Chapter 3
Small Ruminants and Zoonotic Infections: Live or Dead—Direct or Indirect

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Abstract Sheep and goat can be infected with several pathogens. While some may have a great impact on the small ruminant industry, others are more important as zoonotic agents. Human infections can occur from contact with both live and dead animals, animal products and wastes. Several microorganisms may cause severe infection if transmitted to vulnerable people, such as pregnant and immune-comprised persons. The present chapter will briefly deal with a selected number of zoonotic microorganisms where small ruminants play an important role as hosts for human infection.

3.1 Introduction

Sheep and goats which were domesticated by humans as early as 9000 years ago are distributed worldwide and their products, such as milk, meat, wool and skin are used extensively. Microorganisms have therefore been shared between humans and small ruminants for a long period of time. In this context, small ruminants may carry zoonotic pathogens, either transiently or permanently. The risk of transmission to humans varies considerably due to geographical area, seasons, climatic conditions and management systems. The impact on human health will also depend on pathogen species/subspecies, virulence of the pathogen, level of exposure, presence of co-infections, susceptibility of the host, the host immune status and the transmission route.

In general, microorganisms can be transmitted from small ruminants to humans by direct contact, aerosols, milk, meat, or indirectly by manure, feces, urine and wool. For instance, the development of zoonotic infections through exposure to manure, either directly or indirectly, constitutes a real and significant risk of human health. Contamination of ground, irrigation or drinking water provides not only a source of infection, but also a mean to spread the pathogens (Milinovich and Klieve 2011).

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Water and food-borne zoonotic pathogens, such as *Camplylobacter jejuni*, *Cryptosporidium parvum*, *Escherichia coli* (EHEC), *Giardia duodenalis* and *Salmonella* spp. are widespread and have a wide host range. *Salmonella* spp. for instance, are ubiquitous in nature and have been isolated from a wide variety of vertebrate hosts. *Salmonella* is also commonly found in farm animals, their environments and is one of the most important food-borne zoonotic microorganisms (Milinovich and Klieve 2011). Infections from goat products due to consumption of raw or uncooked goat meat, milk and cheese have been documented. Severe gastroenteritis and even fatalities may occur (Desenclos et al. 1996; Espié and Vaillant 2005). However, human salmonellosis may not normally arise as a result of contact with small ruminants, since this transmission pathway seems to be less frequent than that from other animals such as cattle and poultry (Kirby 1985; Rabinowitz and Conti 2010). Similarly, all the above mentioned pathogens are important zoonotic agents, but small ruminants have normally a less important role as reservoir for human infection compared to other species (Palmer et al. 1998). These microorganisms will therefore not be described further in this condensed review.

Some microorganisms commonly found in small ruminants have a limited zoonotic potential. For instance, sheep on *Ixodes ricinus* tick-infested pasture in northern Europe are often infected with *Anaplasma phagocytophilum*, which may cause serious and fatal infection. However, phylogentic studies show that strains/variants of *A. phagocytophilum* isolated from sheep differ from isolates normally affecting humans, indicating that sheep are rarely reservoir hosts for human infection (Scharf et al. 2011).

Other links between diseases in humans and animals are still debated. For instance, *Mycobacterium avium* subspecies *paratuberculosis* (MAP) causing Johne's disease in ruminants has been isolated from some humans with Crohn's disease, a chronic granulomatous infection of the human intestine (Sharp 2007; Smith and Sherman 2009). The association between this bacterium and the disease, however, is still unclear. The presence of MAP in a percentage of Crohn's disease patients is either associated with the pathogenesis of the disease or these patients may be more likely to be colonized by this organisms. The unanswered question raises the issue of meat, milk and water contamination by MAP and human health (West et al. 2009; Singh and Gopinath 2011). It is also still questioned if Borna disease virus, where sheep is one of the principle species affected, could cause infection in humans (Chalmers et al. 2005; Dürrwald et al. 2007). These issues will not be discussed further in this chapter.

3.2 Specific Infections

Zoonotic pathogens detected in small ruminants of which several could cause severe infection in humans, are listed in Tables 3.1, 3.2, and 3.3. These lists, however, are not complete. Microorganisms may for instance be transmitted to humans due to the lack of normal hygiene procedures when handling infectious material. Common pathogens, such as *Staphylococus aureus* and *Trueperella pyogenes*, which regularly cause infections in small ruminants, are not covered by this chapter. In this short

Table 3.1 Zoonotic bacteria, *Rickettsia* and *Chlamydia* detected in small ruminants

Pathogen	Host	Distribution	Transmission	Clinical symptoms (small ruminants)	Clinical symptoms (human)	References
Anaplasma phagocytophilum (several variants)	Several mammals	Northern hemisphere (Ixodes-tick)	Ticks	Fever, abortion (secondary infections)	Flu-like	Woldehiwet 2010
Bacillus anthracis	Several mammals	Worldwide	Aerosols, cutaneous, oral (spores)	Found dead	Variable, cutaneous, pulmonary and intestinal form	Turnbull 1998
Borrelia burgdorferi sensu lato	Several, incl. small rodents, birds	Northern Hemisphere (Ixodes-tick)	Ticks	Subclinical, arthritis	Variable acute subacute -chronic form	Stanek et al. 2002
Brucella melitensis (*B.abortus*)	Several, mainly small ruminants	Widespread, especially Mediterranean, Middle East	Oral (aerosols, cutaneous)	Abortion, arthritis	Variable, undulating fever chronic	See text
Burkholderia pseudomallei	Several	Widespread, mainly tropical areas	Oral, insects, vertical transmission	Abscesses, weight loss, polyarthititis meningoencephalitis	Pneumonia, septis, genitourinary infection, abscesses, suppurative parotitis, encephalomyelitis	Cheng and Currie 2005 Smith and Sherman 2009
Camphylobacter jejuni	Several, esp. poultry	Widespread	Oral	Abortion, watery diarrhoea	Flu-like, diarrhoea	Skirrow 1998
Chlamydopila abortus	Several, mainly small ruminants	Widespread	Aerosols	Abortion	Abortion, stillbirth, puerperal sepsis, renal failure, hepatic dysfunction, DIC	See text
Corynebacterium pseudotuberculosis	Several, incl. domestic animals	Widespread	Cutaneous, oral	Caseous lymphadenitis	Suppurative granulomatous lymphadenitis	Thomas 1998 Smith and Sherman 2009
Coxiella burnetii	Several, incl. livestock	Widespread	Aerosols, oral (cutaneous, ticks)	Abortion, stillbirth, weak offspring	Flu-like, pneumonia, endocarditis, hepatitis	See text

Table 3.1 (continued)

Pathogen	Host	Distribution	Transmission	Clinical symptoms (small ruminants)	Clinical symptoms (human)	References
Dermatophilus congolensis	Several species	Worldwide	Cutaneous	Dermatitis (exudate)	Dermatitis	Stewart 1972a, b Hyslop 1980
Escherichia coli (EHEC)	Several	Worldwide	Oral	Enteritis, diarrhoea, septicaemia, mastitis	Variable diarrhoea, haemorrahagic colitis HUS	Nelson et al. 1998 Smith and Sherman 2009
Francisella tularensis	Several hosts, esp. rodents	Worldwide	Aerosols, oral, cutaneous, ticks	Sepsis	Variable bubonic—intestinal -pneumonic form	Pearson 1998
Leptospira spp. serovar Pomona (serovar Hardjo-bovis)	Several mammals, incl. cattle, pig	Unknown	Oral, cutaneous	Fever, depression, dyspnea, weakness, anaemia, icterus, haemoglobinuria	Flu-like, encephalitis	See text
Listeria monocytogenes (*L. ivanovii*)	Several	Worldwide	Oral, cutaneous	Abortion, encephalitis, septicaemia, mastitis, diarrhoea, ocular disease	Meningitis, encephalitis, septicaemia	See text
Mycobacterium avium subspecies *paratuberculosis* (John's disease)	Several, esp. ruminants and rabbits	Widespread	Oral	Progressive weight loss, diarrhoea, intermandibular oedema	Chronic bronchitis, cervical lymphadenopathy, disseminated disease (Crohn's disease?)	Gallagher and Jenkins 1998 Smith and Sherman 2009
Salmonella spp.	Several	Worldwide	Oral	Gastroenteritis, septicaemia, abortion	Enteric fever, gastroenteritis, diarrhoea, septocaemia	Humphrey et al. 1998 Mearns 2007 Smith and Sherman 2009
Yersinia enterocolitica, Y. paratuberculosis	Several	Widespread	Oral	Enteritis, mastitis, abortion, ill-thrift	Enterocolitis, polyarthritis, erythema nodosum, exudative pharyngitis, sepsis	Butler 1998 Smith and Sherman 2009

Table 3.2 Zoonotic virus detected in small ruminants

Pathogen	Host	Distribution	Transmission	Clinical symptoms (small ruminants)	Clinical symptoms (human)	References
Louping-ill (genus *Flavivirus*)	Several, especially sheep and grouse	Scandinavia, United Kingdom	Ticks, (*Ixodes ricinus*) (Aerosols, oral, cutaneous)	Variable, subclinical—incoordination—paralysis	CNS-symptoms	Reid and Chianini 2007
Nairobi sheep disease (genus *Nairovirus*)	Small ruminants	Africa	Ticks (mainly *Rhipicephalus appendiculatus*)	Fever, diarrhoea, gastroenteritis, death	Rare benign illness	Swanepoel 1998 Smith and Sherman 2009
Orf (genus *Parapoxvirus*)	Several, mainly small ruminants	Worldwide	Cutaneous (oral)	Wart-like outgrows	Skin lesions	See text
Rabies (genus *Lyssavirus*)	Several	Widespread	Bite, (Aerosols, oral)	Behaviour changes, paralysis, paralytic/furious condition, death	Non-specific (prodromal period) Paralysis, aggression, unconsciousness, paralysis	King 1998
Rift Valley Fever (genus *Phlebovirus*)	Ungulates	Africa, Arabian Peninsula	Mosquitoes (*Aedes* spp.). Other insects	Abortion, fever, listlessness, recumbency	Flu-like Menigoencephalitis, haemorrhagic fever, photophobia, retinitis	See text
Wesselsbron disease (genus *Flavivirus*)	Several, incl. domestic ruminants and rodents	South Africa	Mosquitos (Aedes), (Aerosols, oral)	Abortion, sudden death	Fever, rash, arthralgia	Leake 1998 Smith and Sherman 2009

Table 3.3 Zoonotic parasites and fungus detected in small ruminants

Pathogen	Host	Distribution	Transmission	Clinical symptoms (small ruminants)	Clinical symptoms (human)	References
Cryptosporidium parvum	Several, esp. cattle	Worldwide	Oral	Subclinical (adults) Watery diarrhoea (young animals)	Diarrhoea	Wright and Coop 2007 Smith and Sherman 2009
Dicrocoelium dendriticum	Several, esp. domestic ruminants	Widespread	Oral	Subclinical Weight loss, Anaemia	Subclinical Constipation/diarrhoea, hepatomegaly	Smith and Sherman 2009
Echinococcus granulosus	Several (intermediate hosts) Canids (end host)	Widespread	Oral	Mainly subclinical	Variable (localisation/size) Shock/pulmonary oedema	See text
Eurytrema pancreaticum	Several domestic animals, esp. ruminants	Asia, South America	Oral	Subclinical, ill-thrift, weight loss emaciation	Non-specific	Lloyd and Soulsby 1998 Taylor et al. 2007 Smith and Sherman 2009
Fasciola hepatica	Several ruminant Snail: (intermediate hosts)	Worldwide	Oral	Acute, subacute, chronic (anaemia, icterus, submandibular oedema, death)	Variable Acute (hepatic) -chronic (biliary) phase	Mas-Coma 2005 Smith & Sherman 2009 Fried and Abruzzi 2010
Giardia duodenalis	Several species	Worldwide	Oral	Subclinical, enteritis, diarrhoea	Variable, diarrhoea, chronic syndrome	Thompson 1998 Taylor et al. 2007
Oestrus ovis	Small ruminants	Unknown	Flies	Nasal discharge, sneezing, rubbing (noses) (unthriftiness/ incoordination)	Catarrhal conjunctivitis, stomatitis	Beesley 1998 Taylor et al. 2007

Table 3.3 (continued)

Pathogen	Host	Distribution	Transmission	Clinical symptoms (small ruminants)	Clinical symptoms (human)	References
Schistosoma spp.	Several	Widespread, esp.tropics, subtropics)	Cutaneous	Rhinitis, enteritis, hepatitis, pneumonia	Multisystemic Non-specific Dermatitis	Taylor 1998 Smith and Sherman 2009
Taenia multiceps (*Coenurosis cerebralis*)	Sheep	Unknown,	Oral	Depression, blindness, convulsions	Variable, (localisation)	Lloyd 1998
Toxoplama gondii	Multiple intermediate hosts, Felid family (end host)	Worldwide	Oral (tissue cysts)	Abortion, stillborn, weak offspring	Mild—transient—serious abortion, congenital lesions	See text
Tricophyton verrucosum	Several, mainly cattle	Worldwide	Cutaneous	Alopecia, scaling, crusting, folliculitis	Dermatophytosis	Sparkes 1998

review, the focus is on the distribution, hosts, disease manifestations, transmission, diagnosis, treatment and control measures on a selected number of zoonotic micro-organisms where small ruminants may play an important role as reservoir hosts for human infection. Focus will be on the following pathogens: *Brucella melitensis*, *Chlamydia abortus*, *Coxiella burnetti*, *Echinococcus granulosus*, *Leptospira interrogans*, *Listeria monocytogenes*, Orf-virus, Rift Valley fever virus and *Toxoplasma gondii*.

3.2.1 Brucellosis

3.2.1.1 The Pathogen

Bacteria of the genus *Brucella* are Gram-negative coccobacilli. There are four important species that may cause infection in humans, whereas *B. melitensis* is considered as the most invasive producing the most severe disease in humans (Godfroid et al. 2005; El-Koumi et al. 2013). *B. melitensis* is associated with small ruminants, although *B. abortus* may occasionally cause infection in sheep and goats.

3.2.1.2 Occurrence

Worldwide, particularly in the Mediterranean region, Middle East, parts of Asia and Africa and Central and South America (Corbel 1997; Castrucci 2007).

3.2.1.3 Hosts

B. melitensis is primary found in sheep, goats and camels, but cattle, dogs and rats can also acquire the infection (Castrucci 2007).

3.2.1.4 Disease in Small Ruminants

B. melitensis may cause abortion, and occasionally orchitis and arthritis. Usually, abortion occurs from mid to late pregnancy. The infection may persist in the udder to the following pregnancies. Excretion of bacteria may last for 2 months in vaginal discharges and up to 180 days in milk after delivery or abortion (Castrucci 2007; Scott 2007; Smith and Sherman 2009).

3.2.1.5 Disease in Humans

It has been estimated that around 500,000 cases of human brucellosis occur annually (Franco et al. 2007). The incubation period varies from 1 week to several months. Human brucellosis can be both an acute and a chronic febrile illness with a variety

of clinical manifestations. The patient may show fever, chills, headache, muscle and joint pains, malaise, nausea, night sweats and lack of appetite for 3–6 weeks. The condition may also show a variety of non-specific haematological changes, such as anaemia and leucopenia (Plommet et al. 1998; El-Koumi et al. 2013).

3.2.1.6 Transmission (Small Ruminants-Human)

The main route of entry is via the nasopharynx, although a cutaneous route of infection does also exist. Material from abortions represents the main source of transmission in ruminants, with the excretion of enormous numbers of bacteria in the placenta, fetal fluids, and fetus (Castrucci 2007). Humans, however, are mainly infected through ingestion of fresh (unpasteurized) milk, cheese and meat, but also through direct contact with infected animals, semen, vaginal fluids or infectious aerosols (Castrucci 2007; Smith and Sherman 2009). The environmental resistance of the pathogens varies; the organisms can for instance survive in dust for 3–44 days, in tap water for 30 days, on pasture between 15 and 35 days, and in liquid manure at 15 °C or below for up to 8 months (Plommet et al. 1998; Castrucci 2007).

3.2.1.7 Diagnosis in Small Ruminants

If abortion occurs, *B. meliensis* infection can be confirmed by bacteriological methods (aborted fetus and placenta) or serology (aborted ewe/doe). The diagnosis in the chronic stage of the infection is difficult, since the infection may become non-apparent. In non-pregnant ewes, the bacterium is not excreted from the vagina. However, during pregnancy, excretion starts at the time of delivery or abortion and could continue for several months (Castrucci 2007). There are several serological tests available, such as the standard agglutination test (SAT), Rose Bengal test, compliment fixation test and ELISA.

Although all organs may be infected, microscopic examination should focus on material with suspected large amounts of bacteria, such as placenta, fetus, and vaginal discharges in case of abortion. Stained tissue smears, bacterial culture or PCR can be used for identification (Pommet et al. 1998; Redkar et al. 2001).

3.2.1.8 Treatment and Control

Brucellosis has been controlled in many countries however it remains an important health issue in many developing countries. *B. melitensis* is considered as a important food safety concern in human, because it may be present in dairy products made from milk of infected small ruminants. The bacteria survive for days in fresh milk, weeks in ice cream and months in butter, although the bacteria are killed by pasteurization and are sensitive to common disinfectants (Godfroid et al. 2005).

Chemotherapy is not 100% effective, so little is accomplished with the control and eradication of brucellosis in small ruminants. The best scheme is to identify

and cull the infected animals (Castrucci 2007). Vaccination of sheep and goats with an attenuated strain of *B. melitensis* is considered to be the main control strategy. Vaccination prevents abortion and reduces pathogen shedding from immunized animals, although the vaccine may retain some degree of virulence which may result in abortion and excretion in milk. The vaccine may also interfere with serological testing (Godfroid et al. 2005). In addition, vaccination of replacement animals is not sufficient to control the disease, especially in countries with high prevalence, uncontrolled animal movements, nomadic and low socioeconomic conditions, and illegal import of animals (Ebrahimi et al. 2012).

Surveillance, testing and massive immunization of animals, and national brucellosis control are necessary to eradicate the disease (El-Koumi et al. 2013). For human consumption, unpasteurized milk and milk products should be avoided. No human vaccine exists, however recent results are promising in developing a recombinant vaccine against *B. melitensis* (Gomez et al. 2013).

3.2.2 Chlamydiosis (Ovine Enzootic Abortion (OEA))

3.2.2.1 The Pathogen

Ovine enzootic abortion (OEA) is caused by the obligate intracellular Gram-negative bacterium *Chlamydia abortus*. The organism belongs to the family *Chlamydiaceae* and genus *Chlamydia*, which comprise two distinct developmental forms, a small extracellular infectious elementary body (EB) and a larger intracellular non-infectious, metabolically active reticulate body (Longbottom and Coulter 2003).

3.2.2.2 Occurrence

C. abortus is recognised as a major cause of reproductive loss in sheep and goats worldwide, although the disease does not appear to be a problem in either Australia or New Zealand (Aitken and Longbottom 2007).

3.2.2.3 Hosts

Main hosts are small ruminants, but the organism can also infect cattle, pigs, horses and deer, although such infections are thought to be less common (Aitken and Longbottom 2007).

3.2.2.4 Disease in Small Ruminants

Infection in animals is usually asymptomatic, except abortion, although some behavioural changes or a vaginal discharge may be observed. Ewes/does may deliver

stillborn or weakly offspring that fail to survive. The majority of infected placentas will have thickened red intercotyledonary membranes, dark red cotyledons and have a creamy-yellow coloured exudate on the surface. An infectious vaginal discharge may be observed for several days following abortion, but otherwise the ewes/does are clinically normal and are considered immune to further disease (Longbottom and Coulter 2003; Aitken and Longbottom 2007; Smith and Sherman 2009).

3.2.2.5 Disease in Humans

Although rare, the greatest threat of human infection is to pregnant women, where the outcome of infection in the first trimester of pregnancy is likely spontaneous abortion, while later infection causes stillbirths or preterm labour (Hyde and Benirschke 1997). Several cases of abortion, puerperal sepsis and shock, including renal failure, hepatic dysfunction and disseminated intravascular coagulation, as well as death have been reported (Buxton 1986; Bloodworth et al. 1987).

3.2.2.6 Transmission (Small Ruminants-Human)

Most cases in humans are associated with direct exposure to infected sheep or goats via aerosols. The major sources of infection are contact with placental membranes, dead fetuses, live lambs/kids born to infected mothers and vaginal discharges (Aitken and Longbottom 2007; Smith and Sherman 2009).

3.2.2.7 Diagnosis in Small Ruminants

A presumptive diagnosis of infection can be made based on abortion in the last 2–3 weeks of gestation and examination of the placenta. Pathological changes involve both the intercotyledonary membranes and the cotyledons. This is usually confirmed by the identification of large numbers of EBs in stained smears prepared from the placental membranes and cotyledons using for instance a modified Ziehl-Nielsen procedure. Other methods of antigen detection include immunohistochemical staining of tissue sections, immunoassays, DNA amplification methods, and isolation in cell-culture. Serological testing is normally performed by the complement fixation test on paired blood samples. However, none of the current serological tests have been proven to be suitable for detecting infection prior to abortion and are not able to differentiate vaccinated animals from those infected with wild-type strains (Longbottom 2008; Sachse et al. 2009).

3.2.2.8 Treatment and Control

If OEA is suspected to be present in a flock or herd, the administration of long-acting oxytetracyclines will reduce the severity of infection and losses resulting from

abortion. Although such treatment will reduce losses and limit the shedding of infectious organisms, it does not eliminate the infection nor reverse any pathological placental damage already done; thus abortions or the delivery of stillborn or weakly lambs can still occur and the shed organisms are a source of infection for naïve animals (Longbottom and Coulter 2003; Aitken and Longbottom 2007). Animals that have aborted are considered immune to further disease. Ewes, however, may become persistently infected carriers and continue to excrete infectious organisms at the next oestrus (Papp et al. 1994; Papp and Shewen 1996).

In humans, early therapeutic intervention is important, whereas tetracycline, erythromycin and clarithromycin should be used. Severely ill patients require supportive therapy (Sillis and Longbottom 2010).

During an OEA outbreak the primary aim is to limit the spread of infection to other naïve animals. Affected animals should be identified and isolated as quickly as possible. All dead fetuses, placental membranes and bedding should be carefully destroyed and lambing pens cleaned and disinfected. Pregnant women and immune-compromised individuals are advised not to work with sheep, particularly during the lambing period and should avoid all contact with possible sources of infection. Basic hygiene procedures, including thorough washing of hands and the use of disposable gloves are essential when handling potentially infected materials (Winter and Charnley. 1999; Longbottom and Coulter 2003).

Live-attenuated vaccines based on a temperature-sensitive mutant *C. abortus* strain have been used for several years. These vaccines must be administered at least 4 weeks prior to mating and cannot be used in combination with antibiotic treatment. Good protection from abortion is obtained, but does not completely eradicate the shedding of infectious organisms at parturition. Moreover, some vaccinated animals still abort as a result of wild-type infections. Vaccine development to produce the next generation OEA vaccine continues to progress. This is likely to be a subunit vaccine based on protective recombinant antigens identified through comparative genomic and proteomic approaches (Longbottom et al. 2013; Entrican et al. 2012).

3.2.3 Contagious Ecthyma (orf)

3.2.3.1 The Pathogen

Contagious ecthyma is caused by orf-virus, a DNA- and poxvirus belonging to the genus *Parapoxvirus*.

3.2.3.2 Occurrence

Orf-virus is distributed worldwide.

3.2.3.3 Hosts

Several ruminants may be affected by orf-virus, especially small ruminants.

3.2.3.4 Disease in Small Ruminants

Orf-virus affects the skin primarily around the mouth and udder. There is considerable heterogenicity between virus isolates, but it is still not confirmed if different virulence exists. Genetic differences in orf virus strains seem to be due to geographic locations and animals hosts involved (Reid and Rodger 2007; Li et al. 2012).

The clinical manifestation is variable. Symptoms are seen most frequently in young lambs, normally in two peaks, first in spring shortly after lambing and then 3–4 months later. Morbidity usually approaches 100 %, while in most outbreaks the mortality is low. However, occasionally up to 80 % mortality has been recorded. Severity of outbreaks seems to be attributed to environmental factors (Reid and Rodger 2007).

The lesions usually develop at sites where the skin or the mucous membranes are traumatized. The first clinical signs are local erythema, followed by formation of papules, vesicles, and pustules ending in scab formation. Without secondary infections the lesions resolve within approximately 4 weeks. In natural cases, proliferation often gives rise to wart-like outgrows which may develop into extensive cauliflower-like structures that persist for a long period. Lesions are normally found around the mouth and nostrils, but may also develop on the buccal cavity, eosophagus, ears, axilla, poll, lower limbs and coronet. The infection can also spread to the udder thus increasing the risk of mastitis (Reid and Rodger 2007; Smith and Sherman 2009; Li et al. 2012).

3.2.3.5 Disease in Humans

In humans, after an incubation period of 3–7 days, a macropustular reaction occurs, most commonly found on one finger. As in small ruminants, the development stages comprise erythema, papules, vesicles, pustules and scabs. Several lesions may be present on hand and arm, but single lesions are more common. These are usually raised, circular or oval and about 0.5–1.5 cm in diameter, often with central vesiculation and pustulation. The lesions will normally heal and detach after 6–8 weeks without leaving a scar. However, secondary bacterial infection can cause complications, especially lymphangitis and lympadenitis of the draining lymph nodes which may be associated with flu-like symptoms. Infection may in some cases develop into a generalized reaction, including widespread maculopapular eruption and erythema multiforme. Extensive lesions have especially been seen in immunosuppressed people (Martin 1991b; Reid and Rodger 2007).

3.2.3.6 Transmission (Small Ruminants-Human)

Humans are mainly infected by direct contact with lesions from live animals. Infection can also be transmitted by fomites. Persons directly handling infected animals, particular when bottle-feeding lambs, shearing and slaughtering sheep are especially at risk (Reid and Rodger 2007).

3.2.3.7 Diagnosis in Small Ruminants

Diagnosis is mainly based on clinical signs, such as papillomatous lesions around the lips and nostrils. However, the clinical picture may be atypical and laboratory confirmation is necessary. Electron microscopy has earlier been used to verify the diagnosis, but PCR-methods are now available (Reid and Rodger 2007).

3.2.3.8 Treatment and Control

Outbreaks spread rapidly in a flock, with most animals becoming affected within a few weeks. Such outbreaks will last for 6–8 weeks. No specific treatment is available. The main treatment is to avoid secondary infections. A live vaccine is available in some countries. If vaccination during an outbreak is considered necessary, an autogenous vaccine can also be prepared (Reid and Rodger 2007). Vaccine development using a DNA-vaccine has recently showed promising results (Zhao et al. 2011).

Persistently infected animals with no clinical symptoms have been described. The importance of these animals in the epidemiology of the infection is unknown. The virus may survive in buildings and handling facilities between epidemics. Orfvirus is known to survive in dry scabs for a long period, up to 23 years at 7 °C, but the infectivity is lost more rapidly at higher temperature and at more moist conditions. Disinfection of the actual pens should be performed. Infection in humans can normally be avoided through good hygienic procedures. Protective gloves should be used when handling infectious animals or infective material (Reid and Rodger 2007; Smith and Sherman 2009).

3.2.4 Echinococcosis (Hydatidosis)

3.2.4.1 The Pathogen

E. granulosus is a tapeworm that belongs to the class *Cestoda* and the family *Taeniidae*. Several species of genus *Echinococcus* exist, but it is mainly *E. granulosus* that involves small ruminants as intermediate hosts. Ten genetic types (G1–G10) of *E. granulosus* have been characterized, of which two (G1 and G2) are "sheep" strains (Moro and Schantz 2009). G1 and G6 ("camel" strain) affect goats (Smith and Sherman 2009).

3.2.4.2 Occurrence

E. granulosus is widespread in areas where sheep are reared (Brunetti and White 2012).

3.2.4.3 Hosts

The definite host are domestic dogs and some wild canids. There are several intermediate hosts such as sheep, goats, cattle, swine, camelids, cervids, lagomorphs and humans. The sheep strain G1 is most commonly associated with human infection (Moro and Schantz 2009).

3.2.4.4 Disease in Small Ruminants

Cestode eggs which contain oncospheres must be ingested in order to continue the life cycle of the parasite. After ingestion, the larval stage will develop to cysts (hydatid cyst) in different organs, most commonly in liver and lungs. No definite clinical symptoms have been observed in small ruminants, even in cases with multiple cysts in either liver or lungs (Taylor et al. 2007).

3.2.4.5 Disease in Humans

E. granulosus cysts in humans may take years to develop and produce clinical symptoms. Many cysts remain asymptomatic throughout life and are only discovered by accident. However, the infection can result in respiratory distress and abdominal enlargement depending on which organ is affected. Clinical symptoms depend on the location and size of the *E. granulosus* cyst, and are mainly due to the pressure on the actual organ and on surrounding tissues. In man, the hydatid cysts may be 5–10 cm in diameter or even larger (Martin 1991a). The most common localization is the liver (70%), followed by the lungs. Rupture of the cyst is often fatal, due to anaphylactic shock or pulmonary oedema (Moro and Schantz 2009; Brunetti and White 2012).

3.2.4.6 Transmission (Small Ruminants-Human)

The dog-sheep-dog cycle is the most important cycle in several endemic areas. Small ruminants normally contract *E. granulococus* by gracing on pasture contaminated by dog faeces containing cestode eggs. The dogs are again infected by ingestion of viscera with fertile cysts (Moro and Schantz 2009).

Man can be infected by direct contact to dogs or indirectly through contaminated food, water and infected objects. Dogs may carry eggs on the body surface and a

person can become infected by touching the animal. Close contact with dogs and lack of hygiene are important factors for transmission. Another important source of human infection is through vegetables and water contaminated with eggs. Ingestion of infected flies may also transmit the infection (Lawson and Gemmell 1990). However, direct transmission from small ruminants to man has not been observed (Moro and Schantz 2009; Smith and Sherman 2009).

3.2.4.7 Diagnosis in Small Ruminants

Numerous tests have been developed for the diagnosis in humans although few reliable serological tests are available for small ruminants. Various imaging techniques can be used to identify the hydatid cysts, but post mortem examination is still the most reliable method for diagnosis in intermediate hosts (Moro and Schantz 2009; Smith and Sherman 2009).

3.2.4.8 Treatment and Control

The main control measurement is to interrupt the transmission cycle from the intermediate to the definite host. The infection cycle would be halted if dogs lack access to the viscera of intermediate hosts. In addition, the number of dogs might be reduced or treated with efficient anthelmintics. Treatment of infected sheep/goat in order to stop the infectivity of the cysts is not yet possible. Recombinant vaccines have been developed both for sheep and dogs with promising results (Lightowlers et al. 1999; Zhang and McManus 2008).

Oncospheres have little resistance to desiccation and high temperature, however they may survive in water/damp sand for 225 days at 6 °C (Lawson and Gemmell 1983). Hygiene is important to prevent human infection, as eggs may be swallowed with uncooked vegetables contaminated with dog faeces or from fingers contaminated from soil or the fur of an infected dog. Close contact with possibly infected dogs should therefore be avoided. Early diagnosis in human is important to avoid complications and rupture of the cysts. Surgery was earlier the traditional approach for treatment in humans, but anthelmintics, percutaneous procedures and a watch-and-wait approach are now more commonly used (Brunetti and White 2012).

3.2.5 Leptospirosis

3.2.5.1 The Pathogen

Leptospirosis is caused by helical Gram-negative organisms of the family *Leptospiracea* and the genus *Leptospira*. More than 250 serovariants have been detected (Cerqueira and Pichardeau 2009). The main serovariants infecting small ruminants are *L. borgpetersenii* serovar Hardjo-bovis and *L. interrogans* serovar Pomona.

However, the importance of sheep as a maintenance host of serovar Hardjo-bovis has yet to be unravelled (West et al. 2009).

3.2.5.2 Occurrence

Leptospira involving small ruminants have a worldwide distribution.

3.2.5.3 Hosts

Several hosts are involved, including cattle and swine.

3.2.5.4 Disease in Small Ruminants

There are several serovariants of *L. interrogans* serovar Pomona. Virulence of the strains varies, whereas the majority of leptospiral infections in small ruminants are subclinical. However, septicaemia, depression, anorexia, and in some cases haematuria may occur. Severe illness is characterized by jaundice, haematuria and haemoglobinuria, which may develop to a fatal outcome. Abortion may also occur (Smith and Sherman 2009; West et al. 2009).

Sheep could be infected with *L.* Hardjo-bovis, but are usually asymptomatic and studies indicate that sheep are only transiently infected with this serovar (West et al. 2009). In addition, *L. grippotyphosa*, *L. icterohemorrhagiae* and *L. serjoe* have been involved in clinical leptospirosis in goats (Smith and Sherman 2009).

3.2.5.5 Disease in Humans

Human disease varies widely according to the serovar of *Leptospira* involved. The incubation period varies from 2 to 30 days. In the acute febrile stage, the clinical symptoms are related to a generalized vasculitis, such as severe headache, muscle pain, conjunctival suffusion, rash, and photophobia. Intrauterine infection and fetal death may occur in pregnant women. The infection may proceed to aseptic meningitis and renal failure (Ellis 1998).

3.2.5.6 Transmission (Small Ruminants-Human)

Leptospires persist in the kidney and genital tracks of carrier animals and are excreted in urine and genital fluids. Survival outside the host is favoured by warm and moist conditions. Transmission is mainly due to direct or indirect contact with persistently infected animals and occurs through contact with infected urine, products of abortion, handling of infected kidneys, and ingestion of infected milk. Leptospires gain access to the host mainly through mucous membranes, and abraded and water-softened skin (Ellis 1998).

3.2.5.7 Diagnosis in Small Ruminants

The diagnosis is based on laboratory confirmation, such as PCR analyses of blood, CSF or tissue biopsy, and serology (such as Microscopic agglutionation test (MAT) and ELISA). Leptospires in the urine is, however, not a common feature of serovar Pomona infection in sheep (West et al. 2009). No reliable method exists for detection of carrier animals.

3.2.5.8 Treatment and Control

Leptospira are important pathogens in developing countries, where poor work and living conditions increase the opportunity for transmission from animals to man. The infection often occurs after heavy rainfall, when surface water accumulates in the paddocks. Clinical cases should be treated with antibiotics (West et al. 2009). Vaccines based on killed whole leptospiral cells have been available for several years. Recent vaccine developments based on recombinant proteins showed promising results (Yan et al. 2010; Félix et al. 2011).

In order to avoid the spread of the infection, infected animals should be identified and contact with carrier animals should be minimized. Prevention should be based on environmental control, such as rodent control, elimination of standing water, and avoidance of damp bedding. In addition, contact with infected herds and import of infected animals should be avoided. In order to prevent the human infections, common water sources or potentially contaminated water supplies should be restricted. Farmers, milkers, slaughterhouse and meat-processing workers as well as veterinarians have an increased risk for exposure (Dorjee et al. 2008; Smith and Sherman 2009).

3.2.6 Listeriosis

3.2.6.1 The Pathogen

Listeria monocytogenes is a Gram-positive coccobacillus within the genus *Listeria*. At least 16 serotypes with numerous subtypes of *L. monocytogenes* exist. *L. ivanovii* may occasionally cause abortion in small ruminants, but this bacterium has not yet been associated with human disease (Smith and Sherman 2009).

3.2.6.2 Occurrence

L. monocytogenes is ubiquitious in the environment.

3.2.6.3 Hosts

Several animals including small ruminants can be infected with *L. monocytogenes*. The natural reservoir appears to be the mammalian gastrointestinal tract. Grazing animals will ingest the bacteria and further contaminate vegetation and soil (Scott 2007).

3.2.6.4 Disease in Small Ruminants

There are mainly six manifestations of the disease: abortion, septicaemia, encephalitis, diarrhoea, mastitis and ocular infections. Clinical manifestations vary according to the route of infection. *L. monocytogenes* often affects the pregnant uterus and the central nervous system. During pregnancy, infection spreads to the fetus, which will either be born severely ill or die *in utero* (Scott 2007).

Listeriosis is one of the most common neurological diseases in adult sheep. Sheep aged 18–24 months are often affected due to molar teeth eruption, which may facilitate infection. Lesions are normally localized in the brainstem and clinical signs indicate unilateral dysfunction of the third to seventh cranial nerves. Facial nerve paralysis with dropping ear, muzzle pulled to one side, and lowered upper eyelids are typical symptoms. Profuse salivation and retained food material in the cheek is also typical. Keratoconjunctivitis and iritis may occur, in addition to partial paralysis of the pharynx. The clinical course in sheep and goats is often rapid, and death may occur 4–48 hours after onset of clinical symptoms (Scott 2007; Smith and Sherman 2009).

3.2.6.5 Disease in Humans

Systemic *L. monocytogenes* infection is a serious, but usually sporadic, invasive disease that primarily affects pregnant women, neonates, and immune-compromised persons (Cork and Checkley 2011). Infections can be treated successfully with antibiotics, but 20–40% of human cases are fatal (McLauchlin and Van der Mee-Marquit 1998).

The infective dose of *L. monocytogenes* is not known. The incubation period from food-borne infection varies widely from 3 up to 70 days, with a medium incubation period estimated to be around 3 weeks. There may be strain variation in pathogenicity, but this has to be unravelled more closely. Outbreaks of listeriosis are usually spread via the faecal-oral route, resulting in a self-limiting gastroenteritis in healthy persons. However, cutaneous infection has also been observed in people during deliveries of listeria-infected animals. During pregnancy, infection spreads to the fetus. In non-pregnant human, listeriosis usually presents as meningitis, encephalitis, or septicaemia in the immune-compromised and elderly (McLauchlin and Van der Mee-Marquit 1998; Swaminathan and Gerner-Smidt 2007; Cork 2011).

3.2.6.6 Transmission (Small Ruminants-Human)

Food-borne transmission of *L. monocytogenes* is the main route of infection, whereas unpasteurized dairy products are the main source of human infection. Other sources include uncooked food of animal origin and contaminated raw vegetables. *L. monocytogenes* may also be transmitted by direct contact with infected animals or animal products. In such cases, the disease occurs principally as papular or cutaneous lesions, usually on the arms or the wrist 1–4 days after attending a listeria-abortion. This manifestation, however, has mainly been seen after contact with cattle (McLauchlin and Van der Mee-Marquit 1998; Smith and Sherman 2009).

3.2.6.7 Diagnosis in Small Ruminants

Unilateral cranial nerve paralysis affecting the eye, eyelid, ear and lips with ataxia are typical for listeriosis. Samples from cerebrospinal fluid can support the diagnosis. At post mortem examination, histological lesions such as microabscesses and perivascular cuffing in the brainstem and medulla are pathognomonic of listeriosis. Aborted fetuses due to *L. monocytogenes* are usually autolytic with miliary necrotic foci scattered throughout the liver and spleen, while listeria-septicaemia is often accomplished by focal hepatic necrosis. Listeriosis, however, can only be confirmed by isolation or identification of *L. monocytogenes* (Low and Donachie 1991; Scott 2007).

3.2.6.8 Treatment and Control

Infection can be treated with antibiotics. The drug of choice is high-dosed penicillin. Supportive therapy including fluids and electrolytes are required for animals having difficulty eating and drinking (Scott 2007).

In an outbreak, affected animals should be segregated. In silage-fed ruminants, listeriosis is mainly a winter-spring disease and is normally seen in animals fed with poorly conserved silage. Outbreaks may occur within 10 days of feeding poor silage. Use of the particular roughage should be discontinued. However, due to an incubation period of 1–3 weeks, most of the *Listeria*-infected silage may not be longer available. Animal to animal transmission may occur via the faecal-oral route. A live attenuated vaccine for use in sheep has been developed, but the results from field trial vaccinations are equivocal (Scott 2007). However, new vaccine technologies seem promising in developing a protective immune response against *L. monocytogenes* (Carrasco-Martín et al. 2012; Kim et al. 2012; Lou and Cai 2012; Mohamed et al. 2012).

To avoid infection in humans, hygiene during food preparation and storage as well as avoidance of unpasteurized dairy products are preventive measures. However, *L. monocytogenes* can survive in soil or silage for more than 2 years. It is also found in excreta from apparently healthy animals, although carriage in the gut is

likely to be transitory. Control measures should be focused on avoiding *Listeria*-contaminated food, especially since the bacteria maintain to grow during refrigeration. Pregnant women and immune-comprised individuals are at increased risk for developing listeriosis (McLauchlin and Van der Mee-Marquet 1998).

3.2.7 Q-Fever (Coxiella Burnetii)

3.2.7.1 The Pathogen

Q-fever is caused by the intracellular organism *Coxiella burnetii* within the genus *Coxiella* and the order *Legionellales* (Seshadri et al. 2003). The organism exists in two different antigenic phases. In nature, *C. burnetii* exists in phase I form, which is virulent. However, when cultivated in non-immunocompetent cell cultures or hen eggs the organism mutates irreversibly to the phase II form which is less virulent (Quevedo Diaz and Lukacova 1998). *C. burnetii* has mainly two different morphologic forms, a large and a small form. In addition, an endospore-like structure is observed in the large form, which is highly resistant to environmental degradation, such as high temperatures, ultraviolet light and osmotic shock (Mearns 2007).

3.2.7.2 Occurrence

Q fever is a worldwide zoonosis that occurs in all geographic and climate zones, with the exception of Antarctica and possibly New Zealand (Hilbink et al. 1993; West et al. 2009). However, Q fever is not a reportable disease in many countries, so it is difficult to know exactly where it occurs.

3.2.7.3 Hosts

C. burnetii is able to infect many animal species including mammals, birds and several arthropods. However, cattle, sheep and goat seem to be the primary animal reservoirs for human infection (Maurin and Raoult 1999).

3.2.7.4 Disease in Small Ruminants

In animals, *C. burnetii* infections are generally asymptomatic, except for abortion, stillbirth and the delivery of weak offspring. However, *C. burnetii* may induce pneumonia, conjunctivitis and hepatitis (Arricau-Bouvery and Rodolakis 2005). High abortion rates are rarely observed, although abortion storms in some caprine herds have been described (Sanford et al. 1994). Aborted fetuses appear normal, but infected placentas exhibit intercotyledonary fibrous thickening and discoloured exudates that may be mineralized (Moore et al. 1991).

3.2.7.5 Disease in Humans

In humans, acute Q fever is rarely diagnosed, because of non-specific initial clinical signs, such as fever, pneumonia, headache and weakness. However, chronic infection may result in severe granulomatous hepatitis, osteomyelitis and valvular endocarditis. Chronic infection can manifest itself within a few months or even years after the acute infection (Fournier et al. 1998).

3.2.7.6 Transmission (Small Ruminants-Human)

Contaminated aerosols generated from desiccation of infected placentas, body fluids or dust from contaminated manure are the main sources of both animal and human infection, and the control of fecal excretion and placental bacterial discharge is essential (Arricau-Bouvery and Rodolakis 2005). Grazing contaminated pasture and tick bites are other modes of transmission. The organism is highly infectious, with an infective dose of 1–10 bacteria (Tigertt et al. 1961). Because *C. burnetii* is extremely resistant to desiccation and to physical and chemical agents, it survives in the environment for long periods. The endospore-like form survives in dust for 120 days, in tick faeces for 568 days and in wool for 12–16 months at 4–6 °C (Mearns 2007).

3.2.7.7 Diagnosis in Small Ruminants

Current alternatives to diagnose *C. burnetii* infection in ruminants include serological analysis, isolation by cell culture, live animal inoculation, immunohistochemical and PCR-based detection. In the acute phase of the infection, *C. burnetti* can be detected in lungs, spleen, liver and blood (Fournier et al. 1998; Maurin and Raoult 1999).

Placental smear or impression of placentas could be stained for instance by using a modified Ziehl-Nielsen procedure (Mearns 2007). Several serologic tests are available, such as complement fixation test, ELISA, and a fluorescent antibody test. However, carrier animals may also have an antibody titre increase in late pregnancy (Kovacova et al. 1998; Smith and Sherman 2009). For Q fever diagnostis, it has been recommended to use PCR and immunofluorescence tests of *Coxiella* on parturition products and vaginal secretions at abortion (Arricau Bouvery et al. 2003).

3.2.7.8 Treatment and Control

If Q fever is suspected, aborting animals and other animals in late pregnancy should be treated with tetracycline, although this treatment does not totally suppress the abortions and shedding of *C. burnetii* at lambing (Berri et al. 2005). Placentas and aborted fetuses should be destroyed properly and aborted animals should be

isolated. In addition, materials such as bedding and straw contaminated with birth fluids and other secretions from affected animals should be destroyed (Smith and Sherman 2009).

The spread of *C. burnetii* infection in domestic animals depends on many factors, such as population density of animals, the system of rearing and management at parturition. Because the environment can remain infected for a long time and many species can be carriers, test and cull strategies are not appropriate for infected herds (Smith and Sherman 2009). However, during the recent outbreak of Q fever in humans in the Netherlands, the Dutch Government decided to cull more than 50,000 pregnant ewes and goats in order to halter the worst outbreak of Q fever ever known where more than 4000 human cases have been recorded from 2007–2010. The reason for this strategy was that dairy goats were believed to be the main source of human infection (van der Hoek et al. 2012).

In animals, the uterus and mammary gland of females are sites for persistent *C. burnetii* infection. Reactivation of the bacterium during pregnancy results in shedding of a great amount of infectious agent into the environment during abortion or via birth fluids, placenta and fetal membranes (Sawyer et al. 1987). Over 10^9 bacteria per gram of placenta may be released at the time of delivery (Babudieri 1959). Studies indicate that ewes shed the bacterium mostly in feces and vaginal mucus, while in goats shedding in milk seems to be the most frequent route (Rodolakis et al. 2007; Rodolakis 2009).

In animals, the most effective vaccines are those composed of inactivated whole phase I bacteria. Bacterial shedding in placentas and milk was strongly reduced in experimental infection or in natural Q fever infection in ewes vaccinated with phase I vaccines (Sampere et al. 2003). Since phase I vaccine are dangerous to produce, a subunit vaccine is now being investigated (Arricau-Bouvery and Rodolakis 2005).

To prevent human infection, drinking raw milk or consumption of raw milk products should be restricted. For inactivation, pasteurization of milk at 62.8 °C for 30 min or at 71.7 °C for 15 s is required (Kazar 1999). Q fever often occurs as an occupational disease. Persons at particular risk are livestock handlers, processors of animal products, abattoir workers, those in contact with dairy products, veterinarians and laboratory personnel working with *C. burnetii*-infected animals (Maurin and Raoult 1999). In addition, it is necessary to inform vulnerable persons such as immunosuppressed patients or those suffering from cardiac valvopathy and pregnant women that they must avoid contact with animals during lambing and kidding (Arricau-Bouvery and Rodolakis 2005).

3.2.8 Rift Valley Fever (RVF)

3.2.8.1 The Pathogen

RVFV (Rift Valley fever virus) is a single-stranded RNA-virus in the genus *Phlebovirus* of the family *Bunyaviridae*.

3.2.8.2 Distribution

RVFV is mainly distributed in sub-Saharan Africa, but has also been identified in Northern Africa and on the Arabian Peninsula (Bath 2007).

3.2.8.3 Hosts

RVFV may cause infection in several ungulate species, although their importance as reservoir host has to be unravelled. Mosquito vectors, such as in the genus *Aedes*, may maintain the virus in endemic areas by transovarial transmission. Other insects, such as *Culex* species, may also be involved in epidemics (Bath 2007).

3.2.8.4 Disease in Small Ruminants

RVFV can infect a wide variety of tissues, such as liver, lymphoid and nervous tissue. The incubation period is short, as little as 12 h in young lambs and up to 72 h in adult sheep. High fever, anorexia, listlessness, and recumbence are common in young lambs. However, clinical signs are not always observed, since young animals may die rapidly. Mortality rate may exceed 90 % in lambs under 2 weeks old. Abortion is a common sign in adult animals, and this may occur at any time during pregnancy and reach up to 100 %. Infection in older animals is often subclinical (Bath 2007).

3.2.8.5 Disease in Humans

The largest recorded outbreak in humans was in 1997–98 in East Africa where approximately 89,000 human cases and 478 fatalities were recorded (CDCP 1998). Typical symptoms in humans are flu-like illness after a short incubation period of 2–6 days. Other symptoms are photophobia, retinitis, meningoencephalitis and haemorrhagic fever. The symptoms may be severe in patients with a pre-existing liver disease. Sequelae may include widespread haemorrhages, jaundice, shock, liver and kidney failure and death. Fatality rate is normally less than 1 %, but the death toll can mount to several hundreds in severe outbreaks (Swanepoel 1998; Bath 2007).

3.2.8.6 Transmission (Small Ruminants-Human)

The route of transmission in animals is via different mosquitoes. The virus has been isolated from more than 30 mosquito species. In addition, RVFV has also been isolated from flies and midges (*Culicoides*). Both biological and mechanical vector

transmission may occur. In Sub-Saharian areas the main vector seems to be mosquitoes within the genus *Aedes* (Bath 2007; Smith and Sherman 2009).

The main transmission route in humans is via direct or indirect exposure to infected blood, tissues or body fluids of infected animals, for instance in connection with slaughtering, butchering, obstetrical procedures or treatment of infected animals. Infection may also occur via vectors, aerosols, and consumption of unpasteurized milk. Direct person-to-person transmission has not been reported. Persons at risk are veterinarians, farmers, shepherds and abattoir workers (Swanepoel 1998; Smith and Sherman 2009).

3.2.8.7 Diagnosis in Small Ruminants

The diagnosis is based on clinical symptoms and post mortem examination. In young lambs, widespread haemorrhages and liver necrosis is often recorded. Disseminated intravascular coagulopathy may occur in several internal organs. Samples from spleen, liver and brain should be used for histological examination. The diagnosis, however, has to be verified by PCR, virus isolation, and serological investigations (such as ELISA and hemagglutination-inhibition test) (Bath 2007).

3.2.8.8 Treatment and Control

Outbreak of RVF occurs at irregular intervals. The virus may persist in a vector/natural host cycle and low level of virus activity is found between outbreaks. Infected eggs from *Aedes* species may survive in the soil for years. The single most important responsible factor for an outbreak of RVF is heavy rainfall and widespread flooding which favours multiplication of the vectors. Movement of infected animals or winds that transport infected mosquitoes over long distances may spread the disease to non-endemic areas (Sellers 1980). The development of early warning systems and surveillance in and around endemic areas in order to recognize animal and human cases as early as possible, are crucial in order to control the infection.

There is no treatment available for infected animals, since the disease is usually very acute and the lesions too severe. Control measures rely on the use of efficient vaccines. A live attenuated vaccine is available for non-pregnant animals, while an inactivated whole virus vaccine can be used for pregnant animals. The last vaccine requires a booster and annual revaccination. A recombinant vaccine has recently been developed, but it must be tested in appropriate animal models before used as a livestock and human vaccine (Indran and Ikegami 2012; Morrill et al. 2013). When handling suspicious animals, wearing of eye protection, protective clothing, gloves and masks should be mandatory (Swanepoel 1998).

3.2.9 Toxoplasmosis

3.2.9.1 The Pathogen

Toxoplasma gondii is a protozooan parasite within the family *Sarcocystiidae* and genus *Toxoplasma*. The lifecycle can be divided in two parts, a sexual cycle, restricted to enteroepithelial cells in cats and the production of oocysts, and an asexual cycle (forming tissue cysts) which occurs in a wide range of warm-blooded intermediate hosts. Six major clades of *T. gondii* have been characterized (Buxton and Rodger 2007; Su et al. 2012).

3.2.9.2 Occurrence

T. gondii has a worldwide distribution.

3.2.9.3 Hosts

Multiple intermediate hosts seem to exist, but the most important domestic hosts are pigs, sheep and goats. The final host is in the felid family.

3.2.9.4 Disease in Small Ruminants

Clinical toxoplasmosis causes abortion and neonatal mortality in small ruminants, especially in sheep. Mummified fetuses, stillborn or weak offspring are common features. However, infection in early pregnancy (<55 days) may result in death or expulsion of a small fetus. Clinical signs in aborting animals are usually not observed. Abortion is associated with primary infection during pregnancy in non-immune animals, and is most commonly seen in young animals. A long-lasting immunity develops following primary exposure and animals are unlikely to abort again due to toxoplasmosis (Buxton and Rodger 2007; Smith and Sherman 2009).

3.2.9.5 Disease in Humans

In most cases, toxoplasmosis in human is a disease with relatively mild and transient symptoms. However, primary infection during pregnancy may lead to intrauterine infection, and result in abortion or congenital lesions in the fetus. In addition, in patients with impaired immunity, *T. gondii* may lead to serious and even fatal infection (Dubey and Beattie 1988).

3.2.9.6 Transmission (Small Ruminants-Human)

The proportion of the human population infected with *T. gondii* depends on the age, area and environment. Most human infection appears to result either from exposure to oocysts from a contaminated environment or from ingestion of raw or lightly cooked meat containing tissue cysts. The most common way for infection from small ruminants to humans is by ingestion of tissue cysts. In addition, human infection through drinking of unpasteurized goat milk has been reported. A low risk may also apply when assisting infected animals at lambing or kidding. However, both these last modes of transmission are probably of low significance (Dubey and Beattie 1988; Smith 1991).

3.2.9.7 Diagnosis in Small Ruminants

Abortion due to *T. gondii* occurs mainly in young animals. Typical clinical signs of abortion result following infection in mid-gestation, with ewes and does producing stillborn and/or weekly offspring often accompanied by a mummified fetus. Cotyledons will also show characteristic lesions, such as white foci of necrosis 2–3 mm in diameter which may become mineralized. Diagnosis may include serology (such as Sabin-Feldman dye test, IFAT, MAT and ELISA), histology, immune-histochemistry and PCR methods (Buxton and Rodger 2007; Taylor et al. 2007; Smith and Sherman 2009).

3.2.9.8 Treatment and Control

Susceptible animal get infected by ingestion feed or water contaminated with oocysts. The oocysts are highly resistant and survive for a long period (>500 days) at room temperature in moist conditions. The main source of *Toxoplasma* infection in small ruminants are oocysts excreted from cats. Susceptible cats become infected with *T. gondii* after ingestion of tissue cysts from for instance small rodents and may excrete a large numbers of oocysts, which then sporulate and become infective within a few days and remain so for several months. Infected faeces will then contaminate beddings, stores of hay, concentrates, water supplies and pasture. It has been estimated that although <1 % of the cat population may excrete oocysts at any time, contamination of the environment is readily maintained (Dubey and Beattie 1988; Buxton and Rodger 2007).

During an outbreak of toxoplasma-abortion little can be done. Infected placentas and dead lambs or kids should be buried or disposed to prevent their ingestion by other animals. Animal to animal transmission during lambing or kidding does not appear to occur to any significant extent. More direct preventive measures include chemoprophylaxis, chemotherapy and vaccination. A live vaccine base on an attenuated strain of *T. gondii* has been developed for sheep (Buxton and Rodger 2007; Smith and Sherman 2009).

In humans, as already mentioned, the most common way for infection from small ruminants is by ingestion of raw or lightly cooked meat. Tissue cysts may be viable for the lifetime of infected sheep (Dubey and Beattie 1988). Treatment of meat by curing, smoking, freezing at −20 °C is usually sufficient to kill the encysted *T. gondii*. However, cysts can survive insufficient microwave cooking (Lundén and Uggla 1988). In a recent study, treatment of tissue cysts in infected sheep to prevent human exposure to meat-borne toxoplasmosis have shown promising results (Kul et al. 2013). Shepherds, veterinary surgeons, slaughterhouse staff and butchers are especially at risk for contracting infection from small ruminants.

3.3 Concluding Remarks

Only a limited number of topics are covered by this brief review and important issues such as differential diagnoses are not included or discussed. A correct and swift diagnosis is a prerequisite for proper treatment and control. This may not always be available due to long incubation periods, unspecific clinical symptoms and imprecise diagnostic tests. Some pathogens may survive unnoticed in animals or animal products for a long period of time. Anthrax in humans, for instance, has occurred when handling imported goat skins for drum making, skins contaminated with spores of *B. anthracis* (Anaraki et al. 2008).

Microbial transmission will always occur between species, but the risk of transmission can be reduced with proper hygiene, management, husbandry and prophylactic treatment. In this context, recent vaccine development against several zoonotic pathogens through genomic and proteomic approaches is promising.

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