Review on Small Ruminant Brucellosis in Ethiopia

Alebachew Tilahun[1] Adane Tilahun[1] Arega Tafere[2] Tsehaye Hadush[1] Ayichew Teshale
1.Wolaita Sodo University, School of Veterinary Medicine, Ethiopia
2.Alage Agricultural Technical and Vocational Training College, Ethiopia

SUMMARY
Diseases are among the many factors which limit the economic returns from small ruminants. One of the infectious diseases which particularly impedes international trade is brucellosis. Brucellosis in small ruminants is mainly caused by *Brucella melitensis* and *B. ovis* and in sporadic cases by *B.abortus*. This disease is mainly characterized by abortion with the development of yellowish, sticky layers on the placenta in females. In male animals, it causes orchitis, epididymitis, and arthritis in both sexes. Species of *Brucella* are obligate parasites, requiring an animal host for maintenance. Sexually mature and pregnant animals are more prone to *Brucella* infection. The primary route of dissemination of *Brucella* is the placenta, fetal fluids and vaginal discharges expelled by infected animal after abortion or full-term parturition. *Brucella* species can enter mammalian hosts through skin abrasions or cuts, the conjunctiva, the respiratory tract, the gastrointestinal tract and through reproductive tracts. In active cases brucellosis of small ruminants can be diagnosed by isolation and identification of the responsible micro-organisms using bacteriological tests. *Brucella* can also be detected using molecular tests. Brucellosis is readily transmissible to humans, causing acute febrile illness, undulant fever which may progress to a more chronic form and can also produce serious complications affecting the musculoskeletal, cardiovascular, and central nervous systems. Humans get infected mainly by drinking raw milk, exposure to aborted fetus, placenta of infected animals and by an occupational risk. Brucellosis presents a significant impediment to the economic potential of the large population of small ruminants such as reproductive and productive wastage and trade ban. Small-ruminant brucellosis has been shown to occur worldwide. Since brucellosis has no effective treatment, vaccination, test and slaughter, hygiene and awareness creation are the best control and prevention strategies.
fever,
Keywords: Brucellosis creation awareness, dissemination, test and slaughter, undulant fever

1. Introduction

Goats and sheep are important domestic animals highly adaptable to broad range of environmental conditions. In tropical livestock production systems in Africa (ILRI, 2006). they account around 21% of the global small ruminant population. Small ruminants fulfill a number of economic and social functions. According to statistics from the Central Statistical Agency (CSA, 2005), Ethiopia has over 18 million head of sheep and 24 million goats. 25% of the sheep and 73% of the national goat population inhabit the lowlands (mostly pastoral areas) (PFE, 2004).

In spite of the presence of huge small ruminant population, Ethiopia fails to optimally utilize this resource as a sector. This is because of small ruminant production is constrained by the compound effect of diseases, poor feeding and management, and low genetic endowment (Ibrahim, 1998).Among those factors which limit the economic returns from small ruminants' production diseases stand in the frontline. One of such disease that hampers the productivity of small ruminants and impedes international trade is brucellosis (Ademosoum, 1994).

Brucellosis is an infectious bacterial disease caused by members of the genus *Brucella*. It is disease of worldwide importance and affects a number of animal species. Species of *Brucella* are obligate parasites, requiring an animal host for maintenance. The host range includes humans, ruminants, swine, rodents, canines and marine mammals. Infection occurs through inhalation or ingestion of organisms. High numbers of the organism are shed in urine, milk, vaginal discharge, semen and through discharges of birth of infected animals. Under appropriate conditions, *Brucella* can survive outside the host in the environment for extended periods. They may remain viable in carcass and tissues for 6 months at 0^0C up to 125 days in soil, and as long as 1 year in feces (Glenn and Karen, 2005).

Brucellosis in small ruminants is mainly caused by *Brucella melitensis (B.melitensis)* and *B. ovis* in sporadic case by *B.abortus*. This disease is mainly characterized by abortion with the development of yellowish, sticky layers on the placenta in females. In male animals, it causes orchitis and epididymitis, as well as inflammation of the joints and bursa. The consequences of brucellosis in small ruminants are: infertility, a high mortality rate in lambs and kids, mastitis, reduced milk production (Quinn *et al.*, 1999; Seifert, 1996).

The presence of small ruminant brucellosis in Ethiopia is well established (Ashenafi *et. al.,* 2007; Tekelye and Kasali, 2014; Teshale *et al.,* 2006; Yibeltal *et al.,* 2005); similar to other reports elsewhere it imposes tremendous economic loss due to reproductive wastages such as infertility, abortion, stillbirth, and the likes. On the other hand, the pastoralist communities who have daily contact with their animals and use their products in their dietary habits are being infected by brucellosis.

Therefore, the objectives of this seminar are:

- To overview small ruminant brucellosis.
- To highlight the economic and public health significance of small ruminant brucellosis.
- To show status of small ruminant brucellosis in Ethiopia.
- To highlight the control and prevention of small ruminant brucellosis.

2. Small ruminant brucellosis

This is a disease caused by infection with bacteria of the genus Brucella and it is characterised by abortion in late pregnancy and subsequent high rate of infertility. The disease is zoonotic and occupational causing undulant or Malta fever in man (Lugarno *et al.,* 1996*)*

.

2.1. Ethiology

Brucellosis in small ruminants is mainly caused by *Brucella melitensi*and *B. ovis* and in sporadic cases *B.abortus*. *B .melitensis* is most commonly infects sheep and goats. Breed susceptibility is variable in sheep, but goat breeds are highly susceptible. *B. ovis* primarily affects rams (Glenn and Karen, 2005).

2.2. Epidemiology

2.2.1. Geographic distribution

Small-ruminant brucellosis has been shown to occur worldwide and is principally found in: Mediterranean countries**,** Middle East**,** Africa**,** India**,** China**,** Mexico and Parts of Latin America (Smith and Sherman, 1994). Infection in sheep appears to occur endemically in the Mediterranean region, especially along its northern and eastern shores, stretching through Central Asia as far south as the Arabian Peninsula and as far east as Mongolia. Parts of Latin America are also seriously affected, especially Mexico, Peru and northern Argentina. The disease also occurs in Africa and India. However, North America (except Mexico) is believed to be free, as are Northern Europe (except for sporadic incursions from the south), Southeast Asia, Australia and New Zealand (FAO, 2010).

2.2.2. Host risk factors

Age factor**:**

The prevalence of the disease is most frequently occurred in adult sheep and goats than younger one (Walker, 1999). Sexually mature and pregnant animals are more prone to Brucella infection and brucellosis than sexually immature animals of either sex (Quinn *et al.,* 1999; Radostits *et al.*, 2000). Brucellosis of small ruminants affects sexually matured animals; the predilection sites being the reproductive tracts of the males and females, especially the pregnant uterus. This may result from the fact that sex hormones and erythritol, which stimulate the growth and multiplication of *Brucella* organisms, tend to increase in concentration with age and sexual maturity (Radostits *et al.,* 2000).

Species and breed factor:

Goats are at higher risk of acquiring *Brucella* infection than sheep. This may be due to the greater susceptibility of goats to *Brucella* infection. It could also be partly due to the fact that goats excrete the organism for a long period of time, unlike sheep. This reduces the potential for diseases spread among sheep flocks (Radostits *et al*., 2000).The receptivity of ewes to *B. melitens* varies according to the breed. Milk producing ewes are more receptive than sheep raised for slaughter (Corbel and Brinley-Morgan, 1984).

Sex*:*

Male animals are less susceptible to Brucella infection than females, due to presence of low concentration of erythritol in male relative to female animals (Hirsh and Zee, 1999).

Environmental factor:

Brucella may retain infectivity for several months in water, aborted fetuses and fetal membranes, feces and liquid manure, wool, hay, on buildings, equipment and clothes. *Brucella* is also able to withstand drying particularly in the presence of extraneous organic material and will remain viable in dust and soil. *Brucella* is fairly sensitive to ionizing radiation and is readily killed by normal sterilizing doses of gamma-rays under conditions which ensure complete exposure, especially in colostrums (Glenn and Karen, 2005).

2.3. Transmission

Generally, transmission of small ruminant brucellosis occurs in the same way in sheep and goats as in cattle, materials excreted from the female genital tract forming the main supply of organisms for transmission to other animals and man. Therefore, in most circumstances, the primary route of dissemination of *Brucella* is the placenta, fetal fluids and vaginal discharges expelled by infected ewes after abortion or full-term parturition. Very large numbers of organisms are shed at the time of parturition or abortion. In goats, excretion of the organisms from the vagina is prolonged and copious (2 to 3 months generally). In sheep excretion is generally less prolonged, usually ceasing within 3 weeks after abortion or full-term parturition. Shedding of *Brucella* is also common in udder secretions and semen, and *Brucella* may be isolated from various tissues, such as lymph nodes from the head and those associated with reproduction, and sometimes from arthritic lesions (Alton *et al.,* 1988).

Since *Brucella* species are intracellular pathogens of the animal hosts, the hosts are the reservoirs and can be the source of infection. Organisms reside inside cells of reticulo-endothelial system and reproductive tract and cause life long, chronic infections. Indeed, excretion of *Brucella* species only occurs at certain times, mainly when abortion occurs. During an abortion, billions of *Brucella* species are excreted and this is a major source of infection for congeners and for professionals in contact with aborted materials. Survival time of the organism outside the host is variable and depends on temperature and moisture. Colder weather extends survival time. Ingestion is the most common route of entry, although, exposure through the conjunctival and genital mucosa, skin and respiratory routes occurs (Dwight and Yuan, 1999). The infection is commonly transmitted from one ram to the other by perpetual contact. Transmission may also occur through the ewe when an infected ram deposits his semen and another ram mates her shortly thereafter. The infection is not very common in ewes, and when it occurs it is contracted by sexual contact *B. ovis* does not persist very long in ewes and is generally eliminated before the next lambing period (PAHO, 2001). Only a small proportion of lambs and kids are infected *in vitro* and the majority of *B. melitensis* latent infections are probably acquired through colostrums or milk (Grillo *et al.*, 1997).

2.4. Pathogenesis

Brucella melitensis can enter mammalian hosts through skin abrasions or cuts, the conjunctiva, the respiratory tract, the gastrointestinal tract and through reproductive tracts. In the alimentary tract the epithelium covering the ilealpeyer's patches are preferred site for entry. In the gastrointestinal tract, the organisms are phagocytosed by lymphoepithelial cells of gut-associated lymphoid tissue, from which they gain access to the sub-mucosa and localized to the reticulo-endothelial system and genital (Ackermann *et al.,* 1988).

Organisms are rapidly ingested by polymorphonuclear leukocytes, which generally fail to kill them and are also phagocytosed by macrophages. In macrophages, *B. melitensis* inhibits fusion of phagosome and lysosome and replicate within compartments that contain components of endoplasmic reticulum via a process facilitated by the type IV secretion system. If unchecked by macrophage bactericidal mechanisms, the bacteria destroy their host cells and infect additional cells (Pizarro *et al.,* 1998).Histopathologically, the host cellular response may range from abscess formation to lymphocytic infiltration to granuloma formation with caseous necrosis. Serum complement effectively lyses some rough strains (i.e. those that lack O-polysaccharide side chains on their LPS), but has little effect on smooth strains (i.e. bacteria with a long O-polysaccharide side chain); *B melitensis* may be less susceptible than *B. abortus* to complement-mediate killing (Young *et al.,* 1985). These observations suggest that *Brucella*, like other facultative or obligate intra-macrophage pathogens, are primarily controlled by macrophages activated to enhanced microbicidal activity by IFN-g and other cytokines produced by immune T lymphocytes. It is likely that antibody, complement, and macrophage-activating cytokines produced by natural killer cells play supportive roles in early infection or in controlling growth of extracellular bacteria. In ruminants, *Brucella* organisms by pass the most effective host defenses by targeting embryonic and trophoblastic tissue. In cells of these tissues, the bacteria grow not only in the phagosome but also in the cytoplasm and the rough endoplasmic reticulum (Anderson *et al.,* 1986).

In the absence of effective intracellular microbicidal mechanisms, these tissues permit exuberant bacterial growth, which leads to fetal death and abortion. In ruminants, the presence in the placenta of erythritol may further enhance growth of *Brucella.* Exudates and discharges at the time of abortion may contain up to10^{10} bacteria per gram of tissue. When septic abortion occurs, the intense concentration of bacteria and aerosolization of infected body fluids during parturition often result in infection of other animals and humans (Anderson and Cheville, 1986).

2.5. Clinical Sign and Finding

The main clinical manifestations of brucellosis in sheep and goats are, as in all female ruminants, reproductive failure, abortion and birth of weak offspring. Abortion generally occurs during the last two months of pregnancy and is followed in some cases by retention of fetal membranes.In the male, localization in the testis, epididymis and accessory sex organs is common, and bacteria may be shed in the semen. This may result in acute orchitis and epididymitis and later in infertility. Arthritis is also observed occasionally in both sexes (Fensterbank, 1987).

Animals generally abort once, although reinvasion of the uterus occurs in subsequent pregnancies and organisms are shed with the membranes and fluids. Non-pregnant animals exposed to small numbers of organisms may develop self-limiting, immunizing infections or they may become latent carriers. Persistent infection of the mammary glands and supra mammary lymph nodes is common in goats with constant or intermittent shedding of the organisms in the milk in succeeding lactations, while the self-limiting nature of the disease in sheep, which is seldom accompanied by prolonged excretion of the bacteria, has been observed (Durán-Ferrer, 1998).

The inflammatory changes in the infected mammary gland reduce milk production by an estimated minimum of 10%. Orchitis and epididymitis generally lead to a chronic infection. Infected animals generally develop granulomatous inflammatory lesions which frequently are found in lymphoid tissues and organs such as reproductive organs, udder and supra mammary lymph nodes and sometimes joints and synovial membranes. This disease has no pathognomonic lesions and the changes that can be observed are necrotizing placentitis, palpable testicular alterations, necrotizing orchitis and epididymitis with subsequent granuloma, necrotizing seminal vesiculitis and prostatitis. Some aborted fetuses may have an excess of blood-stained fluids in the body cavities, with enlarged spleen and liver. Others appear normal. Infected fetal membranes show changes affecting part or all of the membrane. The necrotic cotyledons lose their blood-red appearance becoming thickened and dull-grey in color. In the chronic stage of the disease the epididymis can be increased in size up to four or fivefold (Robles *et al.;* 1998).

2.6. Diagnosis

In active case, brucellosis of small ruminants can be diagnosed by isolation and identification of the responsible micro-organisms using bacteriological tests which determine the phenotypic characteristics of the bacteria. *Brucella* can also be detected using molecular tests which take account of all the characteristics of the genome. But in chronic infection the disease is diagnosed by different immunological (allergic test) and serological tests that can be screening and confirmatory serological tests (Quinn *et al*., 1994).

2.6.1. Bacterial Isolation and Identification

Specimen collection: the most valuable specimens for bacterial culture are aborted fetal tissues (especially lung, spleen, and stomach contents), placenta, lymph nodes, post parturient uterus, vaginal discharge, semen, urine and bone marrow. All specimens must be packed separately and transported immediately to the laboratory in ice box with ice packs in leak proof containers). If the specimens are not inoculated immediately, preserve in refrigerator at 4 ^{0}C (Glenn and Karen, 2005).Direct microscopic examination from specimen: smears are made from specimens and stained by modified Ziehl – Neelsen (MZN) stain. *Brucella* appears as small, red - staining coccobacilli in clumps because of their intracellular growth. In gram staining they appear gram negative coccobacilli in clumps. Isolation of pure colony and pure culture: pure colony of bacteria can be obtained by streaking the specimens on appropriate media. Culture material may also be taken from lymph nodes, cerebrospinal fluids, and abscesses. It is recommended the cultures be repeated several times to get pure colony then pure culture (PAHO, 2001).

Identification: of the bacteria begins with colony morphology of the pure colony and culture. They are usually smooth form in the first isolate and they become rough when they are sub cultured (Dwight and Yuan, 1999). Some biochemical tests that must be performed to differentiate species of Brucella are requirements for CO_2, production of H_2S, growth in the presence of stains and agglutination by mono specific sera (Dwight and Yuan, 1999).

Molecular technique: species of *Brucella* can be identified by molecular techniques. Molecular detection of *Brucella* species can be done directly on clinical samples without previous isolation of the organism. In addition, these techniques can be used to complement results obtained from phenotypic tests (Bricker ., 2002). Polymerase Chain Reaction (PCR) and its variants, based on amplification of specific genomic sequences of the genus, species or even biotypes of *Brucella* species are the most broadly used molecular technique for brucellosis diagnosis (Bricker 2002; Xavier *et al*., 2010).

2.6.2. Serological and immunological diagnostic methods

The tests which are used to identify animals with latent infection are immunological and serological tests. These tests are derived from research done mainly on brucellosis diagnosis in cattle. To a large extent the characteristics of the different tests can be transposed to sheep and goat, except for the milk ring test, which is not an accepted test in these species because it generates too many false-positive results (OIE, 2009).

Serological tests:

Are crucial for laboratorial diagnosis of brucellosis since most of control and eradication programs of brucellosis depend on these methods. Several serological methods are currently available; these tests can be classified as screening tests and complementary or confirmatory tests (Nielsen, 2002; Poester *et al*., 2010).

Screening test for brucellosis:-there are many screening tests which are used to diagnose brucellosis in small ruminants. The Rose Bengal Plate Test (RBPT) is the most common screening test for detection of *Brucella* agglutinins. The principle of the test is that the sera collected from animals were mixed with antigen and examined for agglutination (Neilsen and Dunkan, 1990).

The use of the Rose Bengal Plate Test, which is easy to perform and is considered a valuable screening test, is less effective than the CFT at detecting brucellosis in small ruminants. Buffered plate agglutination (BPA) tests are the well-known buffered *Brucella* antigen tests. These tests are rapid agglutination tests lasting 4 minutes and it is done on a glass plate with the help of an acidic-buffered antigen (pH 3.65 ± 0.05). These tests have been introduced in many countries as the standard screening test because it is very simple and thought to be more sensitive than the SAT (Greiner *et al.,* 2009).

Confirmatory serological tests:-There are many serological tests that can be used as confirmatory serological tests for brucellosis. Among them the most common are Complement Fixation Test (CFT), Enzyme Linked Immune Sorbent Assay (ELISA), Serum Agglutination Test (SAT), Agar Gel Immune Diffusion (AGID) test. Among them ELISA and CFT are the most commonly used confirmatory serological tests. The complement fixation test is highly efficient and therefore accepted worldwide (Nielsen, 2002). Due to its high accuracy, complement fixation is used as confirmatory test for *B. abortus*, *B. melitensis*, and *B. ovis* infections and it is the reference test recommended by the OIE for international transit of animals (OIE, 2009). The CFT indicates active *Brucella* infection better than any other serologic test. It detects mostly IgG antibodies which are present in both acute and chronic stages of brucellosis (Seifert, 1996).

Another confirmatory serological test used for brucellosis is Enzyme linked Immune sorbent Assay (ELISA). **S**ince neither a single serological test nor a combined use of several serological tests detects all infected animals in a flock, detection of brucellosis remains a major problem in areas of low prevalence of *Brucellosis.* Most studies agree that the ELISA is as specific as the CFT but it is more sensitive. Yet, for a reliable diagnosis of infected animals studies suggest using the ELISA in combination with other tests (Bercovich *et al*., 1998). Small ruminants should be tested with the ELISA and CFT tests to prevent the spread of brucellosis after an outbreak of the disease in an area with low prevalence of brucellosis or in an area free from brucellosis (Bercovich *et al.,* 1998).

2.7. Significance on Economic and Public Health

2.7.1. Public health importance

Since there is close contact between humans and their livestock, which sometimes share the same housing enclosures, brucellosis is a significant health risk for the entire community. is readily transmissible to humans, causing acute febrile illness – undulant fever – which may progress to a more chronic form and can also produce serious complications affecting the musculoskeletal, cardiovascular, and central nervous systems. Brucellosis is a zoonotic bacterial disease caused by *Brucella* spp. and is primarily a disease of animals whereas humans are accidental hosts (Corbel, 2006). The disease is one of the most widespread zoonotic and is endemic in many countries. It is also considered a neglected zoonotic by the WHO (WHO, 2006). There are six identified species and numerous biotypes. B. *melitensis*causes disease primary among sheep and goats. The bacteria show a strong host preferece although cross-species infections happen, particularly with B. *melitensis* (Corbel, 2006). Clinical manifestation among humans is acute febrile illness which may persist and develop into a chronic disease with serious complications, such as joint illness, organ failure and symptoms of mental illness (Corbel, 2006; Quinn *et al,* 2002). The mortality rate is relatively low, especially when the patient is treated with adequate antibiotics; however this is not the case for everyone in low income countries (Corbel, 2006).In endemic countries humans get infected mainly by drinking unpasteurized milk and/or exposure to aborted fetuses, placentas or infected animals (FAO, 2010).There is an occupational risk to veterinarians, abattoir workers and farmers who handle infected animals and aborted fetuses or placentas. Brucellosis is one of the most easily acquired laboratory infections, and strict safety precautions should be observed when handling cultures and heavily infected samples, such as products of abortion. The most reliable and the only unique method for diagnosing animal brucellosis is isolation of *Brucella*s pecies (Alton *et al*., 1988).

2.7.2. Economic importance

Brucellosis presents a significant impediment to the economic potential of the large population of small ruminants. Since small ruminants and their products is an important export commodity, detaining seropositive animals in quarantine has a negative economic impact. The main economic consequences of brucellosis in small ruminants are: infertility, a high mortality in lambs and kids, outbreak, vaccine and research costs, movement restrictions, culling, market loss due to risk of infected meat, and milk, mortality, morbidity, lower production, loss of exports, loss of animal genetic resources and opportunities occasioned by spending on disease prevention and, mastitis. The reproductive wastage associated with brucellosis is another obstacle to optimal exploitation of the small ruminant sector. Reproductive losses are due to abortion, birth of weak offspring, and infertility (Quinn *et al.,* 1999; Seifert, 1996).

2.7.3. Status of small ruminant brucellosis in Ethiopia

Studies conducted on small-ruminant brucellosis in Ethiopia have indicated that sero-prevalence of the disease is varied from place to place (Ashagirie *et al*., 2011; Bekele *et al.,* 2011) which might be due to the differences in animal production and management systems as well as reasonably difference in agro-ecological conditions of the study places and C (Table 1).Reports indicated that the prevalence of small-ruminant brucellosis was much higher in area where farmers practice the communal use of grazing land than in clan-based flock/herd segregation areas (Yibeltal, 2005). This might be due to mixing animals from various areas in communal grazing system and watering points. reported prevalence proportion of 1.5% in sheep and1.3% in goats in the central highlands, 15% in sheep and16.5% in goats in the Afar region, 1.6% in sheep and 1.7% in goats in the Somali region (Yibeltal, 2005) and 1.6% in sheep and 1.7% in goats in Somali region (Teshale *et al*., 2006).

Table1: Prevalence of small ruminant Brucellosis in different Regions of Ethiopia.

Region	Prevalence		Source
	Ovine	Caprine	
Afar	3.2%	5.8%	(Ashenafi *et al.,*2007)
Somali	1.64%	1.51%	(Mohammed, 2009)
Oromia	1.9%	4.8%	(Haileleul, 2012)
SNNP	1.6%	3.2%	(Mengistu, 2007)
Tigray	1.4%	5.5%	(Teshale et al.,2013)
Amhara	4.89%		(Shimeles, 2008)

2.8. Control and Prevention Strategies

2.8.1. Vaccination

Control of brucellosis can be achieved by using vaccination to increase the population's resistance to the disease. Vaccination practically eliminates the clinical signs of brucellosis and is accompanied by a reduced contamination of the environment as well as exposure of the population at risk to the infectious agent (Nicoletti, 1993).The *B. melitensis* REV 1 vaccine is an attenuated strain of *B. melitensis* and an effective method to reduce the prevalence of brucellosis among whole flocks or herds in low income countries and/or endemic countries (Corbel*,* 2006; OIE, 2009). However, in many countries, where the animals were kept under extensive conditions with nomadic or semi-nomadic husbandry, this approach was impractical and failed to reduce the incidence and prevalence of the disease, because the development of herd immunity was very slow. In addition, the unvaccinated adult animals remain unprotected and the infection can spread (Kolar, 1995).

Vaccination of all animals (young and adults) in a flock or region is an alternative approach for the control of brucellosis in small ruminants. This, mass immunization is indicated where the prevalence of infected animals is high. Mass vaccination of a flock helps to rapidly establish a relatively immune stock, and reduces the level of abortions and excretes of thus reducing contamination of the environment and disease transmission (Kolar,1995).However, this strategy has the limitation that pregnant animals cannot be vaccinated because the vaccine is not innocuous enough for pregnant animals, and the efficacy of the strategy depends on the continuous availability of the vaccine (WHO, 1998).Provided that the prevalence of disease is moderate, financial resources are available, and a well-functioning surveillance by the veterinary service is in place, vaccination of young animals can be combined with a test and slaughter policy in a long term action to control brucellosis in small ruminants (WHO, 1998).

2.8.2. Test and slaughter

It is usually accepted that a programme of eliminating brucellosis by test and-slaughter policy is justified on economic grounds only when the prevalence of infected animals in an area is about 2% or less (Nicoletti, 1993). For the implementation of such a program it is essential that the flocks are under strict surveillance and movement control. Animals must be individually identified and an efficient and well organized veterinary service for surveillance and laboratory testing must be in place (Alton, *et.,* 1988; Nicoletti, 1993).The flock size as well as the prevalence of brucellosis is the most important factors of this strategy which has been shown to be ineffective and unreliable when attempted in large flocks with a high prevalence of brucellosis (Kolar, 1995).

Before embarking on the implementation of such a strategy it is necessary to ensure that the epidemiological situation is favorable, the necessary facilities and financial resources are available, a pool of healthy replacement animals is available and that the resources exist for continuing surveillance for a considerable period. (Nicoletti, 1993). A brucellosis control and eradication plan based on test and slaughter strategy can be either voluntary or compulsory. Voluntary schemes, which apply to individual flocks, may be useful in the early stages of the campaign but may need to be supported by adequate incentives such as a bonus on the sale of milk from brucellosis-free herds or per capita payments. Compulsory eradication is required in the final stages but is often advisable from the start (WHO, 1998).

3. Conclusion and recommendations

Small ruminant brucellosis is one of the common bacterial infections that affect healthy of small ruminants throughout the World, especially in developing country with extensive management system. *Brucella abortus, B. melitensis* and *B. ovis are the causative agents*. Goats and sheep are important domestic animals highly adaptable to broad range of environmental conditions. *Brucella melitensis* is the most virulent, which cause mass abortion in small ruminants during the first outbreak of the disease in the flock. Discharge from reproductive organs of infected animal, aborted fetus and contaminated environment are the common source of infection and transmission is facilitated by contacting with this agents. Isolation and identification of bacteria, serological and immunological tests are the common diagnostic method for diagnosis of small ruminant brucellosis. The disease has high economic impact by reducing reproductivity and productivity of the animal and have zoonotic importance .

Based on the aforementioned conclusion, the following recommendations were forwarded:

- There should be a strategy to regulate the control mechanism of brucellosis in small ruminants at national level.
- Efforts should be made to develop a new vaccine against brucellosis in sheep and goats based on rough strains which is devoid of the disadvantages of the vaccine.
- The government, Public health officers and Veterinarians have to work together to reduce economic and zoonotic impact of brucellosis

4. REFERNCES

Ademosoum A.A. (1994): Constraints and prospects for small ruminant research and development in Africa. Addis Ababa, Ethiopia, Pp.1-5.

Ackermann M.R., Cheville N.F. and Deyoe B.L. (1988): Bovine ileal dome lymphoepithelial cells: endocytosis and transport of *B.abortus* strain 19. *Vet. Pathol*, 25: 28 – 35.

Alton G.G. Jones L.M., Angus R.D., and Verger J.M. (1988): Techniques for the brucellosis laboratory Iran, Paris, Pp. 190.

Anderson T.D. and Cheville N.F. (1986): Ultra structural morphometric analysis of *Brucella abortus*-infected trophoblasts in experimental placentitis: bacterial replication occurs in rough endoplasmic reticulum. *Am. J .Pathol*, 124:226–237.

Anderson T.D., Cheville N.F., and Meador V.P. (1986): Pathogenesis of placentitis in the goat inoculated with *Brucella abortus*. II. Ultra structural studies. *Vet. Pathol*. 23:227–239.

Ashagirie T., Deneke Y., and Tolosa T. (2011): Seroprevalence of caprine brucellosis and associated risk factors in South Omo Zone of Southern Ethiopia, Africa. *J. Micro. Res.*, 5:1682–1476.

Ashenafi F., Teshale S., Ejeta G., Fikru R., and Laikemariam Y. (2007): Distribution of brucellosis among small ruminants in the Pastoral region of Afar, Eastern Ethiopia. *Sci. and Tech. Rev. World Organ. Ani. Health,* 26: 731–739.

Bekel M., Mohammed H., Tefera M. and Tolosa T. (2011): Small ruminant brucellosis and community perception in Jijiga District, Somali Regional State, Eastern Ethiopia. *Trop. Anim. Health. Prod*,43: 893–898.

Bercovich Z., Goler L., Baysal T., Schreuder B., Zijderveld and Fgvan, (1998): Evaluation of the current used diagnostic procedures for detection of *B.melitens* in sheep. *Smal. Rum. Res.,* 31:1-6.

Bricker B.J. (2002): Diagnostic strategies used for the identification of *Brucella.Vet. Microbio.*, 90, 433-434.

Central Statistical Agency (2005): Estimated number of cattle, sheep and goats by region: 2002/2003-2004/2005 (private peasant holdings for rural only). CSA, Addis Ababa.

Corbel M.J. (2006): Brucellosis in humans and animals. The World Health Organization, in collaboration with the Food and Agriculture Organization of the United Nations and the World Organization for Animal Health Geneva: WHO Press.

Corbel M.J. and Brinley-Morgan W.J. (1984): Genus *Brucella* Meyer and Shaw 1920, In Krieg, N.R., Holt, J.G.: Bergey's Manual of Systematic Bacteriology,Vol. 1, Williams & Wilkins, Baltimore-London, Pp.377-388.

Durán Ferrer M. (1998): Comparación entre métodosinmunológicodiagnóstico dela brucellosis ovinapor *Brucella melitensis* eficaciade inmunización de ovejasadultas con la vicuna Rev.1 porvíaconjunctival. PhD Thesis, University of Murcia, Spain.

Dwight C. H., and Yaun Ch. Z., (1999): Veterinary Microbiology; Blackwell Science, Cambridge, Massachusetts Pp. 197- 201.

Food and Agriculture Organization (2010): Animal Health Yearbook, FAO animal production and Health series, FAO, Rome, Italy.Pp.16-21

Fensterbank R. (1987): Some aspects of experimental bovine brucellosis. *Ann. Rech. Vet.* 18:421-428.

Food and Agriculture Organization of the United Nations (2010): *B.melitensis* in Eurasia and the Middle East. FAO Animal Production and Health Proceedings

Glenn J. S., and Karen W. P., (2005): Veterinary Microbiology: Bacterial and Fungal agents of animal diseases; pp.200-203.

Greiner M., Verloo D. and Massis F. (2009): Meta-analytical equivalence studies on diagnostic tests for bovine brucellosis allowing assessment of a test against a group of comparative tests. *Prev. Vet. Med.,* 92:373–81.

Grillo M.J., Barberan M. and Blasco J.M. (1997): Transmission of *B.melitensis* from sheep to lambs. *Vet. Rec.,*

140: 602–605.
Haileleul (2012): Assessment of risk factors and seroprevalence of small ruminant brucellosis in Adamitulu-Jido-Kombolcha District, Oromia Regional State, Ethiopia *Libyan Agric. Res. Cen. J. Intl.,* 3: 47-52.
Hirsh D.C. and Zee Y.C. (1999): Veterinary microbiology. Blackwell Science, Cambridge, Massachusetts, Pp. 196-203.
Ibrahim H. (1998). Small ruminant production techniques. International Livestock Research Institute (ILRI) training manual No. 3. ILRI, Nairobi, Kenya, Pp. 11-47.
International Livestock Research Institute (2006): Domestic animal genetic resources information system (DAGRIS).
Kolar J. (1995): Some experience from brucellosis control with Rev.1 vaccine in a heavily infected country - Mongolia .FAO/WHO/OIE Round table on the use ofRev.1 vaccine in Small Ruminants and Cattle. CNEVA, Alfort, France September 21-22.
Lughano ,Kusiluka ,Dominic ,Kambarage (1996): Common Diseases of Sheep and Goats in Sub-Saharan Africa. Diseases of small ruminants, 3rd ED .A handbook Centre for Tropical Veterinary Medicine. Scotland.
Mengistu M. (2007):Sero epidemiology of Brucellosis in Small Ruminants in Southern Ethiopia Master Thesis, Addis Ababa University, Faculty of veterinary Medicine.
Mohammed H. (2009): Seroprevalence of small ruminant brucellosis in and around Jijiga. DVM thesis, School of Veterinary Medicine, Jimma University, Jimma, Ethiopia
Nicoletti P. (1993): The Eradication of Brucellosis in Ann. *Saudi. Med .J,* 14:4:288-292.
Nielsen K. and Duncan, J.R. (1990): Animal brucellosis.CRC Press, Boca Raton, Florida, 173-179.
Nielsen K. (2002): Diagnosis of brucellosis by serology. *Vet. Microbio.,* 90:447-459.
Office of International Epizooties (2009): Caprine and ovine brucellosis (excluding *B.ovis*).Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, OIE, Paris.
Pan American Health Organization (2001): Zoonoses and communicable diseases common to man and animals Washington D.C., USA. 3rd *Ed.*; Pp.53-55.
Pastoralist Forum Ethiopia (2004): Background the Ethiopian livestock industry. *In* Proc.3rd National Conference on Pastoral Development in Ethiopia: pastoralism and sustainable pastoral development, 23-24 December, Addis Ababa. PFE, Addis Ababa, Pp. 78-79.
Pizarro-Cerda J., Meresse S. and Parton R.G. (1998): *B.abortus* transits through the autophagic path way and replicates in the endoplasmic reticulum of nonprofessional phagocytes. *Infect Immun*., 66:5711–5724.
Poester F.P., Nielsen K. and Samartino L.E. (2010): Diagnosis of brucellosis. *Open Vet. Scien. J.*, 4:46-60.
Quinn P.J., Carter M.E., Markey B.K., and Carter G.R. (1994): Clinical Veterinary Microbiology, Pp. 261.
Quinn P.J., Carter M.E., Markey B., and Carter G.R, (1999): Clinical veterinary microbiology, 1st Ed. Mosby, Edinburgh, Pp.261-267.
Quinn P.J., Markey B.K., Carter M.E., Donnelly W.J. and Leonard F.C. (2002): Veterinary Microbiology and Microbial Disease. Dublin: Blackwell Science Ltd. Pp. 162-167.
Radostits O.M., Gay C.C., Blood D.C. and Hinchcliff K.W. (2000): Veterinary medicine: a text book of the diseases of cattle, sheep, pigs, goats and horses, 9th Ed. W.B. Saunders Ltd, Oxford, Pp. 867-882.
Robles C.A, Uzal F.A., and Olaechea F.V. (1998): Epidemiological observations in a Corriedale flock affected by *B.ovis. Vet. Res. Commun.*, 22: 435-43.
Seifert S.H. (1996): Tropical animal health, 2nd Ed. Kluwer Academic, Dordrecht, the Netherlands, Pp.356-367.
Shimeles A. (2008): Study on sheep brucellosis in selected woredas of the Eastern Amhara regional state, north Eastern Ethiopia, M.Sc. thesis, Addis Ababa University, Faculty of Veterinary Medicine, Debre Zeit, Ethiopia.
Smith M.C. and Sherman D.M. (1994): Goat medicine. Lea and Febiger, Philadelphia, Pennsylvania, Pp. 423-424
Tekelye B. and Kasali O.B. (2014): Brucellosis in sheep and goats in Central Ethiopia. *Bull. Anim. Health Prod.,* 38: 23-25.
Teshale S., Aschalew Z., Gelagay A. and Basu A.K. (2006): Preliminary study on prevalence of *Brucella* antibodies in sheep and goats in Borana, Southern Ethiopia. *J. nat. Hist*., 2 :7-10.
Teshale T., Tadele T., Getachew T., Belay B., and Birhanu H. (2013): Sero-prevalence and risk factors study of brucellosis in small ruminants in Southern Zone of Tigray Region, Northern Ethiopia *Trop. Anim. Health. Prod*. 45:1809-1815.
Walker R.L. (1999): Veterinary microbiology. Black wells Science, Cambridge, Massachusetts, Pp.196-203.
World Health Organization (1998): Human and Animal Brucellosis. Report of a WHO workshop. Damascus, Syrian Arab Republic, Pp. 4-5.
World Health Organization (2006): The control of neglected zoonotic diseases: a route to poverty alleviation: report of a joint WHO/DFID-AHP meeting, 20 and 21 September 2005. Geneva: WHO, with the participation of FAO and OIE.

Xavier M.N., Silva T.M.A., Costa E.A., Paixão T.A., Moustacas V.S., Carvalho C.A., Sant'anna F.M., Robles C.A., Gouveia A.M., Lage A.P., Tsolis R.M., and Santos R.L. (2010): Development and evaluation of a species-specific PCR assay for the detection of *B.ovis*infection in rams. *Vet. Microbio.*, 145:158-164.

Yibeltal M. (2005): A seroprevalence study of small Ruminant brucellosis in selected sites of the Afar and Somali regions, Ethiopia. DVM thesis, Faculty of Veterinary Medicine, Addis Ababa University, Debre Zeit, Ethiopia.

Young E.J., Borchert M., Kretzer F.L., and Musher D.M. (1985):Phagocytosis and killing of *Brucella* by human polymorphonuclear leukocytes. *J. Infect. Dis*., 151:682–690.