

**Sudan University of Science and Technology**  
**College of Graduate Studies**

**Some infertility problems and their economical  
impacts in dairy farm in Eastern Nile Locality**

بعض مشاكل تدني الخصوبه واثارها الاقتصادية بمزارع الالبان في محلية شرق  
النيل

A Thesis submitted in Fulfillment of the Requirements for the  
Degree of Master of Veterinary Medicine

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# **Dedication**

To my parents and family

To my wife

To those from whom I acquired knowledge

And

To every one who supported me through my life

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## **Abstract**

This study was conducted in Eastern Nile Locality Khartoum State, Sudan during the period (2008-2010). The primary objective of the study is to identify infertile cows and the, causes of infertility among the study population besides assess the economic impacts of infertility.

The study was conducted in two phases; in phase one across sectional survey of cross bred dairy cattle was conducted in Eastern Nile Locality (ENL) were a total of 491 cows were examined for infertility by: case history, rectal palpation and serological test beside progesterone hormone levels determination.

In phase two, slaughter house surveys were conducted at Alshaheed-Nasraldeen and Alkadaro slaughter houses, in these survey 164 genital tracts were examined for any abnormality associated with infertility (local cows and heifers were removed).

The study revealed that about 17% of the examined cows were infertile. Eighty seven and half percent (78.5%) of them were due to direct causes. Indirect causes were 12.5% of the total infertile. Follicular cysts were the major cause followed by inactive ovary and Brucellosis had significant effect on infertility.

Economic losses were estimated as due to calves loss which accounted to SDG 65,875per year. Losses due to culling were SDG 36.000 and that due to milk loss was SDG 856,800/year. The cost of veterinary intervention was estimated as cost of diagnosis which accounted to SDG 1455 and the cost of treatment as SDG 3062.

The slaughter house survey revealed that 58 out of 128 examined cows were infertile. The direct infertility causes accounted to 45.31% while the indirect causes accounted for35.1%. Pregnant cows reported as19.59 % the major causes of direct infertility were follicular cyst (11.65%), and metritis (8.84%), persistent Corpus luteum (7.77%) and inactive ovary (7.77%) and pyometra (4.85%). Also the study revealed that 19.53% of the examined cows were pregnant.

## ملخص الاطروحة

أجريت هذه الدراسة بمحلية شرق النيل ولاية الخرطوم -السودان في الفترة (2008-2010) كان الهدف الرئيس للدراسة هو التعرف علي الابقار التي تعاني من تدني الخصوبة واسباب ذلك في مجموعة الدراسة ومن ثم تحديد الأثر الإقتصادي المترتب عن تدني الخصوبة.

أجريت الدراسة علي مرحلتين: في المرحلة الاولى تم إجراء مسح ميداني بمزارع الالبان حيث تم اختبار عدد 491 بقره هجين (فريزيان x ابقار محليه) وذلك عن طريق اخذ تاريخ تدني خصوبة الابقار المختاره ومن ثم متابعتها مرتين اسبوعيا وفحصها عن طريق الجس عبر المستقيم ومن ثم اخذ عينات دم للفحص المصلي وقياس مستوي هرمون البروجستيرون.

المرحلة الثانيه تم اجرائها بمسلخي الشهيد نصرالدين بالمايقوما ومسلخ الكدرو حيث تم فحص عدد 164 عينة رحم جمعت من الابقار تم فحصها عياناً .

اظهرت الدرسة ان 17% من الأبقار التي تم فحصها تعاني من تدني الخصوبة وان اكثر اسباب تدني الخصوبة هي الاسباب المباشره وبنسبها 87.05% مقارنة بالاسباب غير المباشره التي بلغت 12.98%. كما اظهرت الدرسة ان تكيس المبايض هو اكثر شيوعاً يليه كسل المبايض وأن البروسيلا لها اثر واضح في تدني الخصوبة.

من الناحيه الاقصاديه نجد ان فقدان العجول قدر بحوالي 65.875 جنيه سوداني في العام بينما بلغت خساره الناتجه عن استبعاد الابقار 36.000 جنيه سوداني في العام كما اظهرت الدرسة ان الفاقد في انتاج اللبن بلغ 856,800 ج في العام و تمثلت تكلفه التدخل البيطري في التشخيص و العلاج حيث بلغت تكلفه التشخيص والعلاج 1455 ج س و 3062 ج س علي التوالي.

اظهرت نتائج الدرسة في المسلخين ان 58 بقره من جمله 128 بقره تعاني من تدني الخصوبه وان نسبة 45.31% ترجع للاسباب المباشره حيث بينما بلغت نسبة الاسباب الغير مباشره 35.2%. كانت اكثر الاسباب المباشره شيوعا هي تكيس المبايض بنسبة حدوث 11.65% يليه التهاب الرحم بنسبة حدوث 8.84% و كسل المبايض ثبات الجسم الاصفر بنسبة 7.77% لكل. وبلغت نسبة تقيح الرحم 4,85%. كما اظهرت الدرسة وجود 25 بقره حامل بنسبة حدوث 19.53%.

## ABBREVIATIONS

AB:	Abortion
AI:	Artificial insemination
BCS:	Body condition score
BR:	Brucella
CFT:	Complement fixation test
CIDR:	Controlled internal drug release
CL:	corpus luteum
Cls:	corpora lutea
COD:	Cystic ovarian disease
COF:	Cystic ovarian follicle
DIP:	Degraded intake protein
DM:	dystocia
ED:	Edbabikir
ELIZA:	Enzyme-Linked immunosorbent assay
EM:	Embryonic mortality
ENL:	Eastern Nile locality
FC:	Follicular cyst
FSH:	Follicular stimulating hormone
GnRH:	Ganado trophin releasing hormone
HCG:	Human chorionic gonadotropin
HK:	Hilt Kuku
HY:	Hajyousuf
IO:	Inactive ovary
JE:	Jreef east
K:	Kerriab

LH:	Lutinizing hormone
ME:	metritis endometritis
MIC:	Minimum inhibitory concentration
MRT:	Milk ring test
OBA:	Ovario barsual adhesion
PGF2 $\alpha$ :	Prostaglandin F2 $\alpha$
PRID:	Progesterone Intravaginal device
Py:	Pyometra
RBPT:	Rose Bengal plate test
RBS:	Repeat breeder syndrome
RFMS:	Retained fetal membrane
RP:	Rectal palpation
RPT:	Rose Bengal test
RT:	Rectal temperature
SB:	Still birth
SD:	Standard deviation
SH:	Silent heat

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# CHAPTER ONE

## INTRODUCTION

### **Background**

Worldwide, the poor reproductive performance of dairy herd became a major concern especially in Holstein Friesian herd. Decline in conception rates and increase in calving interval were confirmed by many authors (Royal *et al.*, 2000; Lucy, 2001).

Infertility became a major problem in dairy cattle production to the point that it is becoming increasingly apparent, that high yielding cows were unable to conceive under optimum condition. This negative association between production and fertility has been observed in several dairy breeds (Pryce and Veerkamp, 2001). Ten% to 30% of lactations may be affected by infertility and reproductive disorders (Erb and Martin, 1980), and 3-6% of the herd culled annually in developed countries for these reasons. The extent of the problem was likely similar in the tropics, although extensive data were not available (FAO, 1989).

Infertility problems in dairy herds affect profitability through additional expenditure and reduced income. Reduced income as a result of lengthened calving intervals, lower / inputs or outputs / fixed costs ratios in the production process. Direct effects were reduction in milk yield and calf crop per unit of time. A large part of economic consequences of poor reproduction is attributed to culling (Sol *et al.*, 1984).

Sudan is known to possess a considerable number of cattle population accounted to be 29,618 thousand head (Information Centre- MARFR, 2012). However, the average Milk per-capita consumption of Africa per year of about 37.2 kg and in Sudan 180.68kg (FAO, 2000). The country, therefore, resorted to importation of powder milk to meet the domestic demand on one hand, and on the other hand, it exerts enormous efforts to

improve the genetic potentials of local cattle through cross breeding with exotic breeds.

Nowadays the population of crossbred dairy cows in Khartoum State reached 52.8% of the total cattle population estimated at 2, 2843 head (Ministry of Agriculture and Animal Resources, Khartoum State, 2006). About two third of the crossbred cows in Khartoum State were found in Eastern Nile Locality (Ministry of Agriculture and Animal Resources, 2006).

With the progress of the cross breeding program, the foreign blood reached 62.5% in some farms and 81% in others (Ministry of Agriculture and Animal Resources, 2007). This was reflected in improved milk yield reaching 28.4 lbs / day compared to 16.6 lbs/ day in case of the local breeds with an average yield during productive life of 797.32lbs compared to 469.43 lbs for the local breeds (Ministry of Agriculture and Animal Resources, 2007).

### **Research problem**

Although milk yield of the crossbred cows in Khartoum state has improved to some extent than that of indigenous breeds this improvement however, was reciprocated by continuous decline in fertility of crossbred cows in the small holders' farms in the State. This constituted one of the main problems facing the dairy production sector today. The low fertility was manifested by increased number of services per conception which leads to increased open days and extending calving interval to 413 days (Ministry of Agriculture and Animal Resources- Khartoum State, 2006). The economic repercussions of infertility were reduced milk yield, loss of calf crop, increased labor, increased veterinary intervention and treatment costs and increased culling of cows due to failure to become pregnant and the resultant loss of income incurred to dairy farmers (Bellows *et al.*, 1994). Due to all the above mentioned reasons large number of farmers abandoned dairy business. This again bears negative consequences on dairy production and national economy. It is imperative therefore to address this

critical problem of infertility in the crossbred dairy cows and its negative impact on farm economy in order to solve this problem.

### **Objectives:**

The primary objective of this study is to focus on the problem of infertility as a major factor affecting the farm economy in Eastern Nile Locality of Khartoum.

Specific objective:

- To identify infertile cows and to estimate the prevalence rate among the study population.
- To determine the main reasons of infertility among crossbred dairy cows in the study area.
- To estimate the economic loss associated with infertility.
- To determine reasons for culling and slaughtering crossbred cows.

### **Hypothesis**

1. The herd prevalence of infertility is more than 50%, while the individual animal prevalence is about 25%.
2. The main reasons of infertility are brucellosis, cystic ovary and metritis.
3. The economic loss due to infertility accounted to be about 10% of the farm income.
4. The main infertility reasons of culling are >ovarian cyst, metritis, pyometra, inactive ovary.

### **Thesis layout**

The research is organized in five Chapters:

Chapter One: Introduction

Chapter two: literature review

Chapter three: Materials and methods

Chapter four: Results

Chapter five: Discussion



Conclusions and recommendations.

References.

# CHAPTER TWO

## LITERATURE REVIEW

### 2.1. Definition of Infertility

Healthy cattle give expression to normal fertility by producing one viable calf per year. The term fertility as applied to the cow denotes the desire and ability to mate, the capacity to conceive and to nourish the embryo and finally the power to expel normal calf and fetal membrane (Arthur, 1982).

Sterility is a permanent factor preventing production of new generations, and infertility or temporary sterility is the inability to produce viable young calves within stipulated time characteristic for each species (Hafez, 2000).

Cattle are deemed infertile when they are neither normally fertile nor completely sterile. Interest in bovine infertility increased with the introduction of artificial insemination in the 1950s and as the factors involved became known to farmers, herdsmen, physiologists and other workers (Roberts, 1956).

### 2.2. Causes of Infertility:

According to Grohn and Rajala-schultz (2000) infertility causing diseases are the factors that influence fertility, while Lucy (2001) considered the age of the cow, production and nutrition. Whereas Veerkamp *et al.*, (2000) related them to the reproduction physiology of dairy cow.

Arthur (1982) conceived that the causes of infertility are many and can be complex; they are related to Graafian follicle development and maturation, oestrus onset, successful coitus, ovulation, fertilization, implantation, and the development and delivery of the foetus and its membranes. According to Osmanu (1979), anything interfering with these routines, such as diseases, poor nutrition, inadequate herd management, hereditary and congenital factors, hormonal

disturbances or environmental changes, makes the animal infertile, even if they were temporarily.

### **2.2.1. Congenital morphological causes:**

Congenital causes of infertility are often inherited. They include developmental abnormalities of the ovaries, oviducts, uterus, cervix, vagina and vulva. Some are lethal. Common conditions with morphological significance include ovarian aplasia and hypoplasia, anomalies of the tubular genitalia (Vale *et al.*, 1984). Lagerlof, (1963) added the hermaphroditism, freemartinism, arrested development of the Mullerian ducts (White heifer disease) and double cervix.

### **2.2.2 Functional causes of infertility and repeat breeding:**

The causes of functional infertility included cystic and inactive ovaries with anoestrus, early embryonic mortality with repeat breeding and prolonged gestation were responsible for 59.4% of the cases of infertility (Kodagali, 1974). Anoestrus often reflects a hormonal disturbance and accounted for 47.8% of the cases. According to Singh *et al.*, (1981) repeat breeding, where cows require three or more services to conceive, accounted for 11.5% of cases. The author also found functional infertility to be more common than infertility due to infectious diseases (76% vs 24%). Anoestrus is exacerbated by inadequate nutrition, high ambient temperature, and high parasitic burdens and may be due to elevated level of prolactin (Vandeplassche, 1982).

Incidence of repeat breeding was estimated to be 21.9% in 411 Holstein Friesian crosses (Kaikini *et al.*, 1983); and 16.6 to 58.8% in Fulani cattle (Nuru and Dennis, 1976).

Among the most common causes of repeat breeding are factors including sub-fertile bulls, endocrine problems, malnutrition, reproductive tract infections and poor management (Vandeplassche, 1982). Ovarian bursa adhesions, salpingitis, cystic ovaries and endometritis were also common causes of repeat breeding. Sagartz and Hardenbrook (1991) reported that 77% of infertile cows had

endometritis. Histological evidence was observed in 50% of the genital tracts obtained from an abattoir hence it is likely that sub-clinical endometritis is a major contributor to the repeat breeder syndrome of bovine infertility (Noakes *et al*, 2001). Severe endometritis may be associated with a persistent corpus luteum due to toxic damage of the endometrium and can also occur with conditions simulating pregnancy such as pyometra, fetal mummification and maceration (Boyd, 1977). Lamming (1977) reported that persistent corpus luteum is rare in cows with normal uteri.

### **2.2.2.1 Cystic ovaries and retained corpora lutea:**

#### **2.2.2.1.1-Follicular Cysts**

Cystic Ovarian Disease (COD) is a common and economically significant condition of dairy cattle (Johnson and Coates, 2004).

There are several definitions used to describe follicular cysts, and the traditionally accepted definition is that they are defined as anovulatory fluid-filled structures 25 mm in diameter that persist on the ovaries for more than 10 days (Archibald and Thatcher, 1992). Hatler *et al.*, (2003) pointed out that “follicles typically ovulate at 17 mm in diameter, so follicles that persist at that diameter or greater may be considered to be ‘cystic’. Vanholder *et al.*, (2006) suggested that cystic ovarian follicles (COF) should be defined as “follicles with a diameter of at least 2 cm that are present on one or both ovaries in the absence of any luteal tissue and that clearly interfere with normal ovarian cyclicity.

Cystic ovarian follicles develop when one or more follicles fail to ovulate and subsequently do not regress but maintain growth and steroidogenesis. They were defined as follicle-like structures, present on one or both ovaries, with a diameter of at least 2.5 cm for a minimum of ten days in the absence of luteal tissue (Day, 1991).

It has also been determined that follicular cysts are an ovulatory structures so, as long as they persist, cows will remain infertile (Youngquist and Threlfall, 2007).

Cystic ovarian disease (COD) is the “most common endocrine pathology to be found in dairy cows,” and according to Gordon, (1996) the incidence of cystic ovarian disease is believed to vary from 1% to 30% depending on herd and breed conditions. Holsteins are the most susceptible to develop a cystic condition compared to other breeds, and the most likely time of diagnosis is 30-60 day after parturition in high-yielding dairy cows. Ball and Peters (2004) mentioned that follicular cysts, when compared to other ovarian cystic conditions are characterized by thin walls and produce very small amounts of progesterone. They further mentioned that a persistent condition can occasionally lead to increased testosterone levels, causing some cows to exhibit masculine aggressive and sexual behavior. However, most cystic cows will remain in anestrus as long as the condition persists. Monniaux, *et al.*, (2008) reported that the fluid in follicular cysts contains many components, including hormones like estradiol, progesterone, and insulin. Anti-Mullerian hormone, a hormone strongly linked with polycystic ovarian syndrome in humans, is also present in follicular cysts in cattle, but at such a low concentration suggesting that there is no correlation between the hormone and incidence of COD in cattle. There are also a variety of proteins present in the fluid, Maniwa *et al.*, (2005).

The most widely accepted hypothesis explaining the formation of a cyst is that LH release from the hypothalamus-pituitary is altered: the pre-ovulatory LH-surge is either absent, insufficient in magnitude or occurs at the wrong time during dominant follicle maturation, which leads to cyst formation (Day, 1991; Lopez-Diaz and Bosu 1992; Hamilton, 1995; Yoshioka *et al.*, 1996; ) This aberrant LH release does not seem to be caused by a lower GnRH content of the hypothalamus, nor by reduced GnRH receptor numbers or LH content in the pituitary (Brown,1986; Cook,1991).

An altered feedback mechanism of estrogens on the hypothalamus-pituitary can result in an aberrant GnRH/LH release and cyst formation. A

GnRH/LH surge occurring prematurely during follicle growth, i.e. when no follicle capable of ovulation is present, can render the hypothalamus unresponsive to the feedback effect of oestradiol which results in the formation of ovarian cysts (Gümen *et al.*, 2002). To restore the feedback mechanism, the hypothalamus needs to be exposed to progesterone (Ozturk *et al.*, 1998; Gümen and Wiltbank 2005).

In cystic animals, despite the failure of ovulation by the pre-ovulatory follicle, a surge of follicle-stimulating hormone (FSH) would occur, and under conditions of low progesterone concentrations and high LH concentrations, dominant follicles would be caused to grow to a larger size. These oversized follicles are termed follicular cysts that produce high concentrations of estradiol and inhibin, which cause a delay in follicular turnover and are responsible for the persistent cystic condition (Kaneko *et al.*, 2002; Bartolome *et al.*, 2005).

Endocrine profiles of COD cows have shown that, serum LH is high or normal, LH pulse frequency and amplitude are higher than normal, serum FSH is low or normal, and serum inhibin is high (Archbald and Thatcher, 1992; Hamilton *et al.*, 1995). The high baseline LH levels support persistence and growth of the follicle to an abnormally large size (Calder *et al.*, 2001).

### ***Causes***

Despite a broad range of research investigating the biological mechanisms prior to cyst formation, the exact pathogenesis of this disease remains unknown (Hamilton *et al.*, 1995; Silvia *et al.*, 2002; Evans, 2003; Peter, 2004; Vanholder *et al.*, 2006). It is generally accepted that, disruption of the hypothalamo-pituitary-ovarian axis by endogenous and/or exogenous factors, causes cyst formation” (Vanholder *et al.*, 2006).

While there are many factors that have been linked to the predisposition of cattle to develop follicular cysts, the exact cause has not been defined. The factors that do predispose cows to develop follicular cysts have been suggested as genetics, hormonal imbalances, or exogenous factors, among others. For (Hooijer

*et al.*, 2001) the most widely accepted hypothesis is that COD results from a neuroendocrine imbalance involving the hypothalamic-hypophyseal-gonadil-axis. While this general hypothesis may be accepted, it is vital to determine the specific disruption and where along the pathway it occurs in order to fully understand how follicular cysts develop and how they can be treated and prevented.

There appears to be a genetic predisposition to cystic ovaries (Casida and Chapman, 1951.; Erb *et al.*, 1959 and Johannson, 1960), the estimates of the heritability of cystic ovaries range from 0.05 to 0.43. Menge *et al.*, (1962) reported a genetic correlation of 0.22 between milk production and cystic ovaries, High milk production and COD have a positive correlation but the cause and effect have not been determined. Possibly high producing cows have a higher incidence of COD, but delayed conception will increase milk production (Johnson *et al.*, 1966). In fact, the study performed by Hooijer *et al.*, (2001) led to the conclusion that selection for milk yield leads to an increase in COD incidence by 1.5% per 500 kg increase in milk yield. They hypothesized that a possible explanation for the direct correlation between high milk yield and increased COD incidence is that cows in early lactation, which are trying to meet the increased requirements for milk production are more susceptible to environmental changes with hormonal implications as a result.

Gümen *et al.*, (2002) hypothesized that follicular cysts would develop if cows experienced an estradiol-induced GnRH/LH surge in the absence of an ovulatory follicle. They further suggested that estradiol would fail to induce a subsequent GnRH/LH surge in these cows until they were treated with progesterone. Other studies investigated on the relationship between progesterone and ovarian follicular cysts. Hatler *et al.* (2003) looked more closely at this relationship, like many other sources, he pointed out that the basic reason cysts form is an endocrine imbalance, and that the vast majority of data suggest that

cysts are formed because of a failure of the preovulatory LH surge to occur at the appropriate time in follicular maturation.

Hatler *et al.*, (2003) concluded that the intermediate levels of progesterone commonly associated with cysts may predispose subsequent follicles to form cysts and contribute to the phenomenon of cyst turnover. López-Gaitus *et al.*, (2002) also recognized the effect of high milk production on cyst incidence, and determined that older cows were more likely to develop a chronic cyst condition than those in earlier lactations.

A thorough explanation for the biological cause of follicular ovarian cysts was provided by Bartolome *et al.*, (2005) where they mentioned that the initial root of the problem is a dysfunction of the positive feedback of estradiol on release of GnRH. Because of this, function of the hypothalamic-pituitary-ovarian axis is altered, even though the pituitary gland is still able to release LH. They further explained that, because of the hormonal imbalance, the dominant follicle does not ovulate before the transient increase in FSH occurs, stimulating a new wave of follicular development, which occurs under conditions of low progesterone concentrations and high LH concentration. This causes excessive growth of dominant follicles to become follicular cysts, which produce large amounts of estradiol and inhibin, therefore causing a delay in follicular turnover.

Braw-Tal *et al.*, (2009) examined the relationships between levels of certain hormones and incidence of COD. The two hormones that they focused on were insulin and insulin like growth factor-I (IGF-I), which have previously been suggested as indicators between nutritional status and ovarian function in cattle. Both hormones have been shown to stimulate estradiol synthesis in granulosa cells and androgen synthesis in theca cells. Based on the previous studies, Braw-Tal *et al.*, (2009) hypothesized that any failure in function of the insulin/IGF-I system will lead to follicular regression and/or cyst formation. Though they did find a



correlation between insulin/IGF-I and cystic formation, there was very little trace of the hormones found in cystic fluid

### **Prevention and Treatments**

The most economical time to treat COD was as soon as the condition was diagnosed, even without waiting for the voluntary period during which spontaneous recovery may occur the greatest economic benefit of treatment is a reduction in days-open (White and Erab, 1980). Ovulation of the cyst does not occur, but other follicles present at the time of treatment may ovulate (Cantly, *et al.*, 1975, Dinsmore, *et al.*, 1987 Wiltbank, 2002). After the GnRH treatment and subsequent luteinization, the cyst becomes responsive to prostaglandin-F<sub>2</sub> $\alpha$  (PGF<sub>2</sub> $\alpha$ ) because the steroidogenic synthesis pathway has switched from estradiol to progesterone. The newly elevated levels of progesterone are responsible for a restoration of responsiveness to the positive feedback effect of estradiol, resulting in the resumption of normal cyclic ovarian activity after the release of endogenous PGF<sub>2</sub> $\alpha$  and cystic regression.

Follicular cysts are most commonly treated by administration of synthetic GnRH analogs approved for use in lactating dairy cows (Bierschwal *et al.*, 1975; Seguin *et al.*, 1976; Whitmore *et al.*, 1979).

Return to normal cyclic ovarian activity occurs in 72% to 85% of the cows treated with GnRH; the interval from treatment to the first estrus is 19 to 23 days, and pregnancy rates at first estrus range from 46% to 58%. In comparative studies, buserelin (a more potent GnRH analogue) or human chorionic gonadotropin (HCG) produced similar effects to those observed after GnRH treatment (Garverick, 1976; Dinsmore, *et al.*, 1987). They further end with that the underlying reasons for the continued state of anestrus in about 20% of the cows treated with GnRH are not clear. as there is no difference in stimulated LH release between responsive and unresponsive cows. However, the cause could be linked to issues with endogenous progesterone as the anovulatory cows show no increase in

progesterone after the release of LH. Another method that has been used successfully for the treatment of COD is the Ovsynch protocol, originally designed for fixed-time artificial insemination (AI). This protocol has been shown to provide similar pregnancy rates (~27%) for both normal cycling cows and cows with cysts in a large Florida dairy herd.

Ovsynch, a protocol for synchronizing ovulation in lactating dairy cows, uses injections of both GnRH and PGF<sub>2α</sub> (Dinsmore *et al.*, 1990; Pursley *et al.*, 1997), and an effective treatment for ovarian cysts.

Stevenson and Tiffany (2004) are in agreement with the usage of the Ovsynch protocol as a treatment for COD, it's effective at curing the cystic condition and getting the previously cystic animal pregnant after AI.

The third option of COD treatment is the administration of exogenous progesterone (Calder *et al.*, 1999). Progestogens as single or multiple injections, in the form of intra vaginal devices (CIDR or PRID) or as ear implants have been used to treat COD (Todoroki *et al.*, 2001).

Treatment with intravaginal implants for 9 to 12 days decreases LH secretion and results in cyst regression and emergence of a new follicular wave, 5 days after implant insertion. Progesterone works against follicular cysts by restoring the responsiveness of the hypothalamus to the positive feedback of estradiol, resulting in normal estrus and ovulation within 7 days after the implant is removed. Progesterone was such an effective treatment for COD that the resulting estrus rates range was 82-100% and conception rates at first estrus had a range of 18-28% (Brito and Palmer, 2004). Manual rupture of cysts via rectal palpation is not recommended because of the reduced efficacy compared with GnRH (Ijaz *et al.*, 1987) and because adverse side effects including adhesions around the ovary and adnexa may impair fertility (Archibald and Thatcher, 1992).

The treatments that Brito and Palmer (2004) have found to be effective are summarized as:

- Gonadorelin (GnRH) 100g/IM.
- HCG 10,000 IU/IM.
- Dinoprost (PGF2 $\alpha$ ) 25mg/IM.
- Cloprostenol (PGF2 $\alpha$ ) 500mg/IM.
- Progesterone 1, 9 g/Intravaginal implant.

### **Treatment Protocols**

- (1) GnRH (or hCG) + PGF2 $\alpha$  (day 0); PGF2 $\alpha$  (day 9 if no estrus).
- (2) Ovsynch: GnRH (day 0); PGF2 $\alpha$  (day 7); GnRH (day 9); fixed time AI, 16 h after last GnRH treatment.
- (3) Progesterone implant for 12 days (not for dairy cows).

Manual rupture has been used often without problems, but the potential danger of traumatizing the ovary and causing hemorrhage with the subsequent local adhesions should not be overlooked. This method should be weighed against the cost of hormone therapy (Dobson, 1977; Kahn, 2010).

#### **2.2.2.1.2 -Luteinized Cysts**

Luteal cysts are thick-walled, fluid-filled structures 25 mm in diameter that secrete normal to above normal amounts of progesterone. Most luteal cysts probably form through luteinization of a follicular cyst (Garverick, 1997) and can cause infertility if they persist and maintain systemic progesterone at concentrations that inhibit the LH surge and ovulation. The thick wall of luteal cysts is composed of luteal tissue and, in contrast to cystic follicles; the fluid-filled cavities of cystic CL often contain numerous intertwining trabecula that can be easily resolved using ultrasonography. Farin *et al.* (1992) call these cysts luteinized cystic follicles, describing them as cysts with thicker walls that produce high levels of progesterone. In appearance, they are smooth and rounded with a spherical cavity that is lined by a layer of fibrous tissue surrounded by the luteinized cells (Schlafer, 2007). Luteal cysts are considered anovulatory cysts and are associated with infertility and Mucometra in cattle (Foley, 1996). When compared to

follicular cysts, luteinized cysts are more likely to persist over long periods of time and can lead to nymphomania in some animals (Ball and Peters, 2004).

### **Causes**

Schlafer (2007) believed that luteal cysts develop when ovulation fails to occur and the theca undergoes luteinization. They are also often considered by (Vanholder *et al.*, 2005) to be the later form of ovarian follicular cysts. And therefore the causes pertaining to follicular cysts can also be considered the original causes of luteal cysts. According to Peter *et al.*, (2009) the luteal cyst occurs when the cells of the follicular cyst (granulosa and theca) become luteinized and start producing progesterone. Foley (1996) mentioned that luteal cyst incidence increases with age and most often affects cows with high milk production.

### **Prevention and Treatments:**

Gonadotropin releasing hormone (GnRH) analogue or human chorionic gonadotropin (hCG) have been widely used to treat follicular and luteal cyst. They are equally effective treatment, (Jatuporn *et al.*, 2010).

Treatment of persist corpus luteum is usually used with PGF2 $\alpha$  administration, due to its most effective and shortest period to induce estrus respond (Thomas, 2007).

When cows with luteal cysts are treated with GnRH or hCG, regression can be induced 7 to 9 days later with exogenous PGF2  $\alpha$ , shortening the length of time between treatment and the return to normal ovarian cyclicity (Kessler, 1978). On other hand prostaglandin F2 $\alpha$  can be used for treatment of luteal cyst (Kesler and Garverik, 1982; Tebble *et al.*, 2001 Peter, 2004; Jeffs *et al.*, 2011). The study of Antoine and Pattabiraman (1993) revealed that simultaneous treatment with GnRH and PGF2 $\alpha$  allowed an early return to estrus in cows with luteal cysts, 50% of the cows were in estrus before a second PGF2 $\alpha$  treatment 14 days later.

Luteolytic doses of PGF2 $\alpha$  are the ideal treatment for luteal cystic ovary disease, as long as the animal was diagnosed correctly, with estrus showing within

3-5 day Kahn (2010). However, he argued that the treatment can be variable as there is difficulty in accurately estimating the amount of luteal tissue present. There will be no response if a luteal cyst is diagnosed when the structure is actually a normally developing CL or cystic CL, as PGF<sub>2</sub> $\alpha$  is only effective until the 6th day following estrus. Luteal cysts also respond currently to human chorionic gonadotropin (hCG) and pituitary content with high LH extracts are used for treatment Kahn (2010). Synthetic gonadotropin-releasing hormone (GnRH) has been used for the treatment of ovarian cyst successfully (Bierschwal *et al.*, 1975; Amiridis, 2009).

Because estrus detection methods are poor on many farms, the Ovsynch protocol for fixed time artificial insemination, which will work on both follicular and luteal cysts, is considered to be the treatment of choice as it results in timely breeding after treatment for cows with either condition. Contrarily to follicular cysts, however, luteal cysts should not be ruptured manually, as trauma is much more likely to occur (Pursley *et al.*, 1995).

#### **2.2.2.1.3-Cystic Corpora Lutea**

Chuang *et al.*, (2010) defined the cystic corpus luteum (CL) in a cow as luteal tissue initiating from a corpus hemorrhagicum and containing fluid in a central cavity greater than 7 mm in diameter. However, to avoid confusion between the terms cystic corpus luteum, which is considered as a normally functional structure, and luteal cyst, which considered as a pathological condition, the contemporary term “corpus luteum with a cavity” has been suggested to replace the classical term cystic corpus luteum (Peter *et al.*, 2009). Cystic CL occurs spontaneously, when follicles become luteinized without ovulation (Pineda and Dooley, 2003). The incidence of corpus luteum with a cavity, reported to be between 25.2% and 78.8% during diestrus, eventually it decreases as the oestrous cycle advances (Kito *et al.*, 1986; Okuda *et al.*, 1988; Kastelic *et al.*, 1990; Foley, 1996).

Because cystic corpora lutea are found in cows that are normally cycling or pregnant, they are considered to be a normal stage or variation of CL development (Kahn, 2010). Cystic CL has a soft, mushy core area, due to presence of fluid from a degenerating blood clot, compared with the homogeneous, liver-like consistency of the base of a typical CL. The cystic CL as well as the typical CL may or may not have an ovulation crown or papilla at its apex. Absence of this ovulation crown or papilla should not be considered diagnostic of the cystic condition because 10-20% of functional, normal CL fails to develop this feature. Although, diagnosis is not essential as cystic CL are not pathological and therefore do not require treatment, the ideal time for detection of the structure is 5-7 d after estrus. At this point in time, the ovarian structure is near the end of the corpus hemorrhagicum stage of development (Kahn, 2010).

### **Diagnosis of Ovarian Cysts**

#### **Differentiation of Cystic Conditions**

Dawson (1975) mentioned that as follicular cysts and luteal cysts are similar in size and function; it may be difficult to differentiate between them. Diagnosis of cysts has developed over the last 50 years; traditionally rectal palpation of follicles was the only way to monitor follicular structures. Developments in technology have led to methods with increasing, yet varying degrees of accuracy. Transrectal ultrasonography can be utilized to monitor follicle growth and development on a frequent basis (Pierson *et al.*, 1988; Fricke, 2002). Withdrawal and analysis of blood or milk samples for the hormones oestradiol and progesterone can also be utilized for diagnosis (Douthwaite and Dobson, 2000), although there is disagreement by many authors over what the target values should be in either diagnosis (Nakao, *et al.*, 1983; Farin, 1990; Ribadu *et al.*, 1994; Douthwaite and Dobson, 2000; Mueller, 2007). The most convenient method of diagnosis is transrectal palpation as this is quick and requires no equipment, but the

best diagnosis is likely to come from a combination of both transrectal ultrasonography (Farin *et al.*, 1990) and progesterone analysis (Mueller, 2007).

Ultrasonography can be used to determinate the accuracy of differences in appearance of ovarian cyst between the follicular cyst which have uniformly an echogenic structure > 2.5 cm in diameter with inner hypo echogenic thin wall < 3mm and luteal cyst which with spherical anechogenic structure > 2.5 cm in diameter and inner hypoechogenic thick wall of luteinized tissue > 3 mm (Farin *et al.*, 1992). They argue that though it is important to understand the differences between these two types of cysts, treatment is similar for the two conditions so differentiation is not always necessary.

There is a significant difference between luteal cysts and cystic corpora lutea (cystic CLs) (Kastelic *et al.*, 1990). These cystic structures can be differentiated through rectal palpation to determine if the structure is structurally characteristic of a corpus luteum. A cystic CL can be diagnosed if there is detection of a line of demarcation and distortion in the shape of the ovary. However, differentiation can only be successfully confirmed when rectal palpation and ultrasonography are combined. When differentiating between ovarian follicular cysts and normal pre-ovulatory follicles one should look at the number and size of the structures but should especially base their judgment on the basis of uterine tonicity. Upon rectal palpation, cows with ovarian cysts will be found to have multiple follicles that are typically larger-than-normal Graafian follicles with a flaccid uterus in the absence of a corpus luteum. Bartolome *et al.*, (2005) add that in contrarily, cows in proestrus with normal follicles will have an erect, turgid uterus. This uterine tonicity is in response to luteal oxytocin secreted by the ovaries of non-cystic cows. This method is the most effective at differentiating between follicular cysts and normal follicles; however, an ultrasound should also be used to determine the number and size of ovarian follicles in order to confirm the diagnosis.

It may be difficult to determine whether a cow has ovarian cysts or it is in postpartum nutritional anestrus. Though a cystic condition may often be the first guess for the cause of prolonged anestrus, several physiologic (eg. pregnancy), pathophysiologic (eg. postpartum and nutritional anestrus and ovarian cysts), and pathologic (eg. pyometra, hydrometra, ovarian hypoplasia, and granulose cell tumor) conditions result in a lack of cyclic activity, an ovulation, or anestrus (Bartolome *et al.*, 2005). Shallow postpartum nutritional anestrus is an anovulatory condition that can occur during the early postpartum period, and it's characterized by regular follicular waves and lower than normal secretions of luteinizing hormone (LH) and insulin like growth factor 1 (IGF-1). These conditions cause the animal to be unable to support complete follicular development and lead to insufficient estradiol concentrations for ovulation. While this condition is associated with animals in negative energy balance, cows with cystic ovarian degeneration are in positive energy balance. Cows may also have deep postpartum nutritional anestrus, during which the ovaries are small with limited follicular development and follicle stimulating hormone (FSH) secretions are low. Because of this, a simple rectal palpation and/or ultrasound examination can identify whether or not the anovulatory condition is caused by deep postpartum nutritional anestrus. Ovarian cysts are differentiated from shallow anestrus on the basis of the number and size of the follicles, occurrence of follicular waves, body condition score (BCS), and stage of lactation (Johnson, 2004).

#### **2.2.2.2. -Other Ovarian Abnormalities**

##### **2.2.2.2.1 Ovaro-bursal Adhesions**

The Ovaro-bursal adhesions are structures that occur as fibrous bands between the surface of the ovary and the ovarian bursa. Their severity depends on the presence of a few very small strands of fibrous tissue. Though there is no definitive cause of adhesions, they are most likely the result of excessive follicular hemorrhaging during ovulation, trauma to the ovary or bursa caused by rectal



examination, an infection from the uterus, or damage during calving (Peters *et al.*, 2009). Ball and Peters (2004) mentioned that, Ovaro-bursal adhesions generally do not cause reproductive problems in affected cows except, in severe cases, where the adhesion is so large that the fallopian tubes are blocked and fertilization of the ovum is prevented. Peter *et al.*, (2009) added that in extreme cases, adhesions may extend to the opening of the ovarian bursa, resulting in a very narrow opening that may affect fertility.

Referred to this condition as perisalpingitis and considered it as rare conditions. The diagnosis of ovaro-bursal adhesions is typically difficult in live animals, but adhesions may be suspected in cows that persistently return to estrus after breeding. Further examination of the suspected animal's reproductive history and previous treatments for reproductive disorders in combination with ultrasonographic examination can then be used as a basis for diagnosis (Ball and Peters, 2004, Peters *et al.*, 2009).

#### **2.2.2.2.2 Paraovarian Cysts**

Paraovarian cysts is cystic structures that do not occur in the ovaries themselves, but rather in the broad ligament close to the ovaries and the uterine tubes (Rosenberg, 2010). Peter *et al.*, (2009) mentioned that palpation or ultrasonography technique can be used to detect them, and they appear as fluid-filled anechoic structures and are usually round or oval in shape, occur as a single cystic structure, and range from 1-5 cm in diameter. The author further clarify that a larger form of paraovarian cysts may also occur, but this structure is called hydatid of Morgagni and is similar to a reproductive complication in ewes and sows. There are two different types of paraovarian cysts; those derived from the cranial mesonephric tubules are called epooophoron, while those from the caudal tubules are referred to as paroophoron. All paraovarian cysts are benign, with no negative effects on reproduction and fertility (Rosenberg, 2010).

#### **2.2.2.2.3. Fibrin Tags**

Peter *et al.*, (2009) defined the Ovulation tags as small tags of fibrin that most commonly occur in heifers due to bleeding after ovulation and that they are frequently attached to the ovary at the site of a previous ovulation or on the medial attachment of the ovary to the uteroovarian ligament. While there is very little information on ovarian fibrin tags, it seems that they cause no complications to the estrous cycle or conception Peter *et al.*, (2009).

#### **2.2.2.2.4. Granulosa-theca Cell Tumors.**

Granulosa-theca cell tumors are made of both types of follicular cells, and are the most common variety of ovarian tumors in cattle, even though they are rare (<0.5%). They arise from the sex cord stromal tissue within the ovary and may be relatively small, solid and yellow to white or large filled with cysts of varying sizes and weight 11.9-12.3 kg (Peter *et al.*, 2009). Granulosa-theca cell tumors are most commonly benign but may be malignant and often hormonally active. If undiagnosed and left intact, clinical signs progress through various stages beginning with nymphomania and ending with virilism. In some instances, mammary development is observed (Peter *et al.*, 2009).

Diagnosis can be made through observations of clinical signs combined with transrectal palpation and ultrasonography. Clinical signs are abnormal estrous cycles and follicular and luteal inactivity on the contralateral ovary (Peter *et al.*, 2009).

#### **2.2.2.2.5. Rete Ovarii**

In the hilus region of a mature ovary, the rete ovarii is found as a network arrangement of medullary tubules or cords near the mesoovarian (Peter *et al.*, 2009). The medullary cords of the rete ovarii are lined with both cuboidal and columnar epithelium, differentiating to form granulosa-like cells that have a secretory function. Cysts associated with rete ovarii are not pathological in function, but may inhibit ovarian function if the rete ovarii has a space-occupying

lesion. Diagnosis and differentiation from other ovarian structures can be accomplished through transrectal ultrasonic examination of the reproductive tract.

#### **2.2.2.2.6. Ovarian Hypoplasia**

Ovarian hypoplasia is a condition in which one or both ovaries are smaller than normal. The left ovary is most commonly affected by ovarian hypoplasia, with greater than 80% of cases showing left ovarian hypoplasia (Smith, 2009).but both may be adversely affected. This condition is inheritable in the Swedish Highland Breed and potentially other breeds of cattle as well. Bilaterally hypoplastic ovaries results in complete infertility while unilateral ovarian hypoplasia results in lower fertility than normal (Smith, 2009). Ovarian Hypoplasia occurs sporadically in the cattle population. An autosomal recessive gene with incomplete penetrance potentiates the incidence of bovine ovarian hypoplasia. This means that either the bull or the cow can possess a defective gene leading to affected offspring, but its effects can vary in severity. It is possible for one or both ovaries to be affected.

#### **Symptoms:**

In cases of bilateral ovarian hypoplasia heifers do not develop secondary sexual characteristics. These animals are anoestrus and infertile. However, when the condition is unilateral, normal sexual organs and oestrous activity may be observed. Cattle with unilateral ovarian hypoplasia are fertile, although less so than normal. Grossly the affected ovary may be smaller and cord-like or slightly larger and bean shaped. When the condition is bilateral, the vagina and uterus will often stay infantile and fail to develop normally at puberty. If the condition is unilateral, the internal tubular reproductive structures can develop to a normal size (Smith, 2009).

**Diagnosis:**

Bovine ovarian hypoplasia is not easy to diagnose because it can present as complete infertility or subfertile. Diagnosis can be aided by a combination of rectal palpation of the reproductive tract as well as clinical signs. Palpation of small ovaries and an underdeveloped uterus in a cow that is not showing normal signs of estrus may have ovarian hypoplasia. It is important to differentiate ovarian hypoplasia from anestrus due to nutritional deficits or disease as well as non-functional ovaries. (Smith, 2009)

**Prevention:**

The incidence of gonadal hypoplasia can be reduced by using only animals (both male and female) with normal sexual organs as breeding stock. (Smith, 2009). Treatment of ovarian hypoplasia is not successful (Smith, 2009).

**2.2.2.3. Other causes of anoestrus:**

Anoestrous cows have small flaccid uteri and small, inactive ovaries with no palpable corpus luteum or follicle. In contrast, cycling cows are identified by the size and tone of the uterus and the presence of the corpus luteum or follicle or both on either of the ovaries. Nevertheless, cows may show anoestrus despite having normal ovarian structures (FAO, 1989).

Anoestrus is a major problem in the tropics and subtropics, where inadequate nutrition, high ambient temperature, high parasite burdens and disease exacerbate the problem. Low body weight and poor body condition, compounded with lactation stress, can further extend the postpartum anoestrous period (Rajendran, 2010).

Vandeplasseche (1982) indicated that the long anoestrous period in the nursing cow might be due to an elevated level of prolactin, which appears to depress the secretion and release of GnRH, or that the pituitary may be less responsive to GnRH during nursing. Since the anoestrous period tends to be longer

and more common among first-calf heifers, the author also suggested that immaturity could be a contributing factor.

The later author also mentioned that the ovaries of such cows may not be completely inactive, reduction in oestrogen secretion over long periods may result in underdevelopment of other genital organs. Blood or milk progesterone levels are also low. Conditions that simulate pregnancy, such as pyometra (pyometron), severe metritis, foetal maceration or mummification, may cause anoestrus. Pyometra, which often occurs when foetal membranes are retained or following postpartum metritis, is a frequent cause of anoestrus. These conditions damage the endometrial lining of the uterus and reduce secretion of luteolytic prostaglandin. The cyclic activity of the ovary is thus interrupted in the luteal phase and the cow or heifer is anoestrous until the condition is corrected (Vandeplassche, 1982). Without regular veterinary care and palpation, these conditions may remain undiagnosed and the cow may be believed to be pregnant. Although anoestrus can, to some degree, be overcome by treatment, it is more practical to ensure that animals are well managed and are fed to maintain good condition during critical periods, i.e. prior to mating and during lactation, in order to avoid anoestrus. Anoestrus is normal in pregnant, prepubertal and recently-calved animals. Pregnancy is a common cause of anoestrus, but this is often overlooked where service records are poor. Since many treatments for anoestrus terminate pregnancy, the possibility that cows presented as anoestrous are pregnant should be eliminated before treatment begins.

#### **2.2.2.4. Repeat Breeders:**

The term repeat breeder or repeat breeder syndrome (RBS) describes cows that failed to conceive after 3 or 4 inseminations (Enkhia *et al.*, 1983; Parkinson *et al.*, 2001). Repeat breeder cows return to service repeatedly after being bred with a fertile male. According to Parkinson *et al.*, (2001) these cows exhibit normal signs of estrus every 18 to 24 days but require more than 3 services to become pregnant.

Repeat breeding can be caused by a number of factors, including subfertile bulls, endocrine problems, malnutrition, reproductive tract infections and poor management (Vandeplasseche, 1982). According to Allen (1996) adhesions of the ovarian bursa; salpingitis, cystic ovaries and endometritis were found in repeat-breeding cows, some of these cases may be associated with early embryonic deaths, since most of the embryonic losses in cows occur much earlier in pregnancy. This theory is supported by (Ayalon, 1978; Maurer and Echternkamp., 1985) who reported that repeat breeders show a significant level of embryonic deaths. Ayalon (1978) reported that RBS is a major source of economic loss in dairy herds in North America and its prevalence ranges from 10% to 18% between different States. Bhatt *et al.*, (1979) found antibodies to seminal antigen in the genital secretions of 12 repeat-breeding cows in Bikaner, India. Munoz *et al.*, (1980) made a similar observation in Mexico where 32 out of 50 infertile Holstein cows had antibodies against Holstein bull spermatozoa.

According to Espey (1994) ovulation is initiated by the increase of the luteinizing hormone (LH) which results in the rupture of the follicle and release of an egg. Brackett (1980) reported that after ovulation there is a short period during which the egg can be fertilized. Since the period between the first signs of estrus and ovulation occurs at 72 h or later, the cows that ovulate later should be inseminated daily until rectal detection of ovulation is confirmed. This phenomenon is known as a delayed ovulation. Delayed ovulation should not be confused with repeat breeder syndrome (RBS).

Proper diagnosis of the cause of repeat breeding is very important and requires a careful assessment of production and breeding records (Wani, 2012). Repeat breeding can be treated by enucleating the corpus luteum or causing its lysis by prostaglandins, uterine massage or manual stimulation of the clitoris after artificial insemination or infusion of the uterus with 50-200 ml of 1 to 5% Lugol's iodine which has a stimulating effect on the uterus. Enucleation of the corpus

luteum may cause adhesions between the ovary, bursa and fimbria or haemorrhage in the ovary (Roberts, 1973). Hormonal enucleation with prostaglandins avoids this problem. Intra-uterine infusion of antibiotics has been used but there is little evidence that this increases cows fertility during the following cycle. However, fertility tends to increase by the second or third heat after treatment. Human chorionic gonadotropin (hCG) can be administered at the time of AI to promote ovulation, or progesterone hormone can be injected 4 to 5 days after service. A gonadotropin-releasing hormone (GnRH) application, significantly increases the conception rate in repeat breeders (Stevenson *et al.*, 1990, and Morgan and Lean, 1993). On the other hand, Singh *et al.*, (2005) suggested that frequent insemination (every 6 h) can increase the conception rate in cases of RBS. However, no significant results were found by double (repeated) insemination of cows within 12 or 18 h without an application of GnRH, (Stevenson *et al.*, 1990). Therefore, GnRH must be considered in the hormone treatment for RBS. According to Dawson, 1998, and Levine, 1999 and the work Morgan and Lean (1993), the only effective treatment option for RBS in dairy cows is the administration of GnRH at the time of insemination. Strategies may be used to optimize the time of insemination, including an Intravaginal progesterone releasing device through a controlled internal drug release (CIDR) program, as shown by (Day *et al.*, 2000) and the Ovsynch protocol (Pursley *et al.*, 1995).

### **2.2.3. Bacterial And protozoan Infections:**

Pathological lesions were observed in the reproductive tracts of 55.14% of 700 cows examined at post-mortem on two ranches in Shaba, Zaire (Binemo-Madi and Mposhy, 1982). Most of these were on the ovary and salphinx. They may have been caused by abnormal rectal manipulations or bacterial infections of the uterus, vagina and vestibulae. About 45% of the cows were still capable of breeding, indicating that pathological conditions do not necessarily render cows permanently sterile. Their seriousness depends on the location of the infection.

### 2.2.3.1 Brucellosis:

Brucellosis is one of the most common zoonotic diseases globally (Ariza *et al.*, 2007) and important disease of livestock (Nasir *et al.*, 2005) which still Remains as a widely prevalent zoonotic disease of public health and economic importance to livestock owners as well as to a nation (Schelling *et al.*, 2003, Zinsstag *et al.*, 2007)

Brucellosis is a disease with worldwide distribution that affects domestic animals, wild life and humans. In specificity particular, the disease in cattle is caused by *Brucella abortus* and is mainly characterized by abortions, infertility and reduced milk yield (Berhanu *et al.*, 2013).

Other species of Brucella is *B. melitensis*, *B. suis*, *B. ovis* and *B. canis*. De *et al.* ,(1982) in a study of 989 cows in West Bengal, attributed abnormal termination of pregnancy, cervicitis, endometritis, repeat breeding and anoestrus to brucellosis, Campylobacteriosis, leptospirosis and trichomoniasis. Other infections, can lead to inflammation with bursar and uterine adhesions, periglandular fibrosis, hydro- and pyo-salpinx, hydrometra, pyometra, endometritis, vaginitis, and metritis (Rao *et al.*, 1977,; Kaikini *et al.*, 1983,; Hussain and Muniraju., 1984). Other effects include early embryonic loss and repeat breeding, abortion, dystocia, retained membranes, stillbirth, and Prolapse in late gestation.

Brucellosis has been extensively studied, because the disease of major public health importance and it causes widespread economic losses (Abdou, 2000 due to abortion and infertility extended calving intervals and because of it is a zoonotic it poses a serious hazard for human health worldwide (Hamidy and Amin 2002; Angara, 2004; Zinsstag *et al.* 2007).

The reported incidence of brucellosis in Africa varies from zero to 100% (Chukwu, 1985). Some of the variation can be attributed to sampling technique, the breed of herds sampled and the diagnostic tests used. Some cows also only react positively to serological tests when pregnant.



The disease is most serious in cows infected during pregnancy. The bacteria show a preference for the pregnant uterus, foetus and the lymph glands of the udder. The organism may also produce toxins and allergens which cause vascular thrombosis, increase uterine motility, and disturb production of sex steroids and prostaglandins, contributing to abortion at about 6 to 8 months of gestation (Vandeplasseche, 1982). In some cases the dead foetus is not aborted, but is retained in a mummified or macerated form. If a calf is born alive it is likely to be weak and to contract calf scours easily. Many die soon after delivery (Vandeplasseche, 1982).

Aborted infected cows or heifers are a major source of infection for other cattle and people handling them. Aborted material and vaginal discharges from infected females are heavily infected with *Brucella*, and these contaminate pastures, pens and buildings. Organisms are also present in the milk of infected cows. Brucellosis is a professional hazard for cattle keepers and veterinarians. Foetal membranes are commonly retained because of uterine inertia, placentitis or both. Puerperal metritis may develop and cows may remain infertile for some time (FAO, 1989).

After abortion, uterine infection normally declines within a month. The cow may not abort on the next conception, but she will continue to discharge the *Brucella*. Some calves are born infected. Many lose the infection quickly but a few do not. The latter do not show any signs of the disease and represent "latent infection". The organism remains dormant until the animal becomes pregnant. Calves born to serologically positive dams are, therefore, at risk of developing the disease in the future and ought to be carefully screened when pregnant, and milk infection lasts several months or years and may be the source of uterine infection during subsequent pregnancies (Alton, 1981).

## Diagnosis

Brucellosis should be suspected whenever a cow aborts unexpectedly, except in a Brucella-free herd. Confirmation requires bacteriological examination, culture of the organism or serodiagnosis.

A smear from the necrotic surface of placental cotyledons, stained with 20% fuchsin, 3% acetic acid and 10% methylene blue, can assist the first tentative diagnosis of brucellosis. The Brucella stain red against a blue background. Chlamydia may also stain red but are smaller and primarily intracellular (Vandeplasseche, 1982).

The bacteria *Br. abortus* are rarely cultured, partly because diagnostic material, particularly fetuses, usually reaches the laboratory in a condition unsuitable for proper examination. Serological tests are, therefore, commonly employed. However, the various tests used differ in convenience and accuracy. A good serological test would establish early diagnosis, identify chronic infections, and distinguish between the antibodies of vaccination and infection (Fensterbank, 1986).

In the milk ring test (MRT), also called the Bang Ring Test, a drop of haematoxylin-stained antigen is added to 1 ml of milk. This is incubated at 37°C for half to 1 hour. This test is widely used, fairly efficient, economical and easy to perform. A positive result is shown by the development of a clump of stained organisms deposited in a ring on the surface of the preparation. The negative result is bluish milk covered by an uncoloured layer of cream. However, the sensitivity and specificity of this test are low, and test results can even vary for the same animal at different periods. It is therefore used only for quick screening or surveillance of milk samples (Hendry *et al.*, 1985).

In the spot agglutination test, which was developed in the United States (Alton, 1981), a drop each of serum and antigen are mixed on a card or on a

plastic, ceramic or glass plate. This is also known as the Rose Bengal Test (RBT) or rapid plate agglutination test. RBT is performed on serum using stained antigen at pH 3.6. It is economical, simple to perform and gives results in 4 minutes. Like the MRT, it is used as a quick screening test. A positive result is indicated by clear agglutination. To aid judgment, a known positive sample is run at the same time for comparison. The Rose Bengal plate test (RBPT) is the most widely used screening test for brucellosis in both humans and animals for its easiness and apparent simplicity of reading; however, interpretations of the RBPT results can be affected by personal experience (Cho *et al.*, 2010).

More sensitive and specific tests include the complement fixation test (CFT), which detects IgG1 and IgM antibodies (Fensterbank, 1986). This is the most accurate and sensitive test for brucellosis and distinguishes between antibodies of infection and vaccination. However, it must be performed by a trained technician.

The enzyme-linked immunosorbent assay (ELISA) has been used to diagnose brucellosis (Stemshorn *et al.*, 1980), but has not been extensively adopted as a routine test. Diaz *et al.*, (1979) described a simple immuno-diffusion test that they thought could differentiate between infected and vaccinated animals. Kaneene *et al.*, (1979) reported that the level of immunity among vaccinated cattle could be assessed by exposing peripheral lymphocytes to *Brucella* antigen in vitro. They also indicated that this procedure could be used to diagnose the disease (Kaneene *et al.*, 1979).

### **Control**

There is no treatment for brucellosis, prevention accomplished by vaccination and entire herd test and slaughter of (positive) reactors. Official calf hood vaccination of heifer calves, vaccination must done by an accredited veterinarian at calve age that vary from two to four month, using standard dosage vaccine or for four to 12 month using reduced vaccine. Quarantines are imposed on infected herd until the herd has been

proven free of the disease (Wulanto, 2012). Vaccination of calves with B abortus Strain 19 or RB51 increases resistance to infection. Resistance may not be complete, and some vaccinated calves may become infected, depending on severity of exposure. Although incidences of brucellosis in livestock and human populations significantly declined following effective vaccination based control and preventive programmes, it still remains a persistent problem in the endemic areas (Smits, 2005, Mantur and Amaranth 2008).

Brucellosis can be controlled through strict hygiene in the handling of potentially infected material, Cows, especially those that react positively to serological tests for brucellosis, should be isolated from the herd before calving and their calves monitored for latent infection. Pregnant animals should be observed for imminent abortion and all aborted material must be disposed of properly, e.g. by burning or deep burial. Farm laborers should be aware of the dangers of brucellosis and avoid spreading the disease. To eradicate the disease, all infected animals must be slaughtered (FAO, 1989).

Non infected herds must be protected. The greatest danger is from replacement animals. Additions should be vaccinated during calf hood beside the nonpregnant heifers. If pregnant or fresh cows are added, they should originate from brucellosis-free areas or herds and be seronegative. If this is not possible, animals should be tested serologically on the farm of origin prior to purchase and again one month later after arrival on the new farm. They should be kept in quarantine until after the second test. If an animal reacts positively to the MRT or RBT tests, the result should be checked using the CFT test. If the latter is also positive, the animal should be slaughtered (FAO, 1989).

Where the incidence of brucellosis is high, calves should be vaccinated with the attenuated Strain 19 vaccine at 3 to 6 months old. Persistent antibody titers that may be hard to distinguish from infection, and infection of the udder, have been

observed in heifers vaccinated at 8 months or older. Vaccinal titers tend to recede more rapidly when heifers are vaccinated at 6 months or younger (Carroll, 1972) and there is no difference in immunity levels of calves vaccinated at 3,4,6 or 8 months old (Mathei and Deyoe, 1970). Sexually mature animals can be vaccinated with the killed adjuvant vaccine 45/20. The vaccine is given twice, 6 weeks apart, followed by an annual booster.

Bulls should not be vaccinated. Strain 19 organisms have been isolated from the genitalia of vaccinated bulls (Lambert *et al.*, 1964). These could infect their seminal vesicles, epididymides and testes. Bulls often stay in the herd for a short time, during which they do not transmit the organisms naturally. However, bulls at artificial insemination centres must be rigorously tested for the disease. Non-specific post-vaccination reactions to serum agglutination tests have also been observed among AI bulls. Bell (1984) believed that the cause could be an anomaly of the immune system, particularly of IgM.

Prevention of the disease in humans is contingent upon the control of the disease in animals. Once the incidence of the disease is substantially reduced by vaccination, a test and slaughter programme can be attempted to eliminate infected animals. Once a herd has been certified as being free of the disease, continuing vaccination may not be necessary but the herd must be kept closed (Zinsstag *et al.*, 2007).

### **2.2.3.2 Trichomoniasis:**

Bovine trichomoniasis is a venereal disease of cattle caused by the parasite *Trichomonas fetus* it was an important cause of infertility in cattle in many countries (Baquer, 2013). *Trichomonas fetus*, a protozoan about 10 to 25 µm in length and 5-10 µm in width with an undulating membrane. The infection varies in cow from mild vaginitis or cervicitis to endometritis, pyometra, abortion and sterility causing significant economic loss (BonDurant, 2005; Benchimol *et al.*, 2007).

The incidence of trichomoniasis in Africa and the tropics is not widely reported, partly because diagnosis is complex and time-consuming. Consequently, it is not clear if the disease is widespread. De *et al.*, (1982) did not find infected animals among 13 well-managed herds in West Bengal, India. Only one cow from a rural herd was diagnosed as being infected. Klastrup and Halliwell (1977) also failed to demonstrate the disease among 294 slaughtered bulls and 54 others maintained at breeding centres in Malawi. However, in Egypt, Gawade *et al.*, (1981) found an incidence of 4.6% among Holstein bulls, and in Nigeria (Akinboade, 1980) found an incidence of 14.9% among slaughtered animals.

In bulls, the trichomonads normally colonise the crypts of the external mucous membrane of the penis and prepuce. Since these crypts are deeper in older bulls, the prevalence of the disease tends to increase with bull age, for this reason using young bulls is part of a disease management strategy (Baquer, 2013). Infection does not induce any local antibodies or specific agglutinins in the blood of bulls. Bulls carry the disease for a long time without showing symptoms (Helena 2012).

Cows and heifers that have never been exposed to the disease become infected following either natural service by a carrier bull or artificial inseminations with contaminated semen. Following natural service, the protozoa first multiply in the vagina and cervix for about 3 weeks. In about a quarter of the cows, the organisms do not migrate to the uterus. With intrauterine artificial insemination, the uterus is directly infected (FAO, 1989).

Trichomoniasis causes infertility, repeat breeding, delayed return to oestrus after mating, early embryonic death and, sometimes, abortion. It may directly cause the death of the embryo or may do so via uterine endometritis and marked leucocytic diapodesis into the endometrium (Vandeplassche, 1982). The affected cow returns to oestrus or may abort anytime from 2 to 7 months after conception. Affected cows develop agglutinating antibodies in their vaginal mucus. This, together with hormonal changes during subsequent oestrous cycles, tends to protect

the cow during an infection but may not protect her from re-infection. Withdrawing infected cows from breeding for at least 3 months and subsequent use of clean bulls or artificial insemination can help control the disease (FAO, 1989).

### **Diagnosis**

After incubation period, pyometra and abortion in the first trimester occur, which is the first physiological signs of disease occur, resulting in repeat breeding, irregular heat cycle, longer calving and reduce pregnancy rate, the uterus may become infected in some cases (Gregory *et al.*, 1990). Infertility due to early embryonic death is the most economical clinical signs of disease (Bondurant *et al.*, 1990). The symptoms of trichomoniasis and Campylobacteriosis are similar. Both lead to irregular inter-oestrous intervals. They are best differentiated by isolating the causative agents.

Many sampling techniques have been used for obtaining diagnostic specimens in the bull, they including ( swab technique , dry pipette technique , wet pipette technique , the douche technique and metal brush technique ) these techniques focuses on recovering preputial smegma for either direct microscopic evaluation or in vitro cultivation (Parker *et al.*, 1999), A tentative diagnosis may be based on the history and clinical signs to confirm this diagnosis depends on finding the organism in at least one animal in the herd (Clark, 1997) . This is done by an official diagnostic laboratory finding the organism in an aborted fetus, culturing the organism from a vaginal tract swab of a cow or from the pyometral discharge from a cow, and /or finding the organism in a smegma collected from the inside sheath around the penis of one of the herd bulls (Felleisen *et al.*, 1997, Mukhufhi, 2003).

Cervical and vaginal mucus can also be examined but this is only really useful during the first few weeks of infection. Thereafter, the motility of the trichomonads diminishes and only the undulating membrane can be discerned at 250x magnification. In the case of an abortion, microscopic examination of foetal

fluids, placental secretions and foetal abomasal contents can be useful (Felleisen *et al.*, 1997)

Isolation of even one trichomonad, from either the cow or the bull confirms diagnosis. Only few trichomonads may be present and, if the disease is suspected, a second sample should be taken if no trichomonad is found in the first. Samples should be examined within 6 hours of sampling as the parasite tends to die within 6 hours of leaving the animal's body (Felleisen *et al.*, 1997). At temperatures lower than 37°C the undulation of the parasite tends to slow or stop. Thus techniques suitable for field studies in the tropics need to be developed (FAO, 1989). As an alternative to direct examination, preputial washings or purulent discharges can be cultured, preferably within 6 hours of sampling. Immediate inoculation onto specific culture media is advisable. Where this is not possible, especially under field conditions, transport media may be used. Buffered saline solution with foetal bovine serum and lactated Ringer's solution are simple and effective transport media. Vandeplassche (1982) suggested Difco-Lash medium (No.D-1016T), and Oxoid medium as culture media. Klastrup and Halliwell (1977) referred to two trichomoniasis culture media, one with and one without antibiotic, while De *et al.*, (1982) used Douglas' broth and glucose broth serum medium. Both direct examination and culture methods can take time to yield results. Sero-diagnostic procedures are generally unsatisfactory (De *et al.*, 1982).

### **Treatment and control**

Treatment of infected cows with vaginal antiseptics has not been very successful. In animals with pyometra, it is better to enucleate the corpus luteum or to lyse it with prostaglandins. Treatment may be repeated 10 or 11 days later.

Trichomoniasis is a self-limiting disease in the non-pregnant cow with an involuted uterus there is no effective treatment for Trichomoniasis in cattle (Wilson *et al.*, 1979). After being sexually rested for 3 or 5 cycles, many cows develop some



immunity and their fertility improves. Only clean bulls or semen should be used for breeding and cows with abnormal genital tracts should be culled (FAO, 1989).

Carrier bulls and sexually active oxen can re-infect treated, recovered and susceptible females and should therefore be culled. Carrier bulls can be treated, but treatment is lengthy and should not be considered unless the bull is very valuable (Mancebo, 1995).

AI centres must therefore test their bulls regularly to ensure that they are not infected. A few tips for prevention bovine trichomoniasis including (keep fences in good repair to prevent accidental contact with potentially infected cattle, replacement heifers should either be pregnant or less than six months of age, do not retain open females that failed to breed the year before, replacement bulls should be known virgins, or have negative test before they enter a herd (Mancebo, 1995).

#### **2.2.4: Endometritis, Metritis and puerperal metritis**

Puerperal metritis is a severe disease that adversely affects milk production and reproduction, and the cow can have many metabolic disorders undertake potentially life (Overton, 2003). Metritis is defined as inflammation of the muscular wall of the uterus and endometrium (Okker *et al.*, 2002). The serious cases occur during the first 10-14 days postpartum and are sometimes called toxic puerperal metritis, acute metritis postpartum or puerperal metritis (Chenault *et al.*, 2004). The incidence of toxic metritis varies from 2.2% to 37.3%. Cows affected exhibit varying degrees of depression, poor appetite and reduced milk production and are predisposed to suffer from disorders abomasums (Radostits *et al.*, 2000 and Overton, 2003).

If metritis is not rapidly diagnosed and treated it would lead to fecundity and sterility. Therefore, the economic rentability of the herd would be affected seriously (Zidane *et al.*, 2011). Delayed uterine involution and endometritis are generally the most common consequences of retained placenta; they are detrimental to the future breeding of the cow with altered parameters of fertility,

thus causing a prolongation of the calving fertilizing insemination interval (Graves, 2002, Melendez *et al.*, 2004).

In the post-partum normal involution requires 25 to 50 days to complete and includes a reduction in uterine size, necrosis and shrinkage of the caruncle and endometrial epithelization. The size reduction begins immediately after partum, and during the first 10 days is relatively slow compared to what happens between 10-14 days postpartum (Studer and Morrow, 1978; Leslie, 1983). This initial reduction is due to uterine contractions caused by oxytocin, occurring every 3-4 minutes during the first days and possibly persists until the third day postpartum. Suckling is associated with a more frequent release of oxytocin from the pituitary gland than that in the milking and this is possibly why the beef cattle have a shorter period of involution than those in dairy cattle. When doing rectal palpation, the uterus postpartum normal should have longitudinal striations due to the substantial reduction in size (Smith and Risco, 2002).

The involution of the uterus is not a sterile process. There are a lot of postpartum secretions, which need be removed in a few weeks (Leslie, 1983; Youngquist and Shore., 1997). Between 58 and 93% of cows have uterine infection 2 weeks after partum, but only 5-9% to remain infected 45-60 days postpartum (Studer and Morrow, 1978; Leslie, 1983). As Hussain (1989) and Cai *et al.*, (1994) described, the effectiveness of neutrophils depends on their ability to move to the site of infection and destroy the invading pathogen which usually appears on the second day postpartum. Both complementary processes help the response of neutrophil to infection. Other components include eosinophils and mast cells in the endometrial surface. The binding of antigen to IgE unite to mast cell receptors helps the release of cell necrosis factor, histamine, prostaglandins, interleukins and quimotácticos factors for eosinophils and neutrophils. The damage of the surface of the endometrium due to mast cells and eosinophils may allow access of the immunoglobulins of serum to uterine lumen. Contractions of the myometrium and

secretions from the endometrial glands also help to remove potentially harmful bacteria (Paisley *et al.*, 1986). In conditions of Uterine trauma, such as dystocia, manual removal of retained placenta, and intrauterine infusions, reduced the phagocytic activity of uterine and blood neutrophils (Hussain, 1989).

There have been very few papers on the role of humoral immunity in the defense and cleaning of the uterus postpartum. Antibodies against *Streptococcus pyogenes* and *Arcanobacterium hemolyticus* have not been found in the vaginal mucus of heifer until several weeks after partum, regardless of their presence in the serum (Paisley *et al.*, 1986). The heifer may not have adequate levels of antibodies against *A.pyogene* this was found in microbiological culture of the uterus of 30% in heifer vs 6% in cows to 10 days postpartum (BonDurant, 1999).

The steroid hormones may play an important role in uterine defense. In rats the diversity and concentration of immune cells in the endometrium have shown increases in number with increased concentrations of estrogen. High levels of estrogen may also increase the concentrations of IgA and IgG, and increase the efficiency of presenting of antigen to uterine cells. The bacteria begin to decline sometime in the postpartum, when the pituitary is capable of responding to GnRH and begins pulsatility of estrogen (BonDurant, 1999).

Tennant and Peddicord (1968) found that endometritis, as indicated by pus in the vagina, significantly reduces fertility. Cows with endometritis required significantly ( $P < 0.001$ ) more services per conception (2.0 vs 1.6), had lower conception to first service rate (49 vs 62%,  $P < 0.001$ ) and longer calving interval (394 vs 383 days,  $P < 0.001$ ), and more animals were culled for infertility (13.6 vs 6.2%).

Infections of the reproductive tract are usually contracted at parturition. Non-specific infections of the uterus are more common where the placenta is retained, in cows that need assistance with calving and in cows with milk fever (Bruun *et al.*, 2002). Metritis is often associated with uterine atony or inertia. Acute metritis

causes fever and depression within a week of infection, and is commonly followed by chronic metritis, with persistent purulent vaginal discharge. Specific venereal infections, such as trichomoniasis, Campylobacteriosis and brucellosis, may also lead to metritis (Rao *et al.*, 1977; Kaikini *et al.*, 1983; Hussain and Muniraju., 1984).

Pyometra is the accumulation of pus in the uterus (Arthur, 1989). It is a common cause of anoestrus and cows with pyometra should be treated promptly. Postpartum metritis, endometritis and pyometra may be common where cows and heifers are confined at delivery time in a building or area in which others have recently calved (Bartlett *et al.*, 1986, Olson, 1986).

The uterus can resist or eliminate bacteria infection. However, this ability is related to ovarian activity ,The uterus is highly resistant to infection during the estrogenic phase but very susceptible during the period of progesterone dominance, because (1) pH in the uterus is low, allowing greater bacterial growth, (2) the epithelium is less permeable to bacteria and therefore the leucocytic system is stimulated at a later stage, (3) the appearance of leucocytes in the lumen is delayed, (4) the activity of leucocytes is decreased, and (5) uterine secretions have no detoxicating effect (Paisley *et al.*, 1986). As a result, some cases of metritis resolve spontaneously when the animal's oestrous cycles resume, while others remain chronic.

### **Diagnosis**

Timely and accurate diagnoses are essential to ensure appropriate management of uterine infections. However, some researchers (Paisley 1986; Gilbert and Schwark, 1992) suggest that, diagnoses of uterine infections are too subjective and often are inaccurate. Rectal palpation of the uterus, examination of the vagina with a speculum, culture of uterine fluids, and evaluation of uterine biopsies are the techniques available for diagnosing uterine infections , Rectal palpation is probably the most common method for diagnosing uterine infections, but it may be the most

insensitive and nonspecific method available (Bretzlaff, 1987; Gilbert and Schwark, 1992 ). Typically, the size and consistency of the uterus and cervix and the fluid content of the uterus are judged relative to the characteristics considered as normal for the given time postpartum (Bretzlaff, 1987; Youngquist and Shore, 1997). Typically, all palpable uterine fluids (lochia) are voided during the first 2 wk postpartum (Olson *et al.*, 1986). Lochia vary in color from whitish to reddish to dark brown, and the color of lochia is not a reliable sign of a problem. However, if the lochia become fetid, puerperal metritis may be suspected. Puerperal metritis can be a severe problem, and uterine infections that are life-threatening (i.e., septic-toxic metritis) are associated almost exclusively with this condition (Drillich *et al.*, 2001). Other clinical signs include depression, decreased appetite or anorexia, dehydration and lower milk production (Youngquist and Shore, 1997). It is common to have fever, a rectal temperature (RT) above 39 °C is considered to be a valid indication for the start of uterus inflammation (Palenik *et al.*, 2009). Kristula *et al.*, (2001) reported that cows experiencing no clinical problems at calving or during early postpartum had an average RT below 38.9 °C for each day during the first 10 days postpartum. However, cows with metritis may have rectal temperatures within the normal range and may not necessarily develop a fever. Benzaquen *et al.*, (2004) found that over half of the cows diagnosed with metritis during the first week postpartum did not have a fever in addition; some cows may have the typical discharges associated with uterine metritis without being systemically ill. These animals probably can suffer from subclinical metritis. Often, an animal is evaluated because it has a combination of

- Difficulty to remove the placenta.
- Do not eat,
- Is depressed or
- Has reduced milk production

The characteristic of longitudinal folds are that usually visible are absent (Youngquist and Shore, 1997). The most common means of diagnosis of endometritis is transrectal palpation of the uterus. However, this method is subjective and often fails to account for normal events and variability in uterine involution (Morrow *et al.*, 1966; Gier and Marion, 1968) or to have any association with reproductive performance (Studer and Morrow, 1978; Miller *et al.*, 1980; Lewis, 1997).

### **The Risk factors**

During partum the physical barriers to the control of pollution (vagina, cervix and vaginal vestibule) are severely compromised, and then the partum there's a lot of necrotic tissue and fluid creating an ideal environment for bacterial.

Dystocia, retained placenta, and cow age were significant variables in the equation to predict the incidence of uterine infections; however, the coefficient of multiple determination for the model used to develop the equation was 0.06, indicating that factors that were not usually included in cow records probably contributed to the development of uterine infections (Coleman *et al.*, 1985; Peters and Laven, 1996). The incidence of postpartum metritis in cows with retention of fetal membranes can be as high as 90% (Montes and Pugh, 1993). The chances of a cow with retention of fetal membrane to develop metritis is 6 times higher than those of cows free of retained membranes, which is much higher than any other risk factors.

The occurrence of double pregnancy is the leading cause of natural Retained fetal membranes in cattle (Smith and Risco, 2002). The percentage of twins in Holstein in the United States has increased in recent years due to the selection of to increase milk production (Kinsel, 1999, Wiltbank *et al.*, 2000).

### **Treatment**

Programs for the prevention or reduction of uterine infections included the local or systemic administration of antibiotics and/or the application of hormones (Dobson and Noakes 1990; Cairoli *et al.*, 1993; Lewis 1997; Arlt *et al.*, 2009). In

recent years, while searching for new, more efficient methods for treatment and prophylaxis, light therapy and the promising method of low intensity laser irradiation have been widely discussed. Low-intensity laser irradiation affects cell metabolism, stimulates regeneration, and reduces pain and inflammation (Hung *et al.*, 2009).

There is a lot of controversy about appropriate treatment regimes for post calving uterine infections (Currin, 2010). Studies on the treatment of subclinical endometritis with prostaglandin F<sub>2</sub>alpha or analogues, intrauterine antibiotics or proteolytic enzymes showed heterogeneous results (Lincke, 2007; Galvão 2011). In the treatment of metritis/endometritis antibiotics (Goshen and Shpigel, 2006), enzymes (Drillich *et al.*, 2001), hormones (Sheldon *et al.*, 2006) and an immunomodulator (Kaczmarowski *et al.*, 2004), in addition to other medicines (Paisley *et al.*, 1986; Malinowski, 1995) and antiseptic have been used.

### **Antimicrobial Therapy**

There are many methods concerning the treatment of specific forms of postpartum metritis with antimicrobial agents. Antimicrobial agents may be needed to control infection caused by bacteria and prevent the progression of disease (Ball *et al.*, 1984; Olson, 1985). Many antimicrobial agents are readily absorbed systemically from the uterus, including sulfonamides, tetracyclines, penicillin's, nitrofurazone, aminoglycosides, cephalosporins, and chloramphenicol have been used singularly or in combination for treating postpartum metritis (Gustafsson, 1984, Gilbert and Schwark, 1992).

A major consideration when using antibiotics in dairy cattle is drug residues, Currently, oxytetracycline is the only antimicrobial agent approved for use in lactating cows and thus labeled for the treatment of postpartum metritis, also Oxytetracycline is specifically labeled for acute or toxic puerperal metritis caused by strains of staphylococci and streptococci (Arrioja, 2001). All other antimicrobial agents are not labeled for treating any form of postpartum metritis. Because of

potential drug residues from extra label drug use, the US Dairy Quality Assurance Program was developed to ensure food safety. When treating postpartum metritis, various treatment routes, such as local (i.e., intrauterine) or systemic (i.e., IV, IM, SC), are available.

The infusion of antibiotic agents into the uterus is a common therapy used on all four forms of postpartum metritis. Local treatment involving intrauterine antibiotic infusion aims to produce an even distribution of an active drug throughout all layers of the uterus, limited systemic absorption, low tissue irritation, and high antibacterial activity within the uterine environment (Gustafsson, 1984; Gilbert, 1992).

Days postpartum, uterine condition, uterine tissue absorptive capabilities, and drug distribution are the major factors affecting the efficacy of most drugs infused into the postpartum uterus (Masera, 1980). The molecular structure of the antimicrobial agent and the vehicle used to deliver the drug influence its absorption into the uterine tissue after local uterine infusion (Gustafsson, 1984). The completely involuted uterus has better absorptive capabilities than the immediate postpartum uterus, Endometritis also results in poor concentration of drugs within the uterine tissue after intrauterine treatment (Masera, 1980; Ott, 1986). The result of poor local uterine absorption is a high concentration of drug on the endometrium but an inadequate concentration in the sub endometrial tissues, vagina, cervix, ovaries, and oviducts (Gustafsson, 1984). Also, the high concentration of drug on the endometrium may result in local irritation of the uterine lining, The environment of the postpartum uterus diminishes the efficacy of many drugs. Such factors as low oxygen tension (Aminoglycosides), antibiotic-degrading enzymes, mucopurulent discharge, and organic debris (sulfanomid) could lead to poor efficacy of certain antimicrobial agents infused into the postpartum uterus (Mickelsen, 1990). A mild case of endometritis may be the only form of



postpartum metritis in which intrauterine treatment is justified (Gustafsson, 1984). However, diagnosing mild endometritis is difficult in clinical practice. More severe pathologic changes, such as those seen in cows with post puerperal metritis, toxic puerperal metritis, and pyometra, may not respond to treatment by intrauterine infusion. One reason could be that these disease conditions greatly reduce uterine tissue uptake of the antimicrobial agent (Ott, 1986; Bretzlaff, *et al.*, 1983). Because of the many acknowledged shortcomings of intrauterine antibiotics, it is believed that intrauterine infusion alone often fails as a therapy for postpartum metritis (Gustafsson, 1984).

In invitro studies using samples of myometrium, it was shown that gentamicin sulfate inhibits the contraction uterine spontaneous or induced by oxytocin and PGF<sub>2</sub> $\alpha$  in a dose-dependent (Ocal *et al.*, 2004).

In a retrospective analysis of 78 cases of postpartum metritis, showed that all that was needed was administer treatment to avoid death while the uterus was recovered only (Pugh *et al.*, 1994). The use of antibiotics as a preventive measure in cases of retained placenta may be useful, but there are very few controlled trials that assessed its efficacy to prevent metritis in particular in relation to their cost and time of completion (Peters and Laven, 1996).

## **Intrauterine Therapy**

### **Non antibiotic Treatment**

Antiseptic agents, such as iodine, chlorhexidine, and saline, have been infused into the uterus, but there have been few studies to determine the efficacy of these compounds on postpartum metritis (Bouters and Vandeplassche, 1977). The only approved non antibiotic antimicrobial drug for intrauterine treatment in the United States is chlorhexidine, The irritating nature of such solutions is thought to increase uterine tone, blood flow, and defense mechanisms (Bretzlaff, 1987). The induced inflammatory response of the uterus is thought to reduce the bacteria level within the uterus and aid in Draining uterine fluids from cows with acute puerperal

metritis is a common, but apparently ill-advised, procedure; manipulation of an infected uterus may exacerbate the problem (Gilbert and Schwark, 1992). Cattle infused with an irritating chemical reportedly had a shortened estrous cycle (8 to 10 days) when the solution was administered early in the diestrus period (Nakahara,1967).On the other hand the infusion of iodine solution in water or saline is the most common. Few studies have assessed the potential harmful effects of the infusion of iodine on the future reproductive function. It has been reported that the infusion of 50 to 100 ml of a 2% solution of povidone iodine once as routine therapy the day 30 of postpartum, had a detrimental effect on fertility in cows with metritis, compared with untreated animals (Youngquist and Shore, 1997).

Intrauterine infusion of various antimicrobial-antibacterial compounds is a traditional treatment, but, based on current evidence, intrauterine infusions do not seem efficacious and may be harmful (Paisley *et al.*,1986; Hussain and Daniel., 1991; Gilbert and Schwark, 1992; Thurmond *et al.*,1993 and Pugh *et al.*,1994 ;).

In general, the infusion of non antibiotic substances into the postpartum uterus is not recommended. This method of treatment can lead to iatrogenic mechanical trauma to the genital tract and secondary bacterial infection through iatrogenic contamination of the genital tract, The ideal treatment should remove harmful bacteria from the uterus without harming the uterus or its own defense mechanisms., When infused into the uterus we have no certainty that the drug is distributed through all layers of the uterus, the ovaries, oviducts and the deep layers of the uterine wall receive minimum levels of antibiotic in the best case (Okker *et al.*, 2002, Sheldon *et al.*, 2006). In addition, intrauterine antimicrobial-antibacterial compounds may reach the systemic circulation and leave residues in milk (Gilbert and Schwark, 1992; Lowder, 1993).

## **Systemic Therapy**

Systemic treatment of all forms of postpartum metritis may be more advantageous than intrauterine treatment. Systemic treatment provides better drug distribution to all layers of the female genital tract and ovaries (Masera *et al*, 1980; Gustfsson and Ott, 1986). It prevents iatrogenic ally induced contamination of the uterus and injury to the uterine endometrium (Mickelsen, 1990). Systemic treatment of postpartum metritis also avoids interference of leukocyte function (Vandeplassche, 1976; Jayappa, 1983) this could be the most valuable reason to choose systemic over local antimicrobial treatment. In addition, systemic antimicrobial treatment using drugs, routes, and doses approved for other disease indications allows the establishment of milk and meat withholding times.

Penicillin administered systemically is a treatment option for endometritis, post puerperal metritis, and toxic puerperal metritis. Penicillin is preferred to other antibiotics because its distribution to all layers of the uterus is excellent, (Masera,1980; Gustafsson,1984) it is inexpensive, and it has established milk and meat withholding times when used according to label ,and most of the bacteria that penetrate the endometrium and cause septicemia are sensitive to penicillin (Ott, 1986; Paisley *et al.*, 1986) Unfortunately, treating postpartum metritis with penicillin is an off-label use because the drug is not indicated for the disease and the dosage of 9,000 to 18,000 U/lb IM once or twice daily is three to six times higher than what is recommended on the label. There is no known withholding time for using penicillin in an off-label manner. However, having available, easy-to-use residue test kit helps make penicillin a preferred treatment of postpartum metritis.

Oxytetracycline is also used to treat postpartum metritis. It is most often used in cows suffering from less severe forms of metritis (i.e., endometritis, post puerperal metritis (Gilbert, 1992). There are many concerns about treating

postpartum metritis with systemic oxytetracycline. Research by (Bretzlaff *et al.*, 1982) revealed that 11 mg/kg IV q12h rather than q24h was needed to provide uterine tissue concentrations that would combat infection. Concentrations slightly higher and more persistent and longer periods were found in the caruncle and endometrium (Bretzlaff *et al.*, 1983). Minimum inhibitory concentrations (MIC) for *Arcanobacterium pyogenes* in samples uterine have been reported as 20.4 g / ml of solution. (Olson *et al.*, 1986); However, a recent report showed high MIC 32 mg / ml solution for various strains of *A. Pyogenes* cultured wombs of cows with metritis (Sheldon *et al.*, 2004). This same work also showed that several strains of *E.coli* were resistant to oxytetracycline. This information suggests that parenteral oxytetracycline is a poor treatment for postpartum metritis this is an impractical regimen. Because of these concerns, the use of systemic oxytetracycline has not been considered the preferred choice for treating postpartum metritis even though it is the only antimicrobial agent approved by the Food and drug administration FDA for this treatment.

Antimicrobial agents have been used in combination to treat certain forms of postpartum metritis. Some authors have recommended systemic penicillin in conjunction with intrauterine oxytetracycline for treating toxic puerperal metritis (Olson *et al.*, 1986; Whitacre, 1992). However, the combination of penicillin's and tetracycline has resulted in residues in milk for excessively long periods (Haaland and Manspeaker, 1984). This results in discarded milk for a prolonged period, thereby decreasing milk revenues. Furthermore, the failure of dairies to heed milk withholding times or perform milk antibiotic residue tests may lead to antibiotic contamination of milk.

Because of concern over antibiotic residues, a reevaluation of the current approach to treating cows affected with metritis is needed. A reliable treatment alternative that controls the effects of postpartum metritis and avoids the problem

of milk antibiotic residues is needed. The use of ceftiofur sodium (which has a zero milk withdrawal time at the recommended label indication, dose, and route) for treating toxic puerperal metritis has been evaluated. It is important to note that ceftiofur sodium is not labeled for treating cows diagnosed with postpartum metritis. Therefore, its use is considered extra label.

The ceftiofur hydrochloride and sodium ceftiofur are concentrated in the uterine tissues at levels that exceed the minimum inhibitory concentration for *Arcanobacterium pyogenes*, *Fusobacterium necrophorum* and *Escherichia coli* (Okker *et al.*, 2002; Smith and Risco, 2002). A higher dose of ceftiofur hydrochloride 2.2 mg / kg IM or SC did not significantly improve the cure rate in cows with metritis extent 14 days after postpartum, compared with a dose of 1.1 mg / kg (Chenault *et al.*, 2004). By contrast, ceftiofur was highly effective against *E.coli*, *A. Pyogenes*, *E. necrophorum* and *P. melaninogenicus* at relatively low concentrations (Sheldon *et al.*, 2004) and concentrated very well in the uterine wall (Okker *et al.*, 2002). They must therefore be the drug of choice for the treatment of metritis in the cow (Sheldon *et al.*, 2004).

Smith *et al.*, (1998) studied the effect of ceftiofur sodium on toxic puerperal metritis. Postpartum dairy cows diagnosed with toxic puerperal metritis based on rectal temperature, milk production, and uterine discharge characteristics were assigned to three different antibiotic groups. The results of this study showed that all groups had a favorable clinical response to treatment based on changes in milk production and rectal temperature. Statistically, no difference in milk yield, rectal temperature, or serum haptoglobin was seen between treatment groups. The study found no difference in treatment efficacy among penicillin, oxytetracycline, or ceftiofur in cows with toxic puerperal metritis.

Several studies have since shown that ceftiofur given at 1 mg/kg/day IM or SC for 3 to 5 days is an effective antimicrobial choice for treating cows diagnosed with toxic puerperal metritis (Zhou *et al.*, 2001, Schmitt, 2001). Postpartum

metritis could be effectively treated with ceftiofur because the antibiotic is known to concentrate in infected tissues in vivo (Clarke *et al.*, 1996). Also, in a study by (Schmitt *et al.*, 2001) concentrations of ceftiofur and active metabolites in plasma, uterine tissues, and lochial fluid exceeded the reported mean inhibitory concentrations of the causative organisms (e.g., *Escherichia coli*, *Fusobacterium necrophorum*, *Arcanobacterium pyogenes*) most often isolated from cows diagnosed with toxic puerperal metritis (Zhou, 2001).

### **Hormone Therapy**

Prostaglandin F<sub>2</sub> $\alpha$  and its analogues have been advocated in managing postpartum metritis. The reasons for using PGF<sub>2</sub> $\alpha$  includes stimulation of uterine contraction, which aids in expelling purulent uterine fluid and debris (Hopkins, 1983, Young and Anderson, 1984); stimulation of leukocytes (Razin and Globerson, 1978; Frank *et al.*, 1983) and luteolytic with induction of an estrous cycle, which consequently reduces progesterone and increases estrogen levels (Gilbert, 1992). Many studies have been conducted to determine the value of administering PGF<sub>2</sub> $\alpha$  to abnormal cows (e.g., cows with dystocia, RFMs, postpartum metritis) during the immediate postpartum period (Richardson *et al.*, 1983; Bonnet *et al.*, 1990 and Okuda, 1998).

The plasma levels of metabolites of PGF reaching a peak in the fourth day of postpartum and then decline, reaching basal levels between 14 and 20 days postpartum (Leslie, 1983). Some publications suggest that higher concentrations longer have been associated with a more rapid uterine involution (Smith and Risco, 2002), and reduces the size of uteri (Melendez *et al.*, 2004). However, it is unlikely to produce a large effect of PGF<sub>2</sub> $\alpha$  in absence of luteal tissue in the ovaries (Frazer, 2001; Smith and Risco, 2002). It is expected that the use of inhibitors of cyclooxygenase such as flunixin meglumine reduce the production of PGF<sub>2</sub> $\alpha$ , however, when it was used, the rate of reduction in size of the uterine horn and cervix were not affected, even with a decrease of 80% in the endogenous

production of PGF<sub>2</sub> $\alpha$ , Intravenous doses (15 mg dinoprost) have been much more effective, but only up to 4 days postpartum (Frazer, 2001).

### **Estrogens**

Estradiol cypionate (the oil-based 17-cyclopentyl propionate ester of estradiol) has been the only approved estrogen for lactating dairy cows in the United States since the 1960 (Gustafsson, 1984). It provides estradiol-17 $\beta$ —one of the most potent estrogens (Bayley, 1999). It is FDA approved for treating anestrus and persistent corpus luteum. It is also indicated for expelling purulent material from the uterus of cows with pyometra and stimulating uterine expulsion of retained fetal membranes (RFMs) in lactating dairy cows (Bayley, 1999). There is a clear association between RFMs and postpartum metritis. Therefore, estrogens may actually prevent postpartum metritis as a result of their positive effect in removing RFMs.

Estradiol cypionate has been used for treating dairy cows affected with metritis or endometritis but has not been approved for this use (Upham, 1996). The protocol for the extra label use of estradiol cypionate is 4 mg IM to cows that have postpartum reproductive problems during the first 10 to 25 days after calving. The treatment is based on the assumption that cows under the influence of estrogens are more resistant to uterine infection. This assumption is based on the observation that cows that cycle regularly after parturition are less likely to develop postpartum metritis than those that do not cycle and that administering estradiol promotes neutrophil phagocytosis in the uterus (Gilbert, 1992). Furthermore, it has been suggested by various clinical reports that estradiol enhances uterine resistance to infection by increasing uterine motility as well as mucus production and flow, which collectively promote the evacuation of purulent material from the uterus and may enhance uterine involution (Carson *et al.*, 1988, Noakes, 1989).

The use of estrogens for preventing postpartum metritis is not without controversy. The value and benefit of estradiol cypionate treatment in cows suffering from postpartum complications have not been substantiated by objective research. Furthermore, it has been reported that the use of estrogens during the postpartum period may be contraindicated. The postpartum use of estrogens has been associated with severe infection of the oviducts and increased incidence of cystic ovarian degeneration (Roberts, 1986). The lack of scientific evidence on the efficacy of this hormone coupled with the potential for public concern with the spurious use of hormones in lactating dairy cows are compelling reasons to conduct controlled clinical trials on using estradiol to treat postpartum metritis.

Oxytocin is very cheap, but is believed to be relatively ineffective as an aid for cleaning the uterus in postpartum cows. It was long assumed that there is a loss of oxytocin receptors in the myometrium after childbirth and that oxytocin is not effective until 48 hours postpartum. Another problem with oxytocin is the short duration of response. When administered to 25 IU IV cows with 1 -4 days postpartum the increase in contractility was for 2 hours and fell to 1.5 hours on day 5 (Frazer, 2001) . Probably a slow and continuous IV infusion of 100 IU of oxytocin in saline solution for 6 hours could be more appropriate. But this scheme is impractical in most situations of large animals (Gilbert and Schwark, 1992). Many of gynecologist have used oxytocin after a dystocia or caesarean section to treat uterine atony, to help expel the placenta and prevent uterine Prolapse. Most preparations contain 20 IU (USP) per ml and the recommendation for handling in obstetrics in cattle is typically 50-100 IU IV, IM or SC. In oxytocin we often use the philosophy of "more is better" and use doses as high as 200 IU. We now know that blood levels of oxytocin induced by the calf to the foot are lower than those induced with a treatment of 10 IU (Frazer, 2005), but to date little has been done to determine the most appropriate dose to induce sustained uterine contractions.



Several other postpartum conditions can reduce fertility. Cervicitis and vaginitis often follow a delayed or complicated delivery. Metritis may cause abscesses in the uterus; if it spreads to the Fallopian tubes it may lead to salpingitis. Scars in the uterus and adhesions between parts of the reproductive tract can result in infertility or sterility. Routine examination of cows 1 or 2 months after delivery can diagnose such conditions early (FAO, 1989).

Irrespective of the condition, treatment should also aim at restoring the animal's normal hormonal status. Thus a persistent corpus luteum must be enucleated or lysed. Inactive ovaries should be stimulated using small doses of oestradiol benzoate (2-5 mg IM) or diethyl stilboesterol (20 mg IM. or orally). Cows should be given a period of sexual rest of 2-3 cycles after treatment (FAO, 1989).

#### **2.2.5 Retained After Birth:**

The fetal membranes or what is called “placenta” is the name given to the membranes that transfer nutrients from the dam to the fetus before birth. These membranes and blood vessels are made by the fetus and connect to the blood supply in the uterus. Across the thin connection between the membranes of the dam and the membranes of the fetus, the essential materials pass to the developing fetus. When the fetus is born, the placenta normally detaches within short time and is expelled. That is why it is referred to as afterbirth (Ball and Peters, 2004).

Retention of fetal membranes, or retained placenta (RP), usually is defined as the failure to expel the fetal membranes within 12 to 24 hrs after calving in bovines. Normally, expulsion occurs within 3–8 hr after calf delivery. The incidence in healthy dairy cows is 4–16.1%, (Mohamed and Amer, 2009). The incidence is increased by abortion (particularly with brucellosis or mycotic abortion), dystocia, twin birth, stillbirth, hypocalcaemia, high environmental temperature, advancing age of the cow, premature birth or induction of parturition, placentitis, and nutritional disturbances. Cows with retained fetal membranes are at increased risk of metritis, displaced abomasum, and mastitis (Merck, 2012).

The placenta, which is of fetal origin, has over 100 highly vascular spots called cotyledons which interconnect with similar highly vascular spots on the uterus called caruncles. Retained fetal membranes occur when the cotyledons fail to separate from the caruncles (Brendan, 2012).

Retention of fetal membranes is mediated by impaired migration of neutrophils to the placental interface in the periparturient period. The impaired neutrophil function extends into the postpartum period and probably mediates the recognized complications of retained fetal membranes. Cows with retained fetal membranes have increased cortisol concentration in late pregnancy. They may also have an altered prostaglandin (PG) E<sub>2</sub>:PGF<sub>2</sub> ratio. Uterine contractility is increased in affected cows. (Placental detachment, rather than uterine motility, is responsible for retention of fetal membranes (Merck, 2012).

A retained placenta usually causes the cow to delay the next pregnancy for 2-6 months, late calving date in the following year and may result in an open cow next year (Borel *et al.*, 2006).

### **Clinical Signs**

Fetal membranes are usually obviously hanging from the vulva, Transient reduction in appetite and milk yield, Bad-smelling discharge from the vulva. Signs of secondary or systemic infection, including pyrexia, inappetance, depression etc (Brendan, 2012).

### **Diagnosis**

Diagnosis is usually straightforward as degenerating, discolored, ultimately fetid membranes are seen hanging from the vulva >24 hr after parturition. Occasionally, the retained membranes may remain within the uterus and not be readily apparent, in which case their presence may be signalled by a foul-smelling discharge. In most cases, there are no signs of systemic illness. When systemic signs are observed, they are related to toxemia. Uncomplicated

retention of fetal membranes is unsightly and inconvenient for animal handlers and milkers but generally not directly harmful to the cow. However, cows with retained fetal membranes are at increased risk of developing metritis, ketosis, mastitis, and even abortion in a subsequent pregnancy (Brendan, 2012).

### **Treatment**

The membranes usually spontaneously pass within 4-10 days as the caruncles undergo necrosis, without the need for treatment. If there are signs of systemic illness, intrauterine oxytetracycline may help to reduce metritis. Intravenous calcium may help if the retention is due to hypocalcaemia. Prognosis is good if no metritis develops, and there appears to be minimal effect on fertility. If foetal membranes are retained for more than 12 hours, metritis is more likely to develop (Brendan, 2012).

Manual removal of the retained membranes is not recommended and is potentially harmful. Trimming of excess tissue that is objectionable to animal handlers and contributes to gross contamination of the genital tract is permissible. Untreated cows expel the membranes in 2–11 days. Routine use of intrauterine antimicrobials has not been found to be beneficial and may be detrimental. Although advocated at various times, oxytocin, estradiol, PGF<sub>2</sub> $\alpha$ , and oral calcium preparations have not been shown to hasten expulsion of retained membranes or to prevent complications. When systemic signs of illness are present, systemic treatment with antimicrobials is indicated. In herds in which incidence of retained fetal membranes is unacceptably high, predisposing causes should be sought and eliminated. Supplementation with vitamin E and selenium for herds in which these nutrients are deficient has been found to be beneficial (Merk, 2012).

Proper animal husbandry can reduce the incidence of afterbirth retention. Animals should be sexually rested for at least 2 months after calving, fed a balanced ration, adequately exercised where they are continuously raised

indoors and immunised against prevalent infectious diseases that cause abortion. Animals should not be unduly stressed and proper sanitation and management must be exercised at delivery; selenium should be added to feed where it is deficient (Youngquist and Bierschwal, 1997).

### **2.2.6 Embryonic Mortality**

Embryonic mortality (EM) in cattle has been defined as the death of in-utero conceptus before day 42 post fertilization. Its occurrence varies between and within species and breeds. Nutritional and endogenous stress may also be contributory factors. Environmental toxicants like pesticides, weedicides and fungicides may also affect embryonic mortality. In cattle at first service the EM may range from 40% usually 20-30% EM is observed in herd. In herd with high repeat breeding. The incidence of EM may be as high as 65%. The EM may be caused by chromosomal abnormalities, dysfunction of corpus luteum, defective implantation and diseases of uterus or the dam in general (Bilodeau–Goeseels and Kastelic, 2003).

First few weeks are critical periods of embryonic survival. Embryonic mortality is most common during IST fortnight of gestation (Wani, 2005). The abnormal implantation, maternal recognition of pregnancy, formation of placenta and cardiovascular system cause mortality during this period (Cross, 2001). At molecular level these defects have been found either in trophoblast (mesenchymal or vascular components) or in placental or cardio-vascular functions of early conceptus. The functions of the placenta and cardiovascular system are interlinked. The placental hormones have relation with maternal and foetal cardiac and vascular function. Some genes essential for the development of these primary organs or systems may be interlinked. Their understanding is of prime importance for understanding early embryonic life (Cross, 2001).

Early embryonic loss in cattle is difficult to study because no sensitive test similar to that used for women and mares exists. The fertilization rate after AI in

beef cows is 90%, whereas embryonic survival rate is 93% by Day 8 and only 56% by Day 12 post AI (Diskin and Sreenan, 1980). In dairy cattle, only 48% of embryos were classified as normal on Day 7 after AI (Weibold, 1988). Thus, substantial pregnancy loss probably occurs within two weeks post AI.

Rectal palpation from 40 to 60 days post AI is the most common method of pregnancy diagnosis in dairy cattle. Several studies have used pregnancy diagnosis based on rectal examination to establish a conception rate from which pregnancy loss can be determined as gestation ensues. Using this technique, pregnancy loss is about 10%, with greater losses in lactating cows compared with heifers (Thurmond *et al.*, 1990, Markusfel-Nir, 1997). Furthermore, the risk of pregnancy loss was more than four times greater during the first compared with the second and third trimesters of gestation (Markusfel-Nir, 1997).

Transrectal ultrasonography was used to determine the timing of pregnancy loss from 28 days post AI to calving in lactating dairy cows (Vasconcelos *et al.*, 1997).

Because fertility assessed at any point during pregnancy is a function of both conception rate and pregnancy loss, factors associated with pregnancy loss may be similar to those responsible for low fertility. Nutrition can have a major impact on dairy cow fertility. The review of (Ferguson, 1996) indicated that nutritional causes of low fertility are more like first due to energy management, second to excessive protein feeding, and third to trace element and vitamin deficiencies. In addition, greater body conditions score losses from calving to breeding result in reduced fertility (Ferguson, 1996).

Specific physiologic mechanisms responsible for pregnancy loss in lactating dairy cows are unknown, but may include lactational stress associated with increased milk production (Oltenucu *et al.*, 1980; Nebel and McGilliard, 1993), negative energy balance, toxic effects of urea and nitrogen (Butler *et al.*, 1995) or reduced ability to respond to increased environmental temperature (Stevenson *et al.*, 1984, Hansen *et al.*, 1992). Beef cows losing weight have a higher incidence of

early embryonic loss than those gaining weight (Dunn and Moss, 1992). This suggests that negative energy balance may be involved when a high incidence of early embryonic loss is observed in dairy cows. Recommendations for minimizing the severity of negative energy balance in high-producing dairy cows include maximizing dry matter intake in early lactation. High circulating urea and ammonia from the feeding of diets high in degraded intake protein (DIP) may adversely affect early embryonic development (Butler, 1998). Further, the feeding of excess DIP may exacerbate negative energy balance and related reproductive problems. Elrod *et al.*, (1993) reported increased early embryonic loss for heifers fed an energy-restricted diet containing high levels of DIP. Ferguson (1996) indicated that cows fed high amounts of DIP showed more irregular intervals between first and second service. Dietary DIP should be restricted to 10%-12% (NRC, 1989). Also, nitrate concentrations in water and forages should be evaluated when herds are experiencing a high incidence of abortions or early embryonic loss (Davison *et al.*, 1965).

### **2.2.7 Abortion**

Abortion is defined as fetal death and expulsion between 42 (an estimated time of attachment) and 260 days (the age at which a fetus is capable of surviving outside the uterus) of gestation. The condition does not include fetal maceration and mummification (Augustine, 2000).

#### **Incidence**

Many abortions go unnoticed or undiagnosed and we always suspect fertility problems when cows are found open rather than embryo/fetal loss. The greatest risk of fetal loss is during the first trimester of gestation and then progressively decreases as gestation advances with a slight increase in the risk toward the last month of gestation (Thurmond *et al.*, 1990). The observed fetal loss is far less than the actual incidence. The cumulative incidence of fetal loss between 31 and 260 days of gestation is 10.8%. Of this only 20% of the fetal losses are detected by

observation of an expelled fetus or fetal membranes and the proportion detected increases with increasing gestational age at time of fetal loss (Forar *et al.*, 1996). The report of (Kinsel, 1999) identified a lesser incidence of 2.9% abortion (3012 lactations out of 103,396) and a detection rate of 45.8% (1380 out of 3012). It is also believed that the abortion rate increases after 5 pregnancies or after 4 calving and furthermore the risk is higher for a cow that already had an abortion.

### **Causes**

Either infectious or non-infectious agents may cause abortion. The infectious causes include bacterial, mycotic, viral, and protozoal. Historically, it has been suggested that 50-65%, 20-25%, and 15-25% of infectious abortions were caused by bacterial, fungal, and viral causes respectively. Based on diagnostic samples submitted to a laboratory in western United States, 45% are attributable to bacterial causes, 31% to mycotic causes, and 15% to viral causes (Bhart, 2004) The non-infectious causes include nutritional factors, chemicals, drugs, toxins, poisonous plants, and hormonal agents. In addition, epizootic bovine abortion is caused by an unknown agent in the foothills of California, Nevada and Oregon, and may be in other parts of the western United States. Under modern systems of dairy management cows continue to experience a seemingly high risk of abortion, despite intensive efforts toward immunization against infective agents. It should be remembered that because of the endemic nature of abortion occurring in vaccinated cattle, factors other than the infectious agents might contribute to the risk of abortion (Bhart, 2004).

### **Diagnosis:**

Only 30% of abortions are currently being diagnosed. Although infectious agents have been incriminated in 20-30% of abortion cases submitted to diagnostic laboratories, their role may even less important if the presence of organisms does not necessarily indicate a causal association with abortion.

The causes of most abortions in dairy cows remain obscure, even though endemic rates may reach or exceed 10%. Other evidence suggesting dubious role for certain infectious agents in abortion is that vaccination and diagnostic efforts have had marginal success in solving endemic abortion problems. Constraints to diagnosis arise in interpreting the results of tests because most abortions are not detected for several weeks or months, by which time appropriate tissues may be unobtainable. Moreover, tissues submitted are usually from fetuses large enough to be noticed by dairy personnel (Augustine, 2000). Potential under-representation of young fetuses by diagnostic follow-up presents a biased view of causes of abortion – the putative causes relating to whatever was found in tissues from older fetuses.

Determining the cause of bovine abortions presents many difficulties. This is evident from the diagnostic success rate of 30 to 40% attained by most diagnostic laboratories around the world and rarely it reaches 50%. One reason for the low diagnostic success rate is that abortion is frequently the result of an event that occurred weeks or months earlier, and the cause of the event, if it was ever present is the conceptus, is often undetectable by the time of abortion (Kirkbride, 1991).

#### **2.2.8 Stillbirth parturition**

Stillbirth parturition is defined as calves that are either born dead, and or die within 48 h after calving (Berry *et al.*, 2007, Bicalho *et al.*, 2007, Gundelach *et al.*, 2009). The incidence of stillbirth parturition in dairy cows seems to have increased in recent years (Meyer *et al.*, 2001; Bicalho *et al.*, 2007 and Hansen *et al.*, 2011). Stillbirth parturition does constitute considerable financial losses to the dairy farmer in different ways. This disorder is associated with increased risk of developing metritis and retained placenta and has a considerable negative effect on lactation performance, conception rate and longevity.

Stillbirth parturition reduces the number of calves for sale and replacement (Correa *et al.*, 1990; Emanuelson *et al.*, 1993; Maizon *et al.*, 2004).



Identification of the risk factors associated with stillbirth can aid in optimizing herd reproductive efficiency. Also, the estimation of the effects of the disease on lactation performance, fertility and survival has great importance to assess the cost-benefits of diagnosis, treatments and prevention efforts.

### **2.2.9 Nutritional Causes:**

Many studies show the influence of nutrition on cattle fertility. Differences in nutrition probably account for most variation in reproductive performance between herds and among animals within herds (Wiltbank *et al.*, 1964; McDowell, 1972; Holness *et al.*, 1978). Level of feeding (Wiltbank *et al.*, 1962) and bodyweight (Ward, 1968, McClure, 1968) affect cow fertility.

The effects of underfeeding are greatest on pre-pubertal animals and lactating cows. Weight loss postpartum, due to underfeeding or high lactation demands, extends the postpartum anoestrous period (Entwistle, 1983). Underfeeding also reduces milk yield, which reduces the growth of the calf. This reduces calf weaning weight and delays puberty, which reduces the potential lifetime productivity of the female calf (Lamond, 1970).

The effects of poor nutrition differ depending on whether the main deficiency is in energy, protein, vitamins, minerals or trace elements. Under traditional management, usually more than one component is deficient (Roberts, 1971).

The effects of nutrition on cattle reproduction are covered extensively here because most cattle in the tropics are poorly fed and improving their feeding can immediately increase their reproductive performance. For more detail, consult (Cunha *et al.*, 1967; McClure, 1968; Lamond, 1970; Preston and Willis, 1974 and Topps 1977).

The nutritional status of animals is difficult to measure, and this complicates interpretation of nutrition x reproduction interactions (Haresign, 1984). An animal's nutritional status is usually assessed on changes in its liveweight and body

condition. However, these are long-term changes while many of the events of reproduction, e.g. ovulation, fertilization and placentation, take only a short time.

The reproductive performance of the postpartum cow is related to nutritional status (Dunn *et al.*, 1969, Van Niekerk, 1982). Cows fed a high energy diet after calving conceive sooner than those with a lower energy intake (Wiltbank *et al.*, 1962, Hill *et al.*, 1970). Although protein is generally regarded as less important than energy for reproduction, low protein intake can also cause infertility. However, it may be difficult to differentiate the effects of low protein intake from concurrent low energy intake, because protein deficiency usually leads to decreased appetite.

Cattle in the tropics are usually dependent on natural pastures and crop byproducts for feed. The crude protein content of the feed is often below 7.5%, which reduces rumen efficiency and reduces the true digestibility of the feed. As a result, lactating cows are unable to meet their nutritional requirements and lose weight and condition during lactation. This prolongs the lactation anoestrous period, and cows tend to calve in alternate years (Ward, 1968). The percentage change in the cow's bodyweight during the first 2 weeks after calving is inversely related to the number of days to first ovulation (Stevenson and Britt, 1980, Butler *et al.*, 1981).

High levels of feeding before calving reduced the postpartum anoestrous period in taurine cows (Bellows and Short, 1978). In addition, more cows exhibited oestrus before the breeding season and subsequent pregnancy rates were increased. King, (1968) estimated that a 1% change in body weight resulted in a 1% change in first service conception rate. Similar results have been found in zebu cattle.

Feeding a high plane of nutrition to five anoestrous cows of each of four breeds for 45 days resulted in 65% resuming cycling and 55% ovulating, whereas the 20 cows kept on a low plane diet neither cycled nor ovulated (Dindorkar *et al.*, 1982).

Few studies have been made on the relationship between bodyweight, condition and hormone synthesis or secretion in zebu cattle, and their results are inconsistent. However, in general the results suggest that poor feeding postpartum reduces luteal function and responsiveness of the ovaries to luteinising hormone (Martinez *et al.*, 1984; Rutter and Randel, 1984, Whisnant *et al.*, 1985).

### **2.3 Economic impact of infertility:**

Bovine reproductive diseases and conditions result in economic losses caused by decreased production and delayed reproduction as well as increased treatment and preventative measurement costs (Bellows *et al.*, 1994). Reproduction can be negatively affected in many ways, Viral and bacterial diseases can cause abortions and dystocia may cause the death of calf or dam and reduce reproductive performance of the dam and subsequent body weight (BW) gain in the calf (Laster *et al.*, 1973; Bellows *et al.*, 1978; Doornbos, 1984; Patterson *et al.*, 1987; Bellows *et al.*, 1988) Retained placentas and cesarean delivery can result in a prolonged postpartum interval to conception, causing increased days open and decreased milk production (Barkema *et al.*, 1992; Van Werven *et al.*, 1992). Infertility, regardless of cause, is a major reason for culling animals. Accurate quantification of these costs is straightforward when parameters are set and data analyses are confined to a few individual operations. On the other hand, national cost estimates based on parameter coefficients from several production studies and disease experiments are difficult to calculate because research goals, study methodology, and quantification criteria differ greatly from study to study (Bellows *et al.*, 1994).

#### **2.3.1 Costs of Reproductive Diseases and Conditions**

##### **Infertility in cows:**

Infertility of (cattle) cows and heifers or the inability to become pregnant in a defined breeding period may have the single greatest effect upon reproduction costs and efficiency (Greer, 1980; Bellows and Short, 1994). Unfortunately, infertility is one of the least easily quantifiable conditions because of widely

differing management practices and environments and the many factors that may contribute to the perceived problem of infertility (Bellows *et al.*, 1994).

In dairy herds, annually infertility incidence rates ranged from 0.2% in heifers to 4.5% in cows, averaging 2.9% cases (NAHMS, 1989). The NAHMS Dairy '96 USDAA, (1996) reported an average prevalence of 116 cases per 1,000 cows. This higher rate is probably due to a broader definition of “not pregnant” 150 d after calving compared with the inability to become pregnant, the definition used in pilot studies.

Costs of infertility have been generally defined to include the cost of replacement of the animal culled and any labor, drug, or veterinary service expenses included in treatment and/or prevention. Greer *et al.*, (1980) concluded that infertility was often implicated as a leading cause of culling. In Colorado, 85% of all cows culled for reproductive problems were nonpregnant or had become pregnant late in the breeding season (USDAN, 1985). A California report stated 4.5% of the studied total cow population was culled because of infertility (Gardner *et al.*, 1986).

Early culling may delay genetic progress; however, the cost of such delays is difficult to quantify. Costs ranged from \$1.80 to \$23.60 per cow inventory in dairy operations with an average of \$15.00 per cow inventory. When extrapolated to the national dairy herd, average NAHMS reported costs of infertility amount to \$137 million per year (USDAN, 1990).

Because operation goals vary widely regarding animal type produced (e.g., purebred vs commercial) and because cattle prices are of a cyclic nature, when suggest that future economic studies should separate replacement animal costs from drug, la labor, and veterinary service costs. This separation is justified because cyclic animal prices may adversely affect cost data when combined with relatively constant drug, labor, and veterinary costs.

## **Abortions and Stillbirths**

Abortions and stillbirths negatively impact the annual cow production rate (Bellows, 1987). Economic losses of stillbirth to the dairy industry in the U.S. just due to the loss of replacement heifers were estimated to be \$125 million per year (Meyer *et al.*, 2001). It was reported that cows with stillbirth parturition were at a 41% increased hazard to die or to be culled from the herd than cows without stillbirth (Bicalho *et al.*, 2007). Also, earlier studies showed that calving associated with dead calves leads to lower lactation performance when compared with live calving (Berry *et al.*, 2007, Bicalho *et al.*, 2007). One or more factors (e.g., parity, age at first calving, calving season and twinning) may contribute to a complex contribution of events when the final outcome is a stillborn calf. Mickelson (1990) found that abortions decreased potential calf crop by 2.3% in beef cow-calf herds of 1,000 cows and stillbirths at 17 and 23 per 1,000 cows in beef and dairy cows, respectively (USDAN, 1988).

In the NAHMS Dairy '96 Study USDAA, (1996), producers estimated that 35 per 1,000 cows aborted their calves. Based on Dairy '96 USDAA, (1996) information and assuming a conservative value of a newborn calf at \$85 per head, abortion and stillbirths cost beef cow-calf operations \$1.90 per cow inventory in lost calves. Unfortunately, information on stillbirths was not collected in the Dairy '96 study. For dairy operations, abortions alone cost \$3.00 per cow inventory in lost calves. Based on these data, national costs associated with stillbirths and abortion costs are estimated at approximately \$64 million yearly for beef cow-calf producers and \$27 million per year for dairy producers.

## **Dystocia**

Of the many factors affecting calf survival, dystocia is the most important (Azzam, 1993, Bellows, 1987, Patterson *et al.*, 1987). Dystocia results in death of calves and cows, production losses in both dam and calf, and delayed reproduction rates (Bellows, 1988, Bellows, 1978, Djemali *et al.*, 1987, Doornbos, 1984).

Patterson *et al.*, (1987) reported that 45.9% of all preweaning deaths can be attributed to dystocia and Laster and Gregory (1973) reported that calves born to cows experiencing difficult births were four times as likely to die as were calves born to cows not experiencing dystocia. Among California dairies, dystocia was responsible for 6.4% of all cow deaths reported and 24% of deaths in first calf heifers (NAHMS, 1998). Tennessee (1988) reported dystocia occurrence at 28.6 per 1,000 cows per year. Averaging NAHMS pilot studies (NAHMS, 1988-1990) reveals a dystocia rate of 59 cases per 1,000 dairy cows and 31 cases per 1,000 beef cows.

In addition to death and the cost of veterinary service of and drug, dystocia often causes other, seemingly less noticeable losses. These losses include lower weaning BW among calves experiencing difficult births, higher number of days open in dams, lower conception rates, and decreased milk production in dairy cows (Bellows, 1994, Bellows, 1988, Dematawewa, 1997– Doornbos, 1984). Dairy cattle requiring cesarean section delivery because of advanced dystocia had longer dry periods (15 d) than the mean dry period of cows undergoing normal parturition (Barkema, 1992). Beef females suffering dystocia also exhibit lower conception rates. Beef cows experiencing dystocia had a 15.6% lower conception rate to artificial insemination and a 15.9% lower overall conception rate than did cows not experiencing difficult births (Laster, 1973). In addition, cows detected in estrus during the insemination period that experienced dystocia had a 6.1% lower conception rate than did cows detected in estrus that did not experience dystocia. We recognize the potential economic importance of these losses, but resulting cost values were not included in studies reviewed and could not be quantified for inclusion in this study.

In 1987 a study involving over 141,000 lactations with dystocia in dairy cattle, first-calf heifers experiencing dystocia scores of 5 (most difficult) produced 465 kg less milk and 20.7 kg less milk fat than did first-calf heifers with dystocia

scores of 1 (least difficult). Second parities with scores of 5 produced 576 kg less milk and 20.9 kg less milk fat than did age contemporaries with dystocia scores of 1, and third and greater parities produced 725 kg less milk and 25 kg less milk fat when compared with age contemporaries with dystocia scores of 1 (Djemali, 1987). In a more recent study involving almost 123,000 lactating dairy cows, dystocia reduced average milk production by 38.4 kg per cow (Dematawewa, 1997). At the national level, these totals 349 million kg lost milk production and translates into a \$12.30 per cow inventory loss Estimate by Smith *et al.*, (1985) found average dystocia costs of \$35 for first-lactation dairy heifers. In NAHMS pilot studies, dystocia often the highest reproductive health cost. In Georgia, yearly costs totaled \$5.70 per cow inventory for prevention alone in dairy cattle; the same survey found annual dystocia costs of \$2.70 per cow inventory in beef cattle (USDAN, 1988). When death loss and treatment costs are included, overall studies reviewed, dystocia is estimated to cost \$11.90 per cow inventory in dairy operations and \$5.50 per cow inventory in beef cow-calf operations. Adding the \$12.30 per cow inventory in lost milk production in dairy herds brings the total cost associated with dystocia to \$24.20 per cow inventory. This is very similar to the total cost estimate of \$24.24 per cow by Dematawewa and Berger, 1997). We estimate the annual total cost of dystocia at \$220 million per year in dairy herds and \$185 million in beef cow-calf herds.

Retained placentas can occur spontaneously or result from conditions that shorten gestation length, and their occurrence can incur added costs and production losses (Laster, 1973). Retained placentas seem more prevalent in dairy cattle than in beef cattle (Merck, 2012), but a difference in rates could be partly due to the higher visibility of dairy cows to dairy managers. For example, a beef producer may consider the placenta retained after 24 h, but a dairy producer may consider it retained after observing it for 10 h.

Dairy cattle showed an incidence rate of 24 per 1,000 cows per year (USDAN, 1988). Other literature (Greer, 1980) reports the incidence of placental retention to range between 3 to 12% following normal Parturition, however, this rate increases dramatically to 20 to 50% following abnormal births or when the reproductive tract was infected. Lech *et al.*, (1988) reported that placental retention in dairy cattle occurs in approximately 8% of normal births but increases to 30 to 50% in births 1 to 2 wk premature was and to 45 to 70% in twin births. Calculated averages from the NAHMS pilot studies (USDAN, 1988) indicate placental retention occurring at a rate of 23 per 1,000 dairy cows and 8 per 1,000 beef cows. Costs of treatment of retained placentas in Georgia herds totaled \$0.03 per cow inventory in beef operations and \$0.40 per cow inventory in dairy farms (USDAN, 1988). However, retained placentas also may cause related costs including decreased Subsequent fertility, longer postpartum intervals, and decreased milk production, as well as increased risk of metritis or pyometra (Lech, 1988). Data from a study in the Netherlands with dairy cattle showed that when placentas were retained for periods >6h, an increase of 17 d to first service and 26 additional d open resulted (Van Werven, 1992). This study also reported a reduction in 100-d milk production of 237 kg per cow for cows with placentas retained for >12 h compared with cows not experiencing retained placentas. Assuming the same milk loss for the U.S. and the 7.8% rate of retained placentas as estimated by NAHMS Dairy '96 (USDAA, 1996) we estimate average annual U.S. milk loss would be 18.5 kg per cow for a total loss of 168 million kg milk or \$54 million per year.

### **Metritis and Pyometra**

Metritis is defined as inflammation caused by mild infection of the uterus, and pyometra is severe uterine infection. The two conditions differ in pathology And treatment, but have been grouped together to facilitate more accurate estimates of incidence rate and economic impact. Metritis and pyometra can lead to greatly reduced fertility, prolong the interval to uterine involution and first



ovulation by 20 or more d (which may increase calving interval 16 to 36 d), and delay conception after ovulation by causing direct damage to the ova or sperm by bacterial or fungal toxins produced by the infection (Lech, 1988). Lech *et al.*, (1988) reported that metritis incidence rates may range from 90 to 260 cases per 1,000 cows and that pyometra may range in incidence from 20 to 60 cases per 1,000 cows in all postpartum dairy cows. In the NAHMS pilot projects, yearly metritis in dairy herds ranged from 81 cases per 1,000 cows in Ohio (USDAN, 1988) to 138 cases per 1,000 cows yearly in Georgia (USDAN,1988). Most data indicate the problem is more prevalent in dairy cattle, but, again, the authors suggest that because of operation nature, some degree of difference in incidence could be attributed to greater detection in dairy herds, which would also lead to higher per cow treatment costs.

Prevention and treatment of metritis in dairy cattle was reported to cost producers \$1.30 per cow inventory in Georgia (USDAN, 1988); Ohio dairy farmers spent an average of \$8.00 per cow inventory (USDAN, 1990). In Iowa beef operations, however, treatment costs were just \$0.04 per cow inventory USDAN, (1987). Again, inadequately standardized cost data makes it difficult to determine true national estimates, but, based on the Ohio and Georgia studies, we estimate metritis and pyometra cost approximately \$4.70 per dairy cow inventory.

### **Failure to Conceive upon First Breeding**

Cows and heifers that do not become pregnant to first service contribute largely unknown but substantial costs to both dairy and beef industries. Considering a normal 285-d gestation length, 60-d postpartum anestrous interval, and a 20-d estrous cycle, a cow must become pregnant in her first estrous cycle after the postpartum anestrous interval to maintain an annual calving interval and high productivity. If a cow does not conceive during this time, the producer incurs losses in the form of decreased weaning BW, decreased milk production, and

increased overall cost per productive unit (kilograms of calf or milk). These losses, although significant, could not be quantified in this study.

### **Total Reproductive Condition and Disease Costs.**

When summed across the different reproductive diseases and conditions, our estimated cost to beef cow-calf producers is \$14.90 per cow, and, for dairy producers, the cost estimate is \$53.20 per cow. An alternative approach is to average the total cost of reproduction conditions and diseases from individual state pilot studies. The advantage of this approach is that it allows more state estimates to be used as some states reported totals only.

For dairy cows, incidence rates of reproductive conditions and disease occurrence ranged from a low of 205 per 1,000 cows in Ohio to a high of 574 per 1,000 cows in Michigan and averaged 350 per 1,000 cows across four states: California (USDAN, 1988, USDAN, 1990), Michigan (USDAN, 1988, USDAN, 1990), Ohio ( USDAN, 1990), and Tennessee (USDAN, 1988). Estimates of total cost for dairy herds ranged from a low of \$28.20 per cow inventory in Tennessee to a high of \$62.80 per cow inventory in Ohio, with a four-state average cost of \$52.00 per cow inventory. With reproductive diseases and conditions estimated to annually cost beef cow-calf producers \$13.10 to \$14.90 per cow inventory, aggregate industry losses to reproductive diseases would range from \$441 to \$502 million annually. For dairy producers, annual costs of reproductive conditions and diseases range from \$52.00 to \$53.20 per cow inventory.

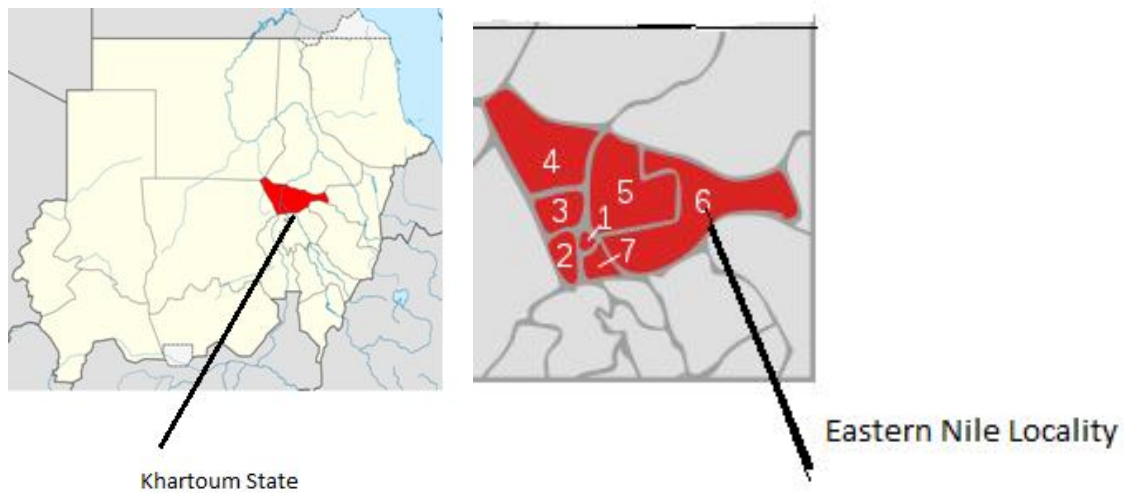
Total annual dairy industry losses associated with reproductive diseases and conditions would range from \$473 to \$484 million, reproductive failure accounts for 20-25% of the reason dairy cows are marketed for beef. Abortions may contribute to this figure, yet on average, less than 3% of cows abort each year (Harris *et al.*, 2001).

## CHAPTER THREE

### MATERIALS AND METHODS

#### 3.1. The study Area

The study was conducted in Eastern Nile Locality (ENL), Khartoum State, Republic of Sudan. The locality is located between longitude 15 37° and 15 78° North and latitude 32 32° to 32 71° East. The area represents a typical semi-arid zone with substantial variation in temperature and humidity. In dry summer months between (March-June), average monthly temperature ranges between 23.0-40.5 C°, while in wet summer (July-October) the average monthly temperature ranges between 24.5-38.7C° with average rain fall reaching 26,08 mm. In winter (November-February) the temperature ranges between 17.0 - 33.8C°. The Humidity varies with season; it reaches an average of 23 % in winter and 22%, 45% in dry summer and autumn respectively.



Source: [http://openi.nlm.nih.gov/detailedresult.php?img=2704173\\_1472-6831-9-15-1&req=4](http://openi.nlm.nih.gov/detailedresult.php?img=2704173_1472-6831-9-15-1&req=4)

The cattle in the locality are kept in farms, each of these farms had shades and fences made of local materials such as haseer, baboons iron poles were used

for construction of animals' houses. Pens were divided according to the different production groups such as milking, dry pregnant cows, replacement heifers and calves. Feeds and water troughs were available in every pen. Hand milking and natural mating were used in all the farms under the study. (Some farmers use artificial insemination).

Animals were generally fed sorghum grass (Abu-70), alfalfa, sorghum straws, groundnut residues, molasses and bagas. Concentrate given depends on the level of milk production and usually ranges between 2-6kg twice daily. Mineral blocks were allowed freely for licking.

### **3.2. Methods of data collection**

**3.2.1. Sources of data:** primary data as well as secondary were used in the study.

**3.2.2. The primary data:** was collected by conducting a survey during two years (2008-2010). To collect data phase we needed to test the hypotheses set. The survey was conducted in two phases: Phase One: In this phase small holder's dairy farms that keep crossbred cows in the study area were surveyed. Phase Two: involved slaughter house studies aiming at determining reasons behind the slaughter of crossbred cows in ENL.

**3.2.2.1. Phase one:** In this phase a across section survey of the small holders dairy farms that keep crossbred cows was conducted. The different types of infertility problems and their prevalence. The objective of the survey was to determine prevalence and types of infertility problems together with data needed to estimate their economic impacts of infertility on dairy production among small holder farmers in ENL. Direct interview with the farmers beside the supervisor observation were used. The interview included the case history with special emphasis on breeding, clinical signs, general observations and records.

#### **3.2.2.1.1 The selected farms.**

Twelve clusters of smallholder's dairy farms in different locations of ENL were selected depending on the consent of the farmer.

### **3.2.2.1.2 The targeted animal population**

Based on phenotypic features and direct interviews with the farmers, crossbred cows in all the selected farms were recorded. Various identification methods were used to identify the animals such as name, number and ear tag. Colours sometimes used together with descriptive feature (e.g. broken horn). The above data were either collected directly from available records or direct interview with the farmer.

### **3.2.2.1.3. Determination of crossbred cows sustaining infertility problems**

The identification of cows sustaining infertility problems was based on history, clinical signs. All animals in the selected farms were subjected to repeated clinic-gynaecological examination. The diagnosis was made on the basis of history, observations, rectal palpation and serological test.

Herds under the study were visited twice a week for clinical monitoring, observations and collection of samples. All suspected animals were kept under close observations for clinical signs and other externally clear lesions. Rectal palpation as a diagnostic method was used as described by (Hafez, 2000).

### **3.2.2.1.4. Laboratory analysis**

Blood samples were collected from milk vein once a week for a period of 3 weeks using plain Vacutainers tubes. These samples were centrifuged (at 3000g for 20 min) and sera were separated. The obtained serum was divided into two parts one for diagnosis of brucellosis and the other for determination of progesterone levels after being stored under -18 C°.

For diagnosis of brucellosis, serum samples were screened using the Rose Bengal Plate Test (RBPT). The test procedure recommended by Alton *et al.*, 1988 was followed. Briefly, 30µL of RBPT antigen and 30 µL of the tested serum were placed alongside on the plate, and then mixed thoroughly. The plate was shaken for 4 min and the degree of agglutination was recorded. The sample classified positive if any agglutination was observed and negative if no agglutination.

Progesterone level was assayed using ELISA kit; this was done according to manufacture instructions "Dialab produktion and und vertreb van chemisch-technischen produkten und laborinstrumenten Gesellschaft m.b.H.

A-2351 winer Neudorf, Austria, Iz-NO sud Hondastrasse, object M55."

#### 1- Principle:

Progesterone (antigen) in the sample competes with horseradish peroxidase-progesterone (enzyme-labeled antigen) for binding onto the limited number of anti-progesterone (antibody) sites on the micro plates (solid phase).

After incubation the bound/free separation is performed by a simple solid-phase washing. The substrate solution (H<sub>2</sub>O<sub>2</sub> /TMB) was added. After an appropriate time has elapsed for maximum color development, the enzyme reaction was stopped and the absorbances were determined. Progesterone concentration in the sample was calculated based on series of standards. The color intensity was inversely proportional to progesterone concentration of the sample.

#### 2-Proceduer:

Two wells were prepared for each of the six points of the calibration curve (CA-CE) and for each sample, one for Blank.

Samples 50µl , calibrators (CA-CE) 50µl, Conjugate 50µl for sample and calibrator were incubated at 37°C for 1 hour, the contents from each well were removed; and the wells were washed with 300µI of distilled water. The washing procedure was repeated the water was drained completely, 100µI substrate solution was add to calibrator, sample and Blank using pipette, then incubated at room temperature (20-25°C) for 15 minutes in the dark. 100µI stop solution was added to all (calibrator, sample and Blank). the absorbance was read (E) at 450nm against Blank.

The results were calculated and the concentration of samples was read.

The levels of progesterone in the collected sera were used to confirm infertility problems that resulted from hormonal imbalance. Persisting levels of progesterone

> 1ng for 28 days was diagnosed as anoestrous condition. However, levels < 1ng persisting for the same period or more was considered as conditions of inactive ovaries or silent heat. Fluctuation of progesterone levels indicates repeat breeding or early embryonic death conditions.

#### **3.2.2.1.5. Treatment of infertility problems**

Manual procedures were used for treatment of some causes such as persistent corpus luteum, follicular cyst, uterine wash, evacuation of uterine contents through rectal massage. Various drugs and hormones (oxytetracycline, penicillin, Estrumate pgf<sub>2</sub>, Fertagel, Reseptal), antiseptics (Iodol iodine, Iotagin) were also used to treat and handle various infertility cases.

#### **3.2.2.1.6. Economic impacts of infertility problems**

Economic parameters associated with tangible impacts included cost of veterinary intervention in the management of infertility problems, expected cost due to calf loss; cost of cows culled and milk loss due to infertility problems.

##### **3.2.2.1.6.1. Estimated cost of veterinary intervention**

Costs due to veterinary intervention include cost of diagnosis and treatment of infertility diseases. These included cost of veterinary drugs, reagents, diagnostic kits, wages accrued to veterinarian paid by the farmer, were used to compute the estimated cost of veterinary intervention.

##### **3.2.2.1.6. 2. Estimated cost of milk loss**

Cost of milk loss was estimated as follows:

Milk loss per day was considered as milk yield / healthy cow /day-milk yield/infertile cows/day.

Monetary value of milk lost/day = milk loss/cow/day X price of milk.

Annual milk loss per cow = milk loss/day X305.

Annual total milk loss = Annual milk loss per cow x number of infertile cows.

### **3.2.2.1.6. 3. Estimated cost of calve loss**

Estimated cost of calve loss (male or female) = number of infertile cows X estimated price of weaned calve (50% male and 50% female);  
= (number of infertile cows X price of one year old male calf + number of infertile cows X price one year female calf) /2

### **3.2.2.1.6. 4. Estimation of cost of culled cows**

Costs of culled cows were estimated as follows:

Cost of a culled cow = price of replacement heifer – price of culled cow.

### **3.2.2.1.6. 5. The total costs due to infertility.**

#### **A. The total cost /year.**

Annual cost of infertility = Annual cost of milk loss + Annual cost of calves loss + Annual cost of culled cows + Annual cost of veterinary intervention.

#### **B. Average annual cost / cow**

Total annual cost of infertility /number of infertile cows.

#### **C. Average annual cost / farm**

Total annual cost of infertility /number of farms.

### **3.2.2.2.Phase Two:**

In this phase two main representative slaughter houses in ENL were selected these were El-Shaheed Naser eldeen in El-maygoma, Haj-yousif and El-Kadaro slaughter houses (during the period when El-Shaheed Nasr eldeen slaughter houses was of work). Each abattoir was visited twice a week for eight weeks.

Animals intended for slaughter were phenotypically identified as crossbred during the ante mortem inspection.

Following slaughter, the reproductive organs were collected, grossly examined and dissected for further investigations. Lesions were determined, evaluated and measured using ruler and caliber according to Drennan and Macpherson *et al.*, (1966) and then photographed.



**3.2.3. The secondary data** either collected directly from available farms records, text books, journal articles and Internet sites.

### **3.3. Data Analysis**

SPSS program (version 10, 2010) was used to manage and analysis the collected data. Descriptive statistics was done to explore the prevalence of infertility diseases and Pearson Chi-square test was used to determine level of significance among clinical cases of cows in different dairy farms. Chosen level of significance is  $p=0.05$ .

# CHAPTER FOUR

## RESULTS

### 4.1 Small holder's dairy farms survey

The survey of small holder's dairy farms involved twelve (12) farms. The total number of cows raised in these farms were 552 cows out of which 491 (88.95%) were crossbred mainly Friesian and local breeds (Table 1).

**Table 1: Distribution of the selected Dairy Farms (n=12)**

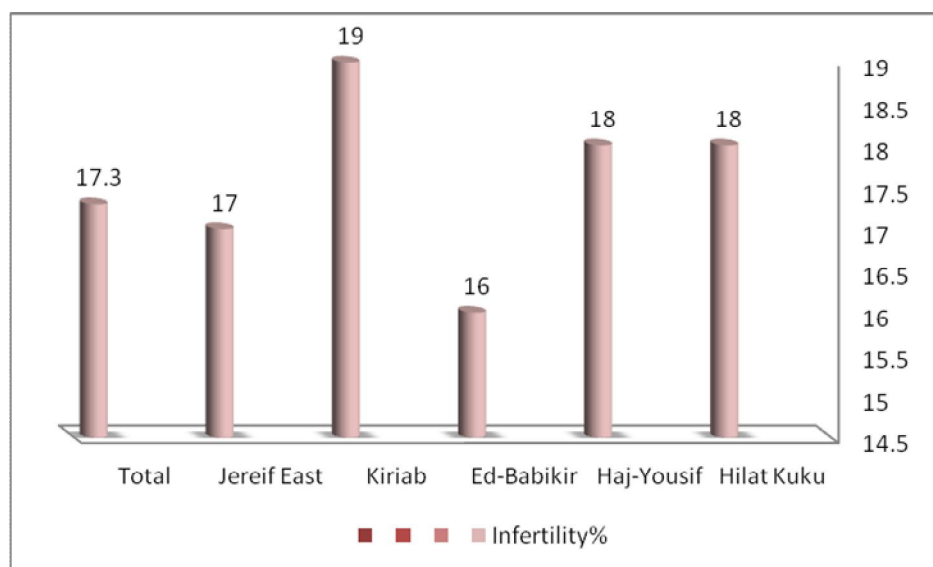
Units	No. of farms	No. of cows	Crossbred	%
Hilat Kuku	4	153	148	97
Haj-Yousif	3	150	136	91
Ed-Babikir	3	149	135	91
Kiriab	1	40	37	93
Jereif East	1	60	35	58
Total	12	552	491	89
%	100	100	88.95	-
Mean±SD	-	46±20.82	40.92±18.86	-

The mean of crossbred cows per farm is 40.92 with a standard deviation (SD) of ±18.86 which indicates that small holder farmers in Eastern Nile Locality (ENL) prefer to raise crossbred more than indigenous cows (table.1)

The distribution of crossbred cows in farms within the studied units. Of ENL, HY and ED possessed significant proportion of crossbred cows than HK, K and JE ( $p<0.05$ ) (Table 2). The high population of crossbred cows (148) and percentage (30.14) seen in HK1-4 was due number of farm included in the study but not the density of crossbred cows.

**Table 2: infertility problems encountered in ENL**

location	No. of farms	Cross bred cows	Infertile cows	Infertility%
Hilat Kuku	4	148	27	18
Haj-Yousif	3	136	24	18
Ed-Babikir	3	135	22	16
Kiriab	1	37	7	19
Jereif East	1	35	5	17
Total	12	491	85	17.3



**Fig. 1: infertility problems encountered in ENL**

#### **4.2 Infertility problems among crossbred cows in the selected farms.**

The study revealed that 85(17.31%) cows sustained reproductive problems with a mean of  $6.07 \pm 4.14$  per farm (Table 3). Cows sustaining infertility problems in ENL were significantly ( $p < 0.05$ ) higher in HY ( $8.0 \pm 2.0$ ) and ED ( $7.33 \pm 4.16$ ) compared to HK, K and JE.

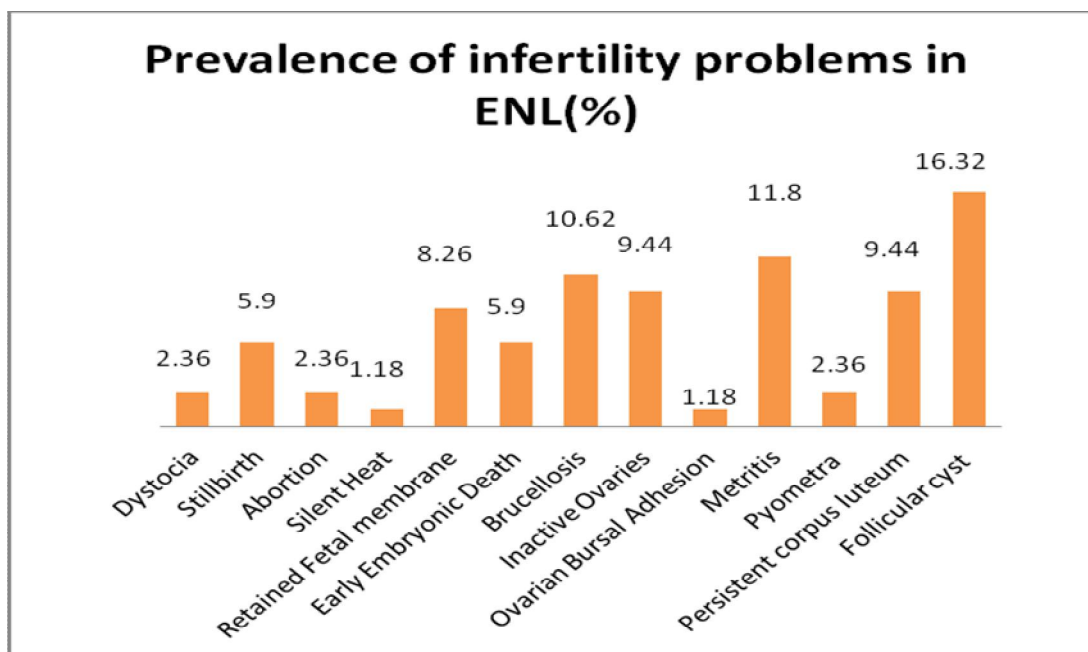
High prevalence of direct reproductive problems in all farms under the study was found to be 87.05% and the indirect reproductive problems affecting reproduction was 12.98% (Table 4).

**Table 3: Types of infertility problems diagnosed in all the farms under study (n=13)**

Type of Infertility	Type of Direct Infertility	Cows Affected	Prevalence
	Follicular cyst	14	16.32a
	Persistent corpus luteum	08	9.44b
	Pyometra	02	2.36e
	Metritis	10	11.8b
	Ovarian Bursal Adhesion	01	1.18e
	Inactive Ovaries	08	9.44b
	Brucellosis	09	10.62b
	Early Embryonic Death	05	5.90d
	Retained Fetal membrane	07	8.26c
	Silent Heat	01	1.18e
	Abortion	02	2.36e
	Stillbirth	05	5.90d
	Dystocia	02	2.36e
Direct Infertility problems		74	87.05
Indirect Infertility problems		11	12.98
Total		85	100
Mean ± SD		6.07±4.14	

a,b,c,d,e for figures within the same column bearing different superscripts differ significantly ( $p<0.05$ )

Cows sustaining follicular cysts showed significantly higher prevalence ( $p<0.05$ ) compared to other infertility problems Fig.2. On the other hand, cows sustaining inactive ovaries and brucellosis showed significant prevalence (Table 4).



**Fig 2: Types of Infertility Problems**

### **4.3. Economic Impacts due to Infertility problems in ENL**

Economic losses incurred to the smallholder dairy farmer in ENL were manifested in cost of calve loss due to prolonged calving interval, cost of cows due to culling, cost of milk loss and cost incurred from veterinary intervention.

#### **4.3.1. Cost due to calves loss**

Expected calves loss from the total affections was 85

The price of one year old male calf = SDG 650

The price of one year old female calf = SDG 900

The average price of one year old calf =  $1550/2 =$  SDG 775

Cost due to calves loss =  $85 \times 775 =$  SDG 65,875

#### **4.3.2. Loss due to culling of infertile cows**

The study revealed that there were 24 cows culled due to infertility

Price of replacement heifer = SDG 3000.

Price of culled cow = SDG 1500.

loss due to culling of infertile cows =  $24 \times 1500 =$  SDG 36000.

### 4.3.3. Loss due to reduction in milk production

The average milk loss per day for each cow was estimated at 24 lb.

Accordingly the total milk loss of 85 infertile cows was found to be 2040 lb per day.

The price of milk = SDG 1.4

The total loss in milk was estimated at SDG 2.856 per day.

The total loss due to reduction in milk production= 2856 X 300 day= SDG 856,800 / year.

### 4.3.4. The cost of veterinary intervention

The cost of veterinary intervention consists of the cost of diagnosis and the cost of the treatment. Table 4 presents the cost of veterinary intervention during the study period.

**Table 4: Total cost of veterinary intervention in the management of infertility problems**

Parameter	Diagnostic Cost(SDG)	Treatment Cost (SDG)	Total value of veterinary SDG
Total	1455	3062	4517
%	32.3	67.7	100
Mean ± SD	121.25± 85.89b	255.17±35.47a	

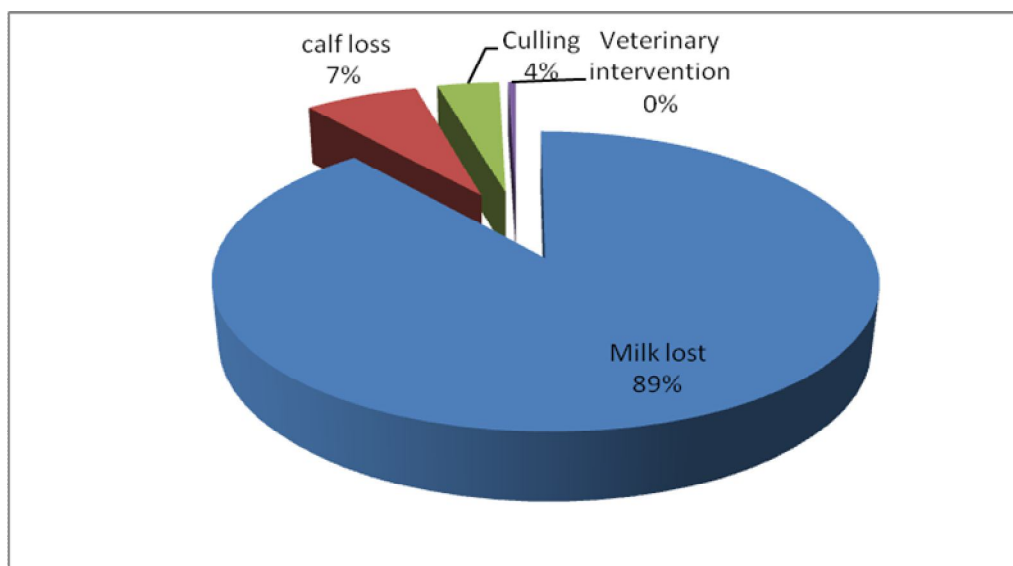
a,b For means within the same row bearing different superscripts differs significantly (p<0.05)

### 4.3.5. The overall economic impact of infertility problems

Monetary equivalent of overall economic impacts due to infertility problems in ENL was estimated at SDG 963,192 per year (Table 5;.Fig.3.).

**Table 5: The total financial losses due to infertility problems in Eastern Nile locality per year (SDG)**

Lost item	Total cost	Cost/cow	Cost/farm	%
Milk lost	856,800	33.6	71,400	88,95
calf loss	65,875	775	5,489.6	6,84
Culling	36,000	423.529	3,000	3.74
Veterinary intervention	4,517	53,141	376.4166	0.47
Total	963,192	11,331	80,266	100



**Fig.3: Total economic loss due to infertility**

#### **4. 4. Slaughter house survey**

The survey revealed that the observed slaughtered crossbred cows with direct infertility problems were 58(56.31%) compared to 45(43.69%) of indirect infertility problems. It was observed that 25 case of early stages of pregnancy passed unnoticed (Table 6).

Direct infertility problems that showed the highest prevalence at slaughter were follicular cyst (11.65%) (Photo, 1) and Metritis endometritis (8.84%) (Photo, 2) than other infertility problems encountered such as persistent corpus luteum,

pyometra (photo, 3), ovarian bursal adhesion (photo, 4), inactive ovaries (photo, 5), Para-ovarian cysts (photo, 6), Hydrosalpinx (photo, 7) and tumors (photo, 8).

**Table 6: Types of infertility problems in culled slaughtered crossbred cows (n=103)**

Infertility Problem	Number Observed	Prevalence%
Follicular cyst	12	11.65
Persistent corpus luteum	8	7.77
Pyometra	5	4.85
Metritis	9	8.84
Ovarian Bursal adhesion	4	3.88
Inactive ovaries	8	7.77
Mucometra	3	2.91
Para-ovarian cyst	2	1.94
Pyo salphinx	2	1.94
Hydrosalpinx	1	0.97
Para fallopian cyst	1	0.97
Ovarian tumors	1	0.97
Cervicitis	1	0.97
Total	58	56.31
Normal Reproductive system	45	43.69
Total	103	100



## CHAPTER FIVE

### DISCUSSION

In the present study the infertility problems in cross dairy cattle accounted to (17.31%). out of which 87.05% were reported to have a known (direct) causes. whereas the cause of 12.98% of them remained unknown causes. The failure to specify the real cause of infertility of 12.98% was attributed to difficult diagnosis of these causes by using rectal palpation.

Infertility rate in the present study comes in agreement with the result reported by (kassa and lema, 2005) who reported 17% infertility rate in Ethiopia.

Follicular cyst as a cause of infertility in the present study (16.74 %) doesn't differ largely from 18% reported by (Yusuf *et al.*, 2012) in cross dairy cattle under tropical conditions. This rate falls in the range between 8.8 to 27.4 % reported by (Tanabe and Brofee, 1982; Zemjanis, 1987) in cattle. This result also agrees with the range 5.6-18.8% in dairy cows reported by (Bartlett *et al.*, 1986) and the percentage probability range between 10 and 13 reported by (Erab, 1980; Bartlett, 1986). In the contrary, the present result is higher than the result of 10% reported by (Soonwuk *et al.*, 1996) and 11.7% prevalence reported (Kang *et al.*, 1994). The present results disagree with (Shamsuddin *et al.*, 1988) who reported 5.95%.

The high prevalence rate of follicular cyst in the present study (16.74 %) may be due to high milk yield and may also be due to high foreign blood (Friesian). Another cause for the high rate is attributed to miss diagnosis of follicular cyst which passes untreated. If the case was diagnosed the treatment may not be response for the lack or poor preservation of GnRH hormone.

On the other hand the prevalence of inactive ovary in the present study which account to 9.4% is lower than the result obtained by (Foote., 1979; Kalis, 1980; Munro, 1982; Mcleod, 1991; Opsomer, 1998).who reported results ranging between 10% to 30%. However, the present result is much lower than the result ranging from 30%-60% reported by (Munro, 1982; McLeod, 1991) and lower than 33.3% prevalence reported by Kang (1994).Whereas the present findings is higher than what reported by Shamsuddin *et al.*, (1988) who reported 5.95% rate.

The occurrence of luteal cyst in the present study (9.4%) is relatively high which may be attributed to wrong diagnosis and treatment of FC that usually ends in formation of luteal cyst or in most occasions the presence of luteal cyst is considered as pregnancy and left untreated. The present results is slightly lower than the result obtained by (Kang, 1994) who reported a rate of 11.7%, and disagree with (Yusuf *et al.*, 2011) who reported 6% prevalence rate. The present findings disagree with Shamsuddin *et al.*, (1988) and Soonwuk (1996) who reported rates of 1.17% and 24% respectively.

The prevalence of Pyometra in the present study (2.3%) is closer the 1.7% result what was reported by Hemayatul *et al.*,( 2012).However this result is lower than the 8.15% rate reported by Shamsuddin *et al.*, (1988). Metritis / endometritis was reported to be high in this study (11.76 %) which may be attributed to many factors, these include unhygienic conditions post partum, incomplete treatment after removal of the retained placenta, unhygienic AI practice, mal diagnosis and treatment of endometritis. The current rate of Metritis endometritis (11.76 %) is in agreement with Erab and Martin, (1980), Kassahun (2003) and Mamo, 2004) who reported 11.5 to 13.6 % rate. This result also agrees with Yusuf *et al.*, (2012) who reported a rate of 12% in cows under tropical conditions, and also in line with Dasharath (2013) who reported 11.21% and is closer to the result obtained by Shiferaw *et al.*, (2005) who reported 15.5% in Ethiopia. Other results were slightly higher than the present result: Gebremarim

(1996) and Oumer (2003) reported rate of 18.7%. The present result is higher than the result of 1.5% reported by Hemayatul *et al.*, (2012) and the result obtained by Zewdu (1992) in Debrezeit who reported rates of 3.1 to 9.9% respectively. However, the present result disagrees with Yoseph, (1999), Gebremarim, (1996) and Ebrahim (2003) who reported prevalence rate of 16.7%, 16.6%, 18.7% respectively.

Ovarian Bursal adhesion (OBA) reported in this study (1.17%) may attributed to hard rectal palpation done by un qualified veterinarian and technician, this result agrees with the result reported by Dobson and kamonpatana (1986) who reported prevalence of 1.8 %. Whereas it is higher than that of Rao (1981), Soonwuk *et al.*, (1996) who reported prevalence rate 0.29% and 0.6% respectively.

In the present study the prevalence rate of brucellosis was found to be (10.85%) which agrees with Cadmus *et al.*, ( 2008) who reported 9.8% .Also the present result comes is similar with the result of 8.1% reported by Chimana *et al.*, (2010) and that of Upadhyay *et al.*, (2007) who reported 12.77% in India .On the other hand the present study reported higher rate than that obtained by Chivandi (2006) in the Goke smallholder dairy project of Zimbabwe who report 4.11% prevalence rate. While it disagrees with Angara *et al.*, (2009) and Asfaw (1997) who reported a prevalence rate of 24.9% in Kuku (Sudan) and 16.7% in Ethiopia respectively. The low prevalence rate of brucellosis in this study was due to the fact that some of the surveyed farms were previously screened by Rose Bengal plate test (RBPT) and the positive reactors were culled.

The Prevalence rate of early embryonic death in this study (5.9 %) could be attributed to infection of the uterus by endometritis or may due to lake of progesterone hormone, also may be due to environmental and nutritional imbalance factors. This result is similar to the result obtained by Forar *et al.*, (1996) who reported a rate of 4.2% in Holstein dairy cows, and is closer to the

result of Paisley *et al.*, (1978) who reported a rate of 3.62%. Contrary it is much lower than the range of embryonic loss of 20% and 40 % reported by Kunz *et al.*, (2002).

The occurrence of Retained fetal membranes (RFM) in the present study was (8.2%) agrees with the results obtained by Erab and Martin, (1980). who reported 7.1% prevalence rate and also agrees with that found by Gaines (1989) who found 10% prevalence rate. Correa *et al.*, (1990) reported range between 5% and 8% which matches present result. The present result also comes in agreement with Goff (2006) who registers 7.8% prevalence rate, LeBlanc., (2008) who reported 8.6% rate, Hemayatul., *et al.*, (2012) who reported 7.1% Zewdu (1992) who reported a range of 8.1-12.5% in cattle in Debrezeit, Correa *et al.*,(1993)who reported 9.5% Han and Kim(2005) who reported range 8.3 to 28.1% prevalence rate. However the result obtained in this work is lower than the result of 14.28% reported by Mamo (2004) and that of Han and Kim (2005). Who reported 14% prevalence rate. The latter attributed the relatively high prevalence of retained fetal membrane to the bad weather condition which affect forage and silage harvest; farms management and nutrition. Although for them direct predisposal factors were brucellosis and dystocia which reported to 22.38% and 6.6% prevalence rate in the study area respectively (Han and Kim, 2005).

The occurrence rate of abortion in this study (2.35%) most probably due to the infection with *Brucella*. the present result similar to that obtained by Berisha (1990) who reported 2.2% abortion prevalence rate around Addis Ababa, also this result agrees with Swai *et al.*,(2007) who reported 3.03% prevalence rate and it comes inline with Ebrahim (2003) who reported 3.19% rate and it also comes in line with the rang 1.5-7.8% reported by Zewdu (1992) in ILCA herd debrezeit. The present result agrees with Bekele *et al.*, (1991) who reported range 1.7-20 % in dairy cattle in central high land of Ethiopia. On the other hand the present result contrasts Shiferaw (1999) and kassahun (2003) who reported 5.33% and 6.3%

prevalence rate respectively. Whereas this result is higher than the result obtained by Hemayatul *et al.*, (2012) who reported 0.9% prevalence rate.

Abortion in Bedelle (Ethiopia) was 13,9% as reported by Takely *et al.*, (1991) and 16.3% during the year 1988-1989 at Ghibe valley these results were higher than the result obtained in the present study. The high rate at Ghibe valley was attributed to seasonal flare of trypanosomiasis in Ghibe valley most of pregnant cows abort due to fever induced by trypanosomiasis Takely *et al.*, (1991).

The prevalence of stillbirth in the present study (5.9%) comes in agreement with many author: Atashi (2011), Meyer *et al.*, (2001), Berglund *et al.*, (2003), Eriksson *et al.*, (2004 ); Hansen *et al.*, (2007), Ghavi *et al.*, (2008), Gurdelach *et al.*, (2009) and Correa *et al.*,(1993) who all reported 6% prevalence rate. However the present study disagrees with the result found by Hemayatul., *et al* (2012) who reported a result of 0.8% prevalence rate. This variation in stillbirth prevalence may be due to the difference in management practices

Dystocia in the present study is reported as 2.35%, this result comes inline with the 2.2 to 4.4% result reported by Zewdu (1992). The present result also agrees with Gebremariam (1996); Swai *et al.*, Hemayatul *et al* (2012); and Grohn *et al.*,(1990) who reported prevalence rate of 3.7, 2.4% , 1.7%, 1.6%, and 1.2% respectively. On the other hand the result obtained in this study does not differ largely from that reported by Ebrahaim (2003); Mohammed (2003); and Mamo (2004) who reported prevalence rates range between 4.3% to 5.7%. However, the present result disagrees with Tadesse (1999), Yoseph (1999) in Holleta, Correa *et al* (1990) and Melkamu (1999) who reported prevalence rate range between 7% to 7.8% prevalence rate on Holsteins Friesian cows.

It is difficult to give exact figure on rate of dystocia because it is influenced by several factors such as nutritional status, age, and parity of the breed of the sire and dam (Morro, 1986; Noakes, 1986).

The estimation of the economic impacts of infertility in this study showed that the cost of milk loss accounted to the highest percentage (88.95 %) compared to the other components. This was logical since the breed under investigation is cross breed of high milk yield. The cost of calves loss is relatively low due to low culling rates although there were many factors justify the culling e.g. brucellosis and infertile cows. Also the mal diagnosis and treatment of infertility leads to increased calves' loss. The cost of culling cows represents 3.74% of the total cost, this would have been more if the farmers adopted culling programmed of Brucella positive and infertile cows but, due to social vision of owners prefer large herd size, so the culling rate is relatively low. The cost of veterinary intervention accounted to only 0.47% of the total cost. That is because the animal fertility management receives less importance from the farmers. Most of them rarely consult gynecologist.

The total economic loss due to infertility problems in this work calculated as SDG 963,192 (US \$481.596) per year and as SDG 65,496 (US \$32.748) annual loss per cow is close to USDAN (1988) Estimates of \$28.20 per cow in Tennessee, in contrast this result contradict Richard (2002) who reported US\$136 per cow.

### **Phase two Slaughter house discussions**

In the present study the rate of total infertility (36.72%) comes in agreement with the result obtained by Tekllu (1999) who reported a rate 37.0% in Addis Ababa. The present result is close to the result found by Ashenafi, (2004 ) who both reported 33.3% rate, Moreover this result is higher than other results reported by many Authors (Abalti *et al.*, 2009; Endalew. 2001; Abdissa. 2000) who reported 31.4%, 26.3%, 23.4% prevalence rate respectively. This variation could be due to geographical variation, breed, and feed and health management.

In this study the occurrence of follicular cyst (11.65%) is in agreement with (Kruif., 1976) and (Kunbhar., 2003) who reported 10% prevalence rate. Moreover, the present result is closer to the result obtained by Vighio (1980) who reported

12.5% percentage rate. However, this result is higher than the result ranging between 1.5 to 4.5% prevalence rates for follicular cyst reported by (Nair and Raja, 1974; Elwishy, 1976 and Alam, 1984)

In the present study luteal cyst accounted to 7.77% which comes in line with (El-Sawaf and Schmidt, 1963) who reported 7.3% incidence rate and Kunbhar *et al.*, (2003) who reported 7.7% prevalence rate. On the other hand the present result is lower than the result range of 10.1% to 15.62% obtained by Averkhin and Vyatikin (1976).

Ovaro-bursal adhesion percentage in this work (3.88%) does not differ largely from the result ranging between 4.8% and 5.7% rates obtained by Perkins *et al.*, (1954) and Berhanu *et al.*, (2013) in cross breed cows in Ethiopia. The present result also agrees with (Abalti *et al.*, 2006) who reported 5.5% prevalence rate and Simenew *et al.*, (2011) who reported 2.5% prevalence rate, However, the present result contrasts Ali *et al.*, (2006) who reported 7.27% prevalence rate, Uqubeab (1986) who reported 6.9% and Arthur (1992) who reported 8% rate.

Causes of ovaro-bursal adhesions are not clear (Roberts 1986). Pregnancy complications retained placenta and endometritis may predisposed adhesions (Lewis, 1997) and also rough rectal palpation (Bondurant, 1999).

In active ovary in the present study represented 7.77% which is closer to the result of 4.6% reported by (Kunbhar *et al.*, 2003).

The prevalence rate of Paraovarian cyst in this study (1.94%) is almost similar to the result obtained by Berhanu *et al.*, (2013) who reported 1.74% in cross cows in Ethiopia. The present result also similar to the high level obtained by Rao and Rajya (1976), Alam (1981) who obtained range of 0.69% to 1.09%. The present result comes agree with the low level range of result obtained by Perkins *et al.*, (1954); Qureshi and Ahmed., (1966); Seitarids and Metaxopoluos., (1971).; Kruif., 1976 and Roinf., (1977) who reported range 1.2 to 8.3% in cattle .The present

result is closer to 0.5% result reported by Simenew *et al.*, (2011) and also closer to the result 0.4% obtained by Berihu *et al.*, (2009).

However, the present result disagrees with Selunskaya (1975), Hamana *et al.*, (1976) and Romaniuk (1976) who reported 15.8%, 14% and 13% prevalence rates respectively.

Ovarian tumor in the present study (0.78%) comes in agreement with (Martinez *et al.*, 1984) who reported 1.2% prevalence rate in Friesian and cross dairy cows.

Incidence rate of Mucometra in this result 2.91% agrees with Berihu *et al.*, (2009) who reported 2.1 % prevalence rate and Berhanu *et al.*, (2013) who reported 3.77% in cross cattle in Ethiopia. It also comes in line with Uqubea, (1986), Feyissa *et al.*, (2000). However, this result is much lower than the result obtained by Gopal, (1977) who reported 25.4% prevalence rate.

The percentage of Pyometra in this study 4.85% is in agreement with Herenda., (1986) and Ali *et al.*, (2006) who reported 5% and 6.36% rate respectively and the high range of result (1.0 to 3.4%) obtained by Zemjanis *et al.*, (1974). This result is closer to result of 2.5% found by (Fathalla *et al.*, 2001), this was due to feeding cows megestrol acetate before slaughtering as indicated by Herenda (1986). The current finding is lower than 14.8 to 35.2 % of Gopal (1977), Vighio (1980), Hussain and Munivaju (1984).

The percentage of endometritis obtained from this study (8.74%) agrees with the finding of 8.57% reported by Berhanu *et al.*, (2013). also it agrees with Feyissa and Bekana (2000) who reported a rate of 9.9% and the lower range 10.8 to 30% found by Averikhin and Vyatikin (1976) , Hamana *et al.* , (1976) ; Gopal., 1977). The result agrees with Ali *et al.*, (2006) who reported 9.9% prevalence rate and Malik *et al.*,1960;Francos.,1974; Kaikini.,1983) who reported rang of 8.76% to 11.3% prevalence rate. The present result agrees with result reported by (Uqubeab. 1986) who found 6.7% prevalence rate.



However, studies of Fivaz and Swanepol (1978), Donigiewicz (1978). Kunbhar (2003) indicated higher values (47% to 90%). The Present result is higher than 1.47% reported by Herenda (1987). The difference could possibly attribute to the difference in breed, age of animals, parity and management employed.

Hydrosalpinx rate in the present study (2.5%) is in agreement with Berhanu *et al.*, (2013) who reported a rate (2.32%) in Ethiopia, This value is lower than the result reported by Vighio (1980) in cattle who reported 39%. He also reported 68.2% prevalence rate of pyo-salpinx, whereas Pyo-salpinx in the present result is 3.75%.

In this study Cysts on oviduct wall was 0.97%, which comes in line with Donigiewicz, (1978) who reported 1.3% and with Berhanu *et al.*, (2013) who reported 1.74%. However, the present result is lower than that obtained by Kunbhar *et al.*, (2003) who reported 4.6% prevalence rate and the result of 16.5% reported by Tsumura, (1982).

The prevalence rate of Salpingitis in the present study (0.97%) is almost similar to the result of 1.3-7.8% reported by Perkins *et al* (1954). Averikhin and Vyatikin (1976) in cows. However, its disagrees with result of 24% found by Seitardis and Tsagaris (1977) in cows. Also it disagrees with the result 35.5% reported by Buchi (1978).

## Conclusions and recommendations

The study concluded that: since crossbreed cows in the study area accounted to 88, 95% which means that owners prefer to raise crossbreed cows than local cows.

- The prevalence rate of Infertility problems in ENL farms (17.31%) is relatively, So Infertility remains the major obstacle of dairy cattle producers, causes significant economic losses.
- In ENL the main causes of infertility were follicular cyst (FC) 16.32%, Metritis endometritis 11.8%, and brucellosis 10.6%.
- The cost components of infertility were milk loss, calves loss, culling and veterinary intervention respectively.
- FC remains the major causes of infertility in slaughterhouse as well as in farms.
- The suggestion grew up from the study is that, cows should be observed during pregnancy and calving, and should be examined routinely by rectal palpation post partum at first month and any infertility problems should be treated early as soon as possible.
- Rectal palpation remains the major way to diagnoses infertility but the use of ultrasonography enhances diagnosis and gives more accurate results.
- Some veterinarian and technicians in the field need more training on infertility diagnosis and treatment, and owners need more awareness about infertility.
- Finally despite of many years of infertility research, it remains poorly understood and further study and investigation deemed necessary.
- Better gynecological management helps a lot in improving the economy of the farm and increases the supply of milk.

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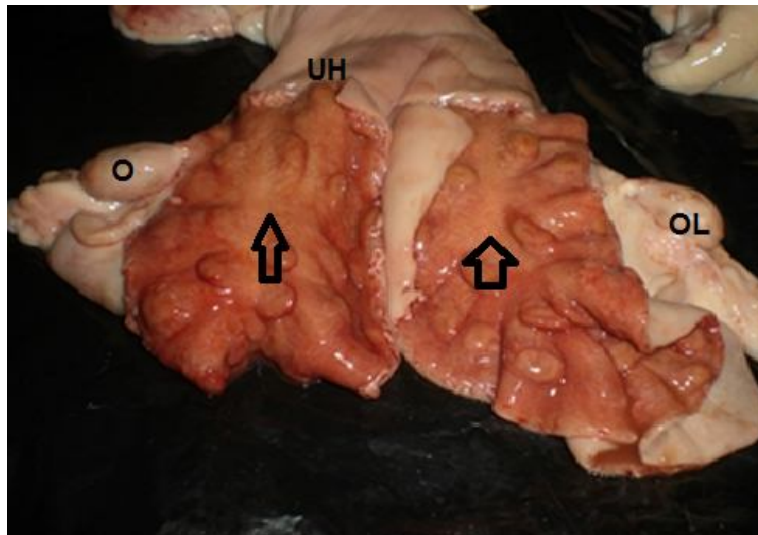
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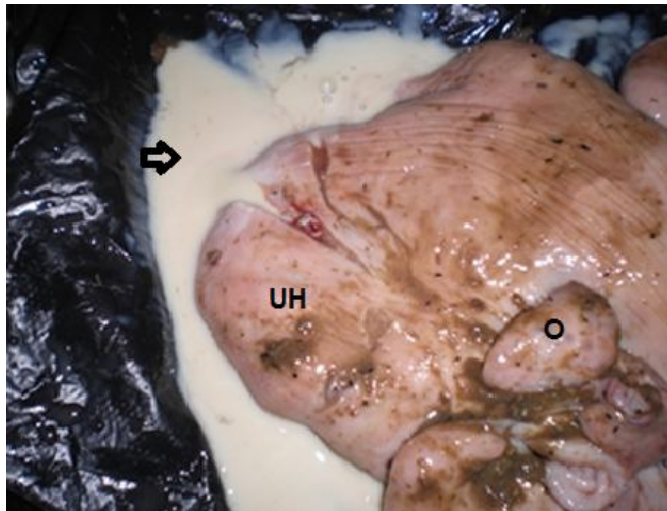
## Appendix: Photographs



**Photo 1:** luteal cyst (white arrow),  
Follicular cyst (black arrow), O, Ovary.



**Photo 2:** Metritis (arrows). Ovary (O), left ovary (OL),  
Right Uterine Horne (UH)



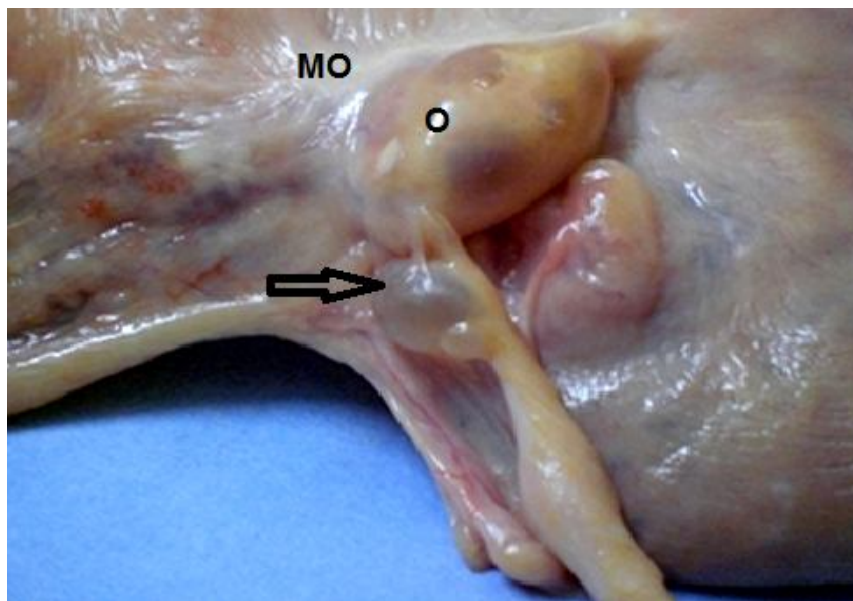
**Photo 3:** pyometra , Pus ( arrow), Ovary (O),  
Uterine Horne (UH).



**Photo 4:** Ovarian adhesions with casious material (black arrow).  
Ovary (O), uterine horn (UH)

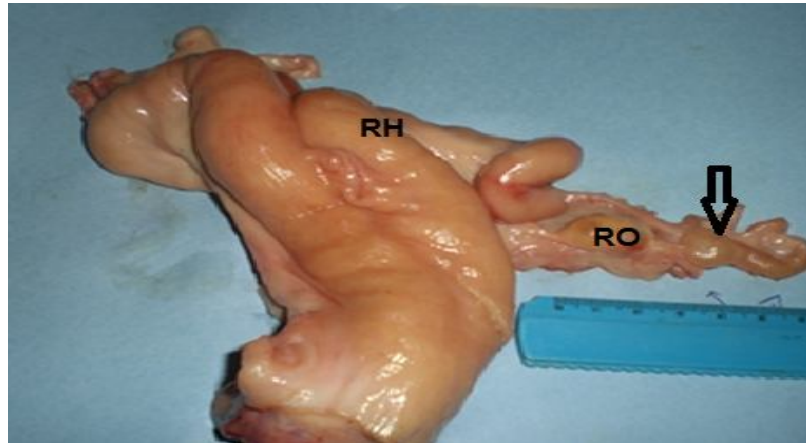


**Photo 5:** Inactive ovary: smooth ovary with out any structures (O), mesoovarian (MO)

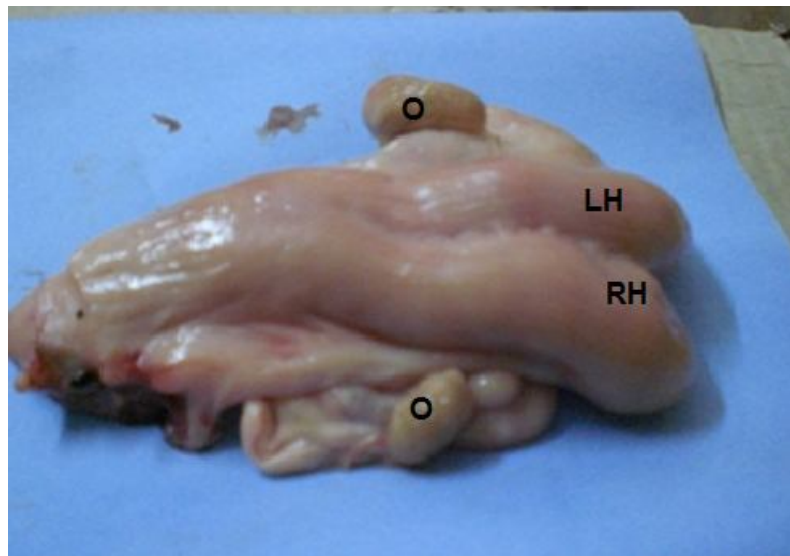


**Photo 6:** Para ovarian cyst( arrow). Ovary (O),

Mesoovarian (MO).



**Photo 7:** Hydrosalpinx (arrow), right ovary (RO),  
Right Home (RH).



**Photo 8:** Ovarian tumors. Ovary (O), Left Uterine Horn (LH),  
Right Uterine Horn (RH).