., 29: 51–60.

Graham. N. M. and **Searle.T.W. (1975). S**tudies in weaner sheep during and after a period of weight stasis i.E nergy and nitrogen utilization Aust.j.agric.RES.**26**:34

Green, J. T. Jr. (1999). Potential for producing meat goats in North Carolina. http:/goats.clemson.edu/.

Giterson, A. L.; Lie Fong, L. A. S.; Wederfoort, I. E. and Schonten, H. (1971). Blood, urine and liquor sugar II. Clin Chem. Acta, 34:489-490.

Grimaud, P. and Doreau, M. (1995). Effect of extended underfeeding on digestion and nitrogen balance in nonlactating cows. J. Anim. Sci., **73**: 211–219.

Grimaud, P.; Richard, D.; Kanwé, A.; Durier, C. and Doreau, M. (1998). Effect of underfeeding and refeeding on digestion in *Bos taurus* and *Bos indicus* in a tropical environment. J. Anim. Sci., **67:** 49 –58.

Grimaud, P.; Richard, D.; Vergeron, M. P.; Guilleret, J. R. and Doreau, M., (1999). Effect of drastic undernutrition on digestion in Zebu cattle receiving a diet based on rice straw. J. Dairy Sci., 82: 974–981.

Gulteridge, R. C. and Shelton, H. M. (1994). Forage tree legumes. In: Tropical Agriculture. Department of Agriculture, University of Queensland, Newzealand.

Guss, S.B. (1977). Management and diseases of dairy goats. Dairy Goat Journal Publishing Corporation, Scottsdale, Arizona.

Hafez, R.H.F. and **Hafez. B. (2000).** Reproduction in farm animals. (7^{th}ed.) Lippincott Wilkins.

Hakkarainen, J. 1993. Bioavailability of Selenium, Norw. J. Agric. Sci. Suppl. 11: 21- 35.

Hall, J.B., Schillo, K.K. Hileman, S.M. and Boiling, J.A. (1992). Dosetyroine act as anational signal mediating the effect of increased feed intake on lutenizing hormone patterns in growth restricted lambs. Biol. Report 46:573-582.

Hanten, W. P.; D. D. Dawson; M. Lwata; M. Seiden, F. G. Whitten and Zink 1998. Craniosacral Rhythm : Retiability and Relationships with Cardiac Therapy. 27 (3): 213 - 218.

Hartley, W.J., and A.B. Grant. 1961. A review of selenium responsive diseases of New Zealand livestock. Fed. Proc. Fed. Am. Sec.Exp. Biol. 20:679.

Hemken, R.W., Hovman, R.J. and Trammell, S. (1998). Selenium for dairy cattle. Arole for organic selenium in biotechnology in feed industry. Proc. Altech 14^{th}, Ann. Symp. T. Plyons and K.A. Jacaues Eds. Nottingham Univ. Press.

Henry, P .R. and C .B. Ammerman. (1995). Selenium Bioavailability. In: Bio-availability of Nutrition of Animals, Amino acids, Minerals, and Vitamins.

V.B. Ammerman, D.H. Baker and A.J.Lewis (Eds.) Academic Press, New york.

Hicks, R. B.; Owen, F. N. Gill, D. R.; Oltjen, J. W and **Lake, R. P. (1990).** Dry matter intake and feedlot beef steers. Influence of initial weight, time on feed and season of the year. J. Anim. Sci., 168: 254 – 264.

Hogan, J.S., W.P. Weiss and K.L. Smith, 1993. Role of vitamin E and Selenium in host defense against mastitis. J. Dairy Sci.,**76**: 2795-2803.

Hogue, D.E. 1985. Vilamin E, selenium and other factors related to nutritional muscular dystrophy in lambs. Prco. Cornell Nutr. Conp. Ithaca, NY. Pp 32-34.

Hooda, O. K. and Naqvi, S. M. K. (1990). Effect of thermal load and feed restriction on relative adaptability of Malpura and Avikalin sheep in semi-arid region. Ind. J. Anim. Sci., 60, 608–611.

Hunter, R.H.F. (1980). Differentiation, puberty and oestrus cycle in physiology and technology of reproductive in female domestic animals, 1-23. Academic Press, London.

ILRI, (1996). Goat types of Ethiopia and Eritrea. Physical description and management systems. International Livestock Research Institute. Farm-Africa London, UK.

Jagusch, K. T.; Duganzich, D.M.; Kidd, G.T. and Church, S. M. (1983). Efficiency of goat milk utilization by milk-fed-kids. N. Z. J. Agric. Res., **26**: 443-445.

Jarry, D.O. **(1996).** The role of selenium and Vit. E in mastitis and reproduction of dairy cattle Irish veterinary J. 49: 362-364.

Kaneko, J. J. (1980). Clinical Biochemistry of Domestic Animals. 3rd ed., Academic Press, New York.

Keery, C. M. and Amos, H. E. (1993). Effects of source and level of undegraded intake protein on nutrient use and performance of early lactation cows. J. Dairy Sci., **76:** 499–513.

Kelly, F. J. 1993. Glutathione content of the small intestine Regulation and Function.Br.J.Nutr.**69**:589-596.

Kelly, M.P., and R. F. Power. (1995). Fractionation and identification of the major selenium compounds in selenized yeast. J. Dairy Sci. 78 (suppl. 1): 237 (Abstr.).

Kelly, W. R. (1984). Veterinary Chemical Diagnosis. Bailiere Trindal, London.

Kincail, R.L. (1995). The biological basis for selenium requirements of animals. The prof. Anim. Sci. 11:26.

Kolb, E., S. KasKousb and J. Scchawer, 1997. Nutrional aspects of the importance, utilization, metabolism and the use of vitamin E and selenium in sheep. Berl. Munch. Tierarztl. Wschr. **110**: 178-184.

Koening, K.M., L.M. Rode, L.M. Cohen and W.T. Bucklet. 1997. Effect of Diet and Chemical Form of Selenium on Selenuim Metabolism in Sheep. J. Anim. Sci. 75: 817 – 827.

Koyuncu, M. and H. Yerlikaya, 2007. Effect of selenium-vitamin E injection of ewes on reproduction and growth of their lambs. S. Afr. J. Anim. Sci., 37: 233-236.

Koyuncu, M., Kara Uzun, S., Ozis, S. & Yerlikaya, H., 2006. Effects of selenium-vitamin E or progestagen-PMSG injections on reproductive performance of ewes. J. Appl. Anim. Res. 29, 137-140.

Lasen, H.J. 1980. Influence of selenium on antibody production in sheep. REs. Vet. Sci. 45:4.

Larson, H. J. 1988. Influence of Selenium on Antibody Production in Sheep. Res. Vet. Sci. 45: 48.

Lawrence, T. L. J. and Pearce, J. (1964). Some effects of wintering yearling beef on different planes of nutrition. I. Liveweight gain, food consumption and body measurement changes during the winter period and the subsequent grazing period. J. Agric. Sci., **63**: 5 – 34.

Lindsay, D.R., Martin, G.B. and Williams, I.H., (1993). Nutrition and Reproduction. Reproduction in Domesticated Animals. World Anim. Sci. Series Pp. 459-491.

Luginbuhl, J. M. (1998). Breeds of goats for meat production and production traits. http://www.cals.ncsu.edu/

Lyons, (1997). The new era in animal nutrition: arrival of scientifically proven natural alternatives. In: Biotechnology in the feed industry, proceeding of the 14th Annual symposium. (T.A. Jacques, eds) Nottingham University Press, Nottingham. UK.

Maas, J.P., M.S. Bulgin and B.C., Anderson. 1984. Nutritional myodegeneration associated with vitamin E deficiency and normal selenium status in lambs. J. Am. Vet. Med. Assoc. 184:201.

McDowell, L.R. 1992. "Minerals in animal and human nutrition." Academic Press, San Diego, California.

Meschy, F., 2000. Recent progress in the assessment of mineral requirements of goats. Livest. Prod. Sci., 64, 9-14.

Mahan, D.C. and Y.Y. Kim. (1996). Effect of inorganic or organic selenium at two dietary levels on reproductive performance and tissue selenium concentrations in first parity gilts and their progeny. J. Anim. Sci. 74:2711-2718.

Mahan, D.C. and N.A. Parrett. **(1996).** Evaluating the efficacy of selenium enriched yeast and sodium selenite on tissue selenium retention and serum glutathione peroxidase activity in grower and finisher swine. J. Anim. Sci. **74**:2967.

Marin-Guzman (1990). Studies evaluating dietary selenium and vitamin E on semen quality in vivo oocyte fertilization spermatozoa ultrasturcture and testicular histology of boars. Ph.d thesis Ohio State univ. Columbus. C citied by Marin Guzman. 1997.

Marin- Guzman J.D. Mahan, Y.K. Chung, J.L. Pate and Pope, W.F. (1996). Effect of dietary selenium and vit. E on boar performance and tissue response semen quality, and subsequent fertilization rate in mature gilts. J. Anim. Scei. 1997. 75: 2994-3003.

Martin.G.B and Walked- Brown, S.W. (1995). Nutritional influence on reportduction in mature make sheep and goats. J.Reprod. Fertility Supp 1.1) 49:437:449.

McDonald, E.E. (1975). Veterinary endocrinology and reproduction 3rd Lea and Febiger. Phildelphia.

MeGuire, T.C., N.E. Pfelffer, J.M. Weiked and **R.C. Bartch**.(**1976).** Failure of colostral immunologbulim transfer in Calves from infections disease. J.Am. Vet. Med. Assoc. 169:713.

Melby, E. C. and Altman, N. H. (1976). Handbook of Laboratory Animal Science. Vol. III. E.C. Melby and N.H. Altman, (eds.), CRC Press, Cleveland.

Milford, R. and Minson, D. J. (1966). Intake of tropical pasture species. Proc. 9th Int. Grassld. Congr. Sao Paulo, pp 815–822.

Morand–Fehr, P.; Boutonnet, J. P.; Devendra, C.; Dubeuf, J. P.; Haenlein, G. F. W.; Holst, P.; Mowlem, L. and Capote, J. (2004). Strategy for goat farming in the 21st Century. Small Rumin. Res., 51:175-183.

Mosoni, L.; Malmezat, T.; Valluy, C.; Houlier, M. L.; Attaix, D. and Mirand, P. P. (1999). Lower recovery of muscle protein lost during starvation in old rats despite a stimulation of protein synthesis. Amer. J. Physiol., 277: 608–616.

Muth, O.H.; J.E. Oldfield, L.F. Remmert and **J.R. Schubert,(1957**). Effect of Selenium and vitamin E on white muscle disease scienc 128:1090.

Ndibualonji, B. B.; Dehareng, D. and Godeau, J. M. (1997). Influence de la mise à jeun sur l'amidoacidémie libre, l'urémie et la glycémie chez la vache laitière. Ann. Zootech., 46: 163–174.

Nolan, J. V. (1993). Nitrogen kinetics. In: Qualitative Aspects of Ruminant Digestion and Metabolism. J. Forbes and J. France, (eds.),Commonwealth Agricultural Bureau (CAB), Farnham, Royal Bucks, U.K., pp. 123–144.

NRC. (1983). Selenium in Nutrition (Rev. Ed.). National Academy of Sciences-National Research Council, Washington, D.C.

NRC,(1989). Nutrient Requirements of Dariy cattle. 6th reviseded National Academy press. Washington, DC.

NRC (1996). Nutrient Requirements of Beef Cattle. 6th ed. National Research Council, National Academy Press, Washington, pp. 16–21.

Olson, O.E., E. J. Novack. E. I. Whitchead, and I. S. Palmer, (1970). Investigation on selenium in wheat. Photochemistry **9**:1181.

Patteson, E.L., R. Milstrey, and E.L.R. Stokstad. 1957. Effect of Selenium in Preventing exudative diathesis in chicks. Proc. Soc. Exp. Bid. and Med. 95: 617-620.

Paulson,G.D.; C.A. Baumann and A.L. Pope.1966. Fate of physiological dose of selenate in the Lactating ewe: effect of sulfate. J. Anima Sci.**25**:1054.

Pehrson, B., M. Kutsson and M. Gyllensward. (1989). Glutathione Peroxidase Activity in heifer fed diets Supplemented with Organic and Inorganic Selenium Compounds. Swedish J. Agr. Res. **19**:53-57.

Peterson ., P.J. and D. J.Spedding 1963. The Excretion by Sheep of Selenium Incorporated into Red Clover (Trifolium Pratense L): The Chemical Nature of The Excreted Selenium and its Uptake by Three Plant Species N. Z. J. Agric. Res. 6: 13 – 23.

Preston, R. L. (2000). Typical Composition of Feeds for Cattle and Sheep. Vol. 36 (10), Beef. Intertec Publ. Co., Overland Park, KS, pp. 10–20.

Preston, T. R. and Leng, R. A. (1987). Matching Ruminant Production System with Available Resources in the Tropics and Sub-tropics. Penambul Books, Armidale, NSW, Australia.

Price, W. J., (1979). Spectrochemican Analysis by Atomic Absorptaion. Heyden and Sons Ltd., Spectrum Honse, London, U.K.

Rammell, C.G, K.G. Thompson, G. R. Bentley and M. W. Gibbons. 1989. Selenium, Vitamin E and Polyunsaturated Fatty Acid Concentration. New Zealand Vet. **37**: 4 -6.

Richold.M.; F.Robinson and R.D.H. Stewart, 1977. Metabolic Studies in Rat of Se Incorporated in Vivo into Muscle.Br.J.Nuttr. **38**:19.

Ritat, A.J., Maxwell, W. M. and **Salamon, S. (1984).** Ovulation and LH Secretion in the goat after Intravaginal Progestagen sponge-PMSG Treatment. J. Reprod.Fertil., **72**:559-563.

Rumsey, T.S. (1975). Vitamin requirements for ruminants. Feedstuffs **47**(7):30.

Sahlu, T.; Goetsch, A. L.; Luo, J.; Nsahlai, I. V.; Moore, J. E.; Galyean, M. L.; Owes, F. N.; Ferrell, C. L. and **Johnson, Z. B. (2004).** Nutrient requirements of goats: developed equations, other considerations and future research to improve them. Small Rumin. Res., **53**:191-219.

Salisbury G.W, Van Demark NL, Lodge J.R. (1978). Phsiology of reproduction and artificial insemination in cattle. 2nd ed, Sanfrancisco. WH Freeman.

Sanz Sampelayo, M. R.; Lara, L.; Gil Extremera, F. and Boza, J. (1995). Energy utilization for maintenance and growth in pre-ruminant goat kids and lambs. Small Rumin. Res., **17:** 25-30.

Schalm, O. W. (1965). Veterniary Haematology. Lea and Febiger, Philadelphia, U.S.A.

Schwartz. K and **C. M. Foltz. (1957).** Selenium as integral part of factor 3 against Dietary necrotic liver degeneration. J. Am. Chem. Soc. **79**:3292-3293.

Seboussi. R. Faye, B. Askar, M., Hassan, K., and Ahadrami, G. 2009. Effect of Selenium Supplementation on Blood Status and Milk, Urine and Fecal Excretion in Pregnant and Lactating Camel. Biol. Trace Elem. Res. **128(1):** 45 – 61.

Seymour, W. M. and Polan, C. E. (1986). Dietary energy regulation during Gestation on subsequent or dried brewers grains J. Dairy Sci., 69: 2837 – 2845.

Shelton, J. M. (1990). Goat production in the United States. Proc., Int. Goat Prod. Symp. A and M Univ., Tallahassee, Florida, USA.

Snedecor, G. W. and Cochran, W. G. (1967). Statistical Methods. Iowa State University Press, Ames, Iowa, U.S.A.

Siddons, R. C.; Nolan, J. V.; Beever, D. E. and Macrae, J. C. (1985). Nitrogen digestion and metabolism in sheep consuming diets containing Contrasting forms and levels of nitrogen. Br. J. Nutr., **54**:175–187.

Sorensen, A.M., Jr. **(1979).** Animal reproduction, principle and practices (Sorensen, Anton Marinus, date).

Thomson, E. F.; Gingins, M.; Blum, J. W.; Bickel, H. and Schurch, A. (1979). Energy metabolism of sheep during nutritional limitation and re-alimentation. In: Energy Metabolism. L.E. Mount, (ed.), Butterworths, London, pp. 427 – 430.

Toukourou, Y. and Peters, K. J. (1999). Impact of feed restriction on the growth performance of goat kids. Archiv- Fur- Tierzucht., 42: 3, 281- 293.

Toussaint, G. (1984). Importance des conditions d'amiance dans la propagation des maladies respiratories en production caprine. In, les Maladies de la Chevre, (Les Colloques de I" TNRA, no. 28) Nirot (France), pp. 309 – 324.

Turner, R. J. and Finch, J. M. 1990. Immunological Malfunctions Associated with Low Selenium – Vitamin E Diets in Lambs Journal of comparative Pathology, 126: 2200 – 2204.

Underwood, E.J. (1981). The Mineral Nutrition of Livestock. Commonwealth Agricultural Bureaux, London, England.

Van Eenaeme, C.; Evrard, M.; Hornick, J. L.; Baldwin, P.; Diez, M. and Istasse, L. (1998). Nitrogen balance and myofibrillar protein turnover in double muscled Belgian blue bulls in relation to compensatory growth after different periods of restricted feeding. Can. J. Anim. Sci., **78**: 549–559.

Varly, H. (1967). Determination of Serum Selenium and Potassium In: Practical Clinical Biochemistry, 4th ed., William Heinemann Medical Books Ltd. and Interscience Books Inc., New York.

Venderland, S. C., J. A. Butler and P. D. Wangher, 1992. Intestinal Absorption of Selenite, Selenate, and Selenomethionine in the Rat. J. Nutr. Biochemistry. **3**: 359 – 365.

Vipond, J. E.; Hunter, E. A. and Margaret, E. K. (1982). Effect of cereal and protein supplements to seaweed (*Brassica napus*) on intake and performance of pregnant and lactating ewes kept indoors. Anim. Prod., **34**: 131 – 137.

Waghorn, G.C. 1986. Changes in Rumen Digested of Cows During A restricted Feeding Period When Offered Fresh Red Clover, Lucerne, or Lucerne Hay. N. Z. J. Agric. Res**. 29:** 233 – 241.

Wahed, R. A. and Owen, E. (1986). Comparison of sheep and goat under stall- feeding conditions: roughage intake and selection. Anim. Prod.,42:89-95ce.

Walsh, D.M., S. Kennedy, W.J. Blanchflower, E.A. Goodall and D.G. Kennedy. 1993a. Vitamin E and selenium deficiencies increase indices of lipid peroxidation in muscle tissue of ruminant calves. Int. J. Vitam. Res. **63**:188-194.

Weiss, W.P.; J.S.Hogan; D.A.Todhunter and **K.L.Smith. (1997).** Effect of Vitamin E supplementation in diets with a low concentration of Selenium on mammary gland health of dairy cows. J. Dairy sci. 80:1728.

Wilson, R.T. (1991). Small ruminant production and Small ruminant genetic Resource in tropical Africa food and Agricultural Organization (FAO) Rome, Italy.

Wolffram, S. , E. Eggenberger and E. Scharrer, 1986. Kinetics of D-glucose and Leucine Transport into Sheep and pig intestinal Brush Border Membrane Vesicle. Comp. Biochem. Physiol. 84A, 589 – 593.

Wolffram, S.,B. Grenacher and E. Scharrer. 1988. Sodium – Dependent L-lactate Uptake by bovine intestinal Brush Border Membrane Vesicle. J. Dairy Sci. **71**: 3267 – 3273.

Wright, P.L. and M. C. Bell, 1966. Comprative Metabolism of Selenium and Tellurium in Sheep and Swine .Am.J. Physiol. **211**: 6 – 10.

Yamini, B., and T. P. Mullaney, (**1985)**. Vitamin E and selenium deficiency as a possiple cause of absortion in food animals. Poc. 28th Ann. Meg. Am. Assoc. Vet. Lab. Diagn., Madison, W I. pp131.

Yeh, J., Q. GU, M. A. Beistein, N. E. Forsberg and P. D. Whanger, **(1997)**. Selenium influence on tissue levels of selenoprotein win sheep. J. Nutr. 127:394.6.

APPENDICES

Appendix 1 : Selenium Premix Product Specification

Appendix 2 : Feeding Experimental Goats

SELENIUM PREMIX
Product Specifications

Product Description
SELENIUM PREMIX is a supplemental organic selenium source for livestock.

Label Information

Guaranteed analysis		Ingredients
Selenium	1000 ppm	Yeast brewers dehydrated and
Crude protein	Min. 40%	sodium selenite.
Crude fat	Min. 0.4%	
Crude fiber	Max. 5.0%	

Physical Characteristics

Appearance: **SELENIUM PREMIX** is a beige free-flowing powder.

Bulk Density: 660.7 kgs/m^3

Storage and Shelf Life

SELENIUM PREMIX should be stored in a cool, dry place. Open containers should be resealed. Shelf life is 36 months.

Packaging

SELENIUM PREMIX is available in 25 kg drums or bags.

Use Rate
Use **SELENIUM PREMIX** to supply dietary Se requirements. Added selenium must not exceed 0.3 ppm in the total diet for poultry, beef and dairy cattle and 0.1 ppm in the total diet for horses.

ALLTECH INC.
3031 Catnip Hill Pike Nicholasville, KY 40356 (606) 885-9613 FAX: (606) 885-6736

Appendix 2 : Feeding Experimental Goats