Chapter 14
Sheep and Goat Pox

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Abstract Sheep pox (SPP) and goat pox (GTP) are viral diseases of sheep and goats caused by sheep pox virus (SPPV) and goat pox virus (GTPV), respectively. SPPV and GTPV belong to the genus *Capripoxvirus* of the family *Poxviridae*, together with lumpy skin disease virus (LSDV) of cattle. They have double-stranded DNA genomes of approximately 134–147 kbp. SPPV and GTPV are closely related to LSDV though they possess specific nucleotide differences suggesting distinct phylogeny. SPP and GTP are notifiable diseases to the World Organisation for Animal Health (OIE). They are highly contagious diseases: the viruses spread through direct contact with lesions or contaminated objects, feed, and wool. SPP and GTP are endemic in Africa (except southern Africa), central Asia, the Indian subcontinent, the Middle East, Turkey, Greece, and some eastern European countries. Clinically, the presence of nodular skin lesions, mostly around the mouth and perineum regions, is a typical sign of the disease. Capripoxviruses classification and their nomenclature have been mainly based on the affected host species, creating a challenge for isolates naming. For a more accurate naming, it is better to use molecular methods as support to identify and classify capripoxvirus isolates. Conventional and real-time PCR methods are available that could help with the simultaneous detection and genotyping of the viruses. SPPV and GTPV as well as LSDV cross-react serologically, making it difficult to differentiate them using serological methods. To prevent and control SPP and GTP, illegal animal movement restrictions and vaccination campaigns with adequate vaccines and sufficient vaccination coverage are two very effective measures. The development of a high-throughput serological assay (ELISA) with better sensitivity and specificity and the development of a safe and effective vaccine, which can support the differentiation of infected from vaccinated animals (DIVA), are highly required.

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History

Sheep pox virus (SPPV) and goat pox virus (GTPV) are members of the genus *Capripoxvirus* (CaPV), subfamily *Chordopoxvirinae*, of the *Poxviridae* family (Tulman et al. 2002; Andrew et al. 2012; OIE 2016). They have large, complex, double-stranded DNA genomes of approximately 134–147 kbp size with 147 putative genes which encode proteins. The genome has a conserved central region bounded by two identical inverted terminal repeats (ITR) at the ends (Tulman et al. 2002). They share a high degree of sequence homology, with 96% identity between SPPV and GTPV (Tulman et al. 2002). The viruses primarily affect sheep and goats causing sheep pox (SPP) and goat pox (GTP), respectively, which collectively constitute the most severe poxvirus infections of small ruminants. SPP and GTP are reportable animal diseases to OIE due to their potential for significant economic impact on small ruminant production industry (OIE 2016).

Sheep pox virus (SPPV) and goat pox virus (GTPV) share the genus *Capripoxvirus* with lumpy skin disease virus (LSDV) which is closely related, though they possess specific nucleotide differences suggesting that they are phylogenetically distinct (Tulman et al. 2002; Lamien et al. 2011a; Gelaye et al. 2015). SPPV and GTPV as well as LSDV cross-react serologically, making it difficult to differentiate them using serological methods (Diallo and Viljoen 2007). Capripoxviruses classification and their nomenclature have been mainly based on the affected host species, creating a challenge for isolates naming. Indeed if some GTPV and SPPV strains produce disease in only either sheep or goats, there are also cases of strains that can cause disease in both animal species.

SPP started in Central Asia and then spread to African countries (Hutyra et al. 1946), while GTP was first reported in Norway in 1879 by Hansen (Rafyi and Ramyar 1959). SPP appeared soon after human smallpox, and its history dates back to second century AD (Hutyra et al. 1946). African, Asian, and European Countries reported SPP through the mid-twentieth century. Both SPP and GTP have been eradicated from many developed countries, yet they are still present, and creating serious health and economic problem in Africa, north of the equator, through Asia, and occasionally spreading from Turkey into Greece (Murray et al. 1973; Kitching 2003; OIE 2016).

Geographic Distribution and Economic Impact

SPP and GTP are endemic in Africa (except for southern Africa), central Asia, the Indian subcontinent, the Middle East, and Turkey, causing high morbidity and mortality in susceptible sheep and goats (Kitching 2003; Bhanuprakash et al.

2005; Zro et al. 2014; OIE 2016). From time to time, Greece and some Eastern European countries reported SPP incursions, with reported outbreak cases from August 2013 until April 2014 (Oguzoglu et al. 2006; Verma et al. 2011; Yan et al. 2012; Zhou et al. 2012; EFSA 2014). Last SPP outbreak occurred in Greece mainland in February 2015; however, it reoccurred in Lesvos Island between December 2016 and December 2017 (European Commission 2017).

SPP and GTP may cause significant damage to wool and hide quality and decrease in mutton and milk production (Babiuk et al. 2008). The mortality rates of SPP and GTP range from 5 to 10% in local goat and sheep breeds in endemic areas. However, imported exotic breeds may display higher rates (OIE 2016). The morbidity rate of SPP and GTP can reach as high as 100% particularly in young lambs, kids, yearlings, and immunologically naïve sheep and goats in natural outbreaks. Factors like hosts (age, sex, breed, nutritional and immunological status), agent (strain, pathogenicity, virulence), harsh environment, poor management, feed scarcity, and inadequate veterinary services have a direct influence on the epidemiology of the diseases. The presence of SPP and GTP in a country limits the export of live animals and animal products to the global trade and also causes potential economic losses due to costs associated with disease control and eradication (Aparna et al. 2016; OIE 2016).

SPPV and GTPV are registered as animal bioterrorist agents by the United States Department of Agriculture, since they (1) produce high morbidity and mortality with sharp production losses, (2) expand quickly to reach wide areas within a few days or weeks, (3) create severe socioeconomic consequences due to the death of the afflicted animals, and (4) could considerably restrict the international trade of animals and animal products (Babiuk et al. 2008; Aparna et al. 2016). SPP and GTP are notifiable animal diseases to OIE (2016).

Epidemiology

Susceptible Hosts

SPP and GTP affect sheep and goats of all ages, both sexes and all breeds, yet are more common and severe in young and old animals (Aparna et al. 2016). Sheep and goats are the natural hosts for SPPV and GTPV, respectively (OIE 2016). Though SPPV and GTPV generally display a host preference/specific for either sheep or goats, some strains can infect and equally cause disease in both species or affect heterologous hosts (Bhanuprakash et al. 2006, 2010; Babiuk et al. 2009a; Lamien et al. 2011a; Gelaye et al. 2015). Unlike LSDV (Lamien et al. 2011a), there is no documented report on the existence of SPPV and GTPV in wild ruminants (Tuppurainen et al. 2015). Additionally, both viruses are considered nonhazardous to human health (OIE 2016).

Transmission

SPP and GTP are highly contagious diseases. The causal agents are transmitted from animals to animals through direct contact with lesions or contaminated objects, feed, and wool. Environmental contamination leads to the virus introduction into the skin wounds. Excreted viruses are detectable in the nasal secretions, milk, feces, and possibly urine from infected animals. The common practice of herding sheep and goats together in one barn at night in endemic countries provides adequate exposure for the circulation of the virus and its maintenance in an area. During an outbreak, the virus is probably transmitted among animals by the inhalation of virus-contaminated droplets. SPPV and GTPV can persist up to 3 months on the wool or hair after the onset of clinical signs and possibly for a prolonged period in the skin nodules and scabs (Bowden et al. 2008). SPPV and GTPV infections do not lead to a carrier stage in infected animals (Bhanuprakash et al. 2006, 2011). Wading of sheep and goat skin through bushes and thorny plants, like Acacia, to nibble leaves, damages the skin, facilitating disease transmission from infected to susceptible animals. Additionally, wounded areas are easily accessible to biting flies, which suck blood helping in transmitting the virus quickly.

Owing to the intentness of the skin, and the significant viral load in the lesions, vectors can spread the viruses indirectly through mechanical transmission. Experimental studies showed that *Stomoxys calcitrans* could transmit, mechanically, SPPV and GTPV (Kitching and Mellor 1986; Mellor et al. 1987). Pre-infected flies spread the virus to susceptible goats, and the virus remains alive for up to 4 days in some flies. The inherent resistance of the virus, the significant virus load in skin nodules of sick animals, and the involvement of vectors able to keep the virus alive for prolonged periods are the essential factors favoring mechanical transmission (Bhanuprakash et al. 2006). Similarly, Nigerian and Oman isolates of SPPV were successfully transmitted between sheep by *S. calcitrans*. In contrast, biting (*Mallophaga* species) and suckling lice (*Damalimia* species), sheep head flies (*Hydrotaea irritans*), and midges (*Culicoides nubeculosus*) fail to transmit the virus (Kitching and Mellor 1986).

The occurrence of global climate change could impact the further extent of these diseases into naive geographic regions due to the spread of insects (Aparna et al. 2016). The appearance of SPP and GTP in disease-free areas is predominantly associated with the illegal animal movement through trade, from infected to previously free regions (Domenech et al. 2006), as well as the lack of adequate or breakdown of veterinary services and regulatory policies (Rweyemamu et al. 2000).

Clinical Signs and Lesions

In natural circumstances, SPP and GTP have an incubation period of 8–14 days, following contacts between infected and susceptible animals. The infections can exhibit mild to severe clinical signs, depending on the immune status of the host and strain of the virus involved (Davies and Otema 1981). Both SPPV and GTPV have

tropism for skin, lung, and discrete sites within the mucosal surfaces of oro-nasal tissues and the gastrointestinal tract, and to a lesser extent, the lymphoid tissue (Bowden et al. 2008). Hence, the tropism of both viruses for the skin as well as minor involvement of liver and spleen suggests that the pathogenesis of capripox disease closely resembles smallpox and monkeypox diseases (Fenner 1988; Zaucha et al. 2001; Jahrling et al. 2004; Babiuk et al. 2008).

Some breeds of sheep perish during the acute infection without showing any skin lesion. In other breeds, the disease begins with an initial rise in rectal temperature to above 40 °C, followed by the development of macules—small circumscribed areas of hyperamia—within 2–5 days. Those macules, which are mainly detectable on unpigmented skin, will evolve into papules, hard swellings of between 0.5 and 1 cm in diameter, covering the body or restricted to the groin, axilla, and perineum.

During SPPV and GTPV infections, cell-associated viremia develops concurrently with the development of macules and papules in the skin of susceptible animals. The viremia persists until the host develops adequate antibodies against the virus (Kitching and Taylor 1985).

Rhinitis, conjunctivitis, and excessive salivation also occur throughout the infection. Pox lesions can widely spread, affecting over 50% of the skin surface. More commonly in enzootic areas, the lesions are restricted to a few nodules under the tail. Internal organs such as the lung and the stomach also develop characteristic pox-like lesions. Infected sheep and goats show fever, ocular and purulent nasal discharge, and cutaneous papules and nodules in areas of the skin with less hair, such as the head and the perineum as shown in Fig. 14.1.

The clinical signs and postmortem lesions vary considerably among breeds and depend on the strain of capripoxvirus (OIE 2016). Indigenous breeds are less susceptible and frequently present few lesions which could be confused with insect bites or contagious pustular dermatitis. However, naive lambs, animals kept isolated or brought into endemic areas, due to the stress of moving over long distances, are more susceptible, often showing generalized lesions and fatalities.

Clinically, it is challenging to distinguish SPPV infection from that of GTPV. Nevertheless, most strains of SPPV and GTPV display host preferences and produce

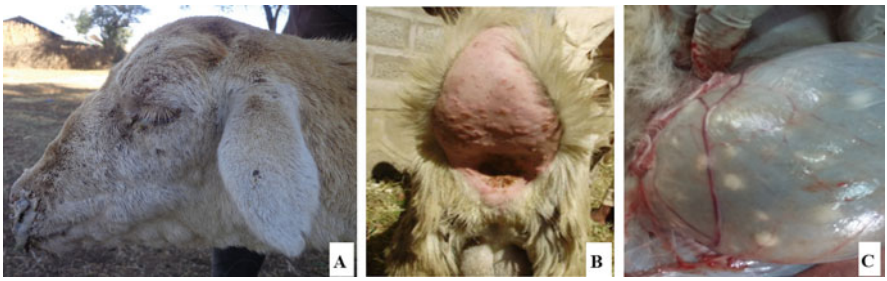

Fig. 14.1 Characteristic lesions of sheeppox observed during outbreak investigation. (**a**) Cutaneous nodular lesions around the face and dewlap area of the ewe with nasal secretion. (**b**) Severely affected male sheep with cutaneous popular and nodular lesions covering the perineum area. (**c**) Multifocal nodular lesion on the rumen mucosa of a sheep. *Courtesy © Esayas Gelaye*

Table 14.1 Evidence of cross infections by capripoxviruses

Strain name	Origin	Species of origin	Genotyping
GTPV Saudi Arabia/93	Saudi Arabia	Goat	SPPV[a]
SPPV OMAN/84	Oman	Sheep	GTPV[a]
SPPV KS-1	Kenya	Sheep	LSDV[a]
LSDV RSA 06 Springbok	South Africa	Springbok	LSDV[a]
LSDV RSA/00 OP126402	South Africa	Springbok	LSDV[a]
GTPV Nigeria goat vaccine	Nigeria	Goat	SPPV[a]
O58/2011	Kenya	Sheep	GTPV[un]
O59/2011	Kenya	Sheep	GTPV[un]
Akaki/2008	Ethiopia	Sheep	GTPV[b]
Metekel/2010	Ethiopia	Sheep	GTPV[b]
Chagni O06/2012	Ethiopia	Sheep	GTPV[b]

The genotyping of outbreak isolates revealed GTPVs collected from sheep in Ethiopia, Kenya, and Oman and SPPVs collected from goat in Saudi Arabia and Nigeria
[a]Lamien et al. (2011a)
[b]Gelaye et al. (2015)
un unpublished

more severe disease in the homologous host (Kitching et al. 1986; Bhanuprakash et al. 2006). Some studies also revealed natural infections, by either SPPV or GTPV, with similar severity and clinical signs in sheep and goats (Lamien et al. 2011a; Gelaye et al. 2015) as shown in Table 14.1.

Diagnosis

Clinical Observation

Clinically, the presence of nodular skin lesions mostly observed around the mouth and perineum regions is helpful to diagnose SPP and GTP (OIE 2016). In endemic areas and chronic cases, the nodular lesions develop scars, and sometimes, in severe situations, the nodules cover the entire body of the animal. Besides, owner's interview and disease history records such as affected host species, morbidity and mortality rates, month/season of disease occurrence, and vaccination history could be used as primary source of information to reach a diagnosis.

Sample Collection and Transport

Active nodular skin or postmortem lesions from skin papules, lung lesions, and lymph node are good samples for virus isolation and antigen detection (OIE 2016). It is advisable to collect samples within the first week following the occurrence of

clinical signs and lesions, before the rise of antibodies (Rao and Bandyopadhyay 2000; OIE 2016). Nasal, oral, and ocular swabs as well as saliva, from clinically diseased or suspected animals, are also good clinical samples for virus isolation and antigen detection (Bowden et al. 2008; Lamien et al. 2011a; Gelaye et al. 2015). Buffy coat obtained from blood collected in an anticoagulant medium through the viremia or within 4 days can be used for virus isolation (Bhanuprakash et al. 2006). However, poxviruses are mainly cell associated; consequently, there may be fewer virus particles in the blood. Representative skin lesions or swab samples from suspected or clinically diseased animals should be collected aseptically, using sterile and labeled containers. Serum samples can be collected from diseased or suspected animals for CaPV antibody detection or from vaccinated animals to evaluate the seroconversion.

It is essential to conduct urgently SPP and GTP diagnosis on representative suspected samples transported to the diagnostic laboratory through maintaining the cold chain. Samples are packed using triple packaging (primary, secondary, and outer containers) and carried following the regulations set for the transport of dangerous goods. Samples from SPP and GTP suspected outbreaks are classified as Infectious Substances Class B (Division 6.2) and must follow the International Air Transport Association (IATA) packing instruction P650, using the UN-approved packaging material. The packaging must bear the labels UN3373, Biological Substance, Category B: hazard for animals, not for human health. It is strictly forbidden to carry infectious substances as carry-on baggage, checked baggage, or in person.

Virus Isolation

Primary lamb kidney and testis cells are commonly used for the isolation and multiplication of infectious SPPV and GTPV (Plowright and Ferris 1958; Kalra and Sharma 1981; Bowden et al. 2008). However, the use of primary cells for virus isolation presents several disadvantages such as the need to continuously establish new cultures, cell lot variation, and contamination with extraneous agents (Babiuk et al. 2008). SPPV and GTPV can also be multiplied using established cell lines such as ESH-L cells (Lamien et al. 2011b; OIE 2016), Vero cells (Singh and Rai 1991; Prakash et al. 1994; Gelaye et al. 2015), MDBK cells (Pandey et al. 1985; Joshi et al. 1995), and OA3Ts cell lines (Babiuk et al. 2007). SGPV can also be propagated on the chorioallantoic membrane of embryonated chicken egg (Kalra and Sharma 1981; Babiuk et al. 2007; OIE 2016). In SPPV and GTPV endemic countries, a biosafety level-II grade laboratory is sufficient for handling clinical samples and for virus isolation, whereas in disease-free countries, virus isolation should be conducted inside biosafety level-III facilities. Infectious viruses induce the formation of distinct plaques with cytopathic effects (CPEs) characterized by elongated cells, ballooning, high refractility, rounding, intracytoplasmic inclusion bodies, plaque formation, and detachment from the tissue culture flask (Soman and Singh 1980). Characteristic poxvirus-induced CPEs can be observed, in infected cells within 7 days, using an

inverted microscope, although sometimes the procedure may need several blind passages (Diallo and Viljoen 2007; Lamien et al. 2011a; Gelaye et al. 2015; OIE 2016). Isolation of SPPV and GTPV can be further confirmed by immunostaining using anti-SPPV and GTPV serum (Gulbahar et al. 2006; Babiuk et al. 2007). Histopathology, immunohistochemistry, and electron microscopy examination of skin nodules are additional options for the SPP and GTP diagnosis (Gulbahar et al. 2006; Bowden et al. 2008).

Antibody Detection

Serological assays can only identify SPPV and GTPV as Capripoxviruses, without discriminating the two viruses from each other. Immunity against SPPV and GTPV is predominantly cell mediated, though humoral antibodies are also detectable. Virus neutralization test can be used to examine serum samples for antibodies in disease-suspected sheep and goats (OIE 2016) and seroconversion following vaccination. Even though virus neutralization is referred to as a gold standard in the OIE Manual for detecting anti-SGP antibodies, it is slow, labor intensive, and not sensitive and requires handling of live virus, which is often not permitted in disease-free countries (Babiuk et al. 2008; OIE 2016). A recombinant capripoxvirus, expressing the green fluorescent protein, has been evaluated for virus neutralization assay (Wallace et al. 2007). The results showed a decline in the time required for the detection of virus neutralization activity from 6 to 2 days (Wallace et al. 2007). Western blotting assays are specific and sensitive enough for virus detection; however, they are expensive and difficult to perform and interpret (Chand et al. 1994). Agar gel immunodiffusion tests are less specific, due to cross-reactivity with orf virus antibodies; consequently, they are not recommended for SGP diagnosis (OIE 2016). In the pursuit for high-throughput and specific serological test, researchers developed several ELISAs using capripoxvirus recombinant proteins for antibody detection (Carn et al. 1995; Heine et al. 1999; Bowden et al. 2009; Tian et al. 2010). For instance, an indirect ELISA, based on the recombinant mature virion envelop protein P32 expressed in *E.coli* and yeast (Bhanot et al. 2009), has been reportedly used for the detection of CaPV-specific antibodies sheep (Heine et al. 1999). However, difficulties for the expression and the instability of the recombinant antigens have compromised these assays.

Babiuk et al. (2009b) developed an indirect ELISA for the detection of antibodies to SPPV, GTPV, and LSDV using sucrose gradient-purified inactivated SPPV as coating antigen. This ELISA is suited for screening sera from all three host species; however, the viral antigen is difficult and expensive to produce in large quantities. Moreover, such an approach is not applicable in disease-free countries. Bowden et al. (2009) also developed an indirect ELISA for the detection of antibodies based on selected capripoxvirus antigenic recombinant virion core proteins. This assay performed well on sera collected from sheep and goat that were infected experimentally with virulent virus isolates; however, the test was unable to detect antibodies in sera from vaccinated sheep and goat. An indirect ELISA for the diagnosis of SPP

and GTP using two synthetic peptides corresponding to the major antigen P32 of capripoxvirus was also reported (Tian et al. 2010); however, this assay performed well only on sera from immunized sheep. Currently, in 2019, there is only one ELISA kit commercially available for the detection of antibodies against SPPV, GTPV, and LSDV (ID Screen® Capripox Double Antigen Multi-species, IDVet, France), even though many development activities are ongoing elsewhere. With the recent spread of capripox diseases into new geographical areas, there is an urgent need for a high-throughput serological test to facilitate the serological surveillance of capripox in countries under threat. The availability of such a test will also facilitate animal screening during live animal export and post-vaccination monitoring during vaccination campaigns.

Nucleic Acid Detection

Various molecular techniques (conventional and real-time PCR) are available for specific and sensitive detection and differentiation of capripoxviruses such as SPPV and GTPV (Verma et al. 2011; Venkatesan et al. 2014). A gel-based PCR, for the generic detection of capripoxviruses, was described by Ireland and Binepal (1998). Similarly, a highly sensitive multiplex conventional PCR method is available for the detection and differentiation of SPPV, GTPV, and orf virus in clinical samples. This assay targets the DNA binding phosphoprotein (I3L) coding gene of capripoxviruses and DNA polymerase (E9L) gene of orf virus (Venkatesan et al. 2014). Another gel-based PCR method targets the 30 kDa RNA polymerase subunit (RPO30) gene (Lamien et al. 2011a) to detect capripoxvirus and differentiate SPPV from GTPV. However, this method is unable to differentiate GTPV from LSDV. Several real-time PCR methods (Balinsky et al. 2008; Bowden et al. 2008; Stubbs et al. 2012; Haegeman et al. 2013) are available for the generic detection of capripoxviruses; however, they do not intend to differentiate SPPV from GTPV and LSDV. Two LAMP PCR methods (Das et al. 2012; Murray et al. 2013) and a field-ready nucleic acid extraction and real-time PCR platform (Amson et al. 2015) are also reportedly used for the generic detection of capripoxviruses. A dual hybridization probe assay, targeting the G-protein-coupled chemokine receptor (GPCR) gene (Lamien et al. 2011b), and a snapback assay targeting the RPO30 gene (Gelaye et al. 2013) offer the possibility to simultaneously detect the three capripoxviruses and differentiate SPPV from GTPV and LSDV. In addition to the above real-time PCR-based methods, the RPO30 and GPCR genes could be sequenced for the phylogenetic classification of the capripoxvirus isolates (Le Goff et al. 2009; Lamien et al. 2011a; Gelaye et al. 2015). Similarly, a sequencing-based method reported by Hosamani et al. (2004) targets the P32 gene for capripoxvirus differentiation. Both conventional and real-time PCR are useful molecular tools for active clinical surveillance of capripoxviruses in an endemic situation, or newly affected countries and regions.

Differential Diagnosis

Diseases that can be confused with SPP and GTP are bluetongue (caused by bluetongue virus), peste des petits ruminants (caused by peste des petits ruminants virus) for respiratory symptoms, contagious ecthyma (caused by orf virus causing proliferative pox lesions on the muzzle and eyes), insect bites, and mange infestation (e.g., psoroptic mange/sheep scab) (Rao and Bandyopadhyay 2000; Bhanuprakash et al. 2006; OIE 2016). They cause similar kind of skin lesions, in affected hosts requiring a differential diagnosis from SPP and GTP. Hence, laboratory confirmation using conventional methods including antigen or antibody-based tests and molecular diagnostic techniques is necessary to confirm the cause of the diseases or outbreaks. Recently, a real-time PCR method able to simultaneously detect and differentiate SPPV and GTPV from other poxviruses affecting sheep and goats such as orf virus has been developed (Gelaye et al. 2017).

Prevention and Control

The immunity developed against poxvirus infection is predominantly cell mediated; thus, the immune status of animals does not correlate with neutralizing antibody titers in serum (Carn 1993). Previous exposure to SPPV and GTPV results in substantial and long-lasting protective immunity against subsequent reinfection with the virus. Live attenuated and inactivated strains of SPPV or GTPV are the most common vaccines in disease-endemic countries. There is only a partial cross-protection when sheep and goat are vaccinated with GTPV vaccine against SPPV and or vice versa (Kitching et al. 1987; Hosamani et al. 2004; Bhanuprakash et al. 2012). In disease-endemic countries, vaccination of small ruminants using a vaccine containing a virus homologous to the circulating isolates is an economical and sustainable means of disease prevention and control (Bhanuprakash et al. 2012; Hosamani et al. 2008). Consequently, better protection against locally prevalent strain for either SPPV or GTPV is achieved using homologous vaccines (Rao and Bandyopadhyay 2000; Bhanuprakash et al. 2005). Generally, inactivated vaccines do not provide adequate and long-lasting protective immunity; however, an inactivated SPPV vaccine would provide a safe and valuable tool to protect sheep and goat against SPPV and GTPV infections, particularly in the case of a first incursion of the virus in the previously disease-free area, or for preventive vaccination in region threatened by SPP or GTP (Boumart et al. 2016). For instance, inactivated SPPV vaccine produced using the Roumanian Fanar (RF) strain showed potential to replace live attenuated vaccine for the prevention and control of SPP in disease-endemic or disease-free countries (Boumart et al. 2016).

LSDV-derived vaccines are also widely used for the prevention and control of SPP and GTP. A single vaccine, through intradermal or subcutaneous route using the OIE recommended dose ($10^{2.5}$ $TCID_{50}$), using the Kenyan sheep and goat pox virus

(O180/KS-1 or O240) strain, protected both sheep and goats against the virulent strains of SPPV and GTPV. This vaccine, used in many countries in the Middle East and Africa, presented proper safety and protection result, though the vaccinal strain is, in fact, an LSDV (Gelaye et al. 2015; OIE 2016). Several recombinant vectored vaccines have been developed based on capripoxvirus. A recombinant capripoxvirus vaccine harboring the F or H genes of PPR virus (rCPV-PPR) provided adequate protection against both capripoxvirus and PPR virus (Diallo et al. 2002; Berhe et al. 2003; Caufour et al. 2014). Currently, a vaccine allowing the differentiation of infected from vaccinated animals (DIVA) is not commercially available against SPP and GTP.

In general, SPPV and GTPV infections are self-limiting; treatment with antibacterial and antifungal against secondary bacterial or fungal infections and supportive care help to improve the health status of infected animals (Smith et al. 2010). SPP and GTP are notifiable diseases as recommended by OIE; therefore, it is mandatory to notify suspicion of infection to the appropriate veterinary authorities and strictly regulate the movements of small ruminants. To implement an effective prevention and control strategy, proper veterinary services with moderately equipped resources, adequate infrastructure and logistic support, appropriate disease surveillance, and diagnostic activities are essential. It is also critical, achieving good vaccinations coverage using effective vaccines to build the herd immunity (at least 80% coverage), controlling the illegal animal movements and animal products. Political stability and economic development are also essential factors for the implementation of effective prevention and successful control strategy. Countries that do not report the occurrence of SPP and GTP diseases should strictly implement testing of animal and animal products before importing from disease-affected countries.

Conclusion

SPP and GTP are transboundary and OIE notifiable small ruminant diseases of low- and middle-income countries. SPP and GTP incidences have steadily increased in new geographical areas of South East Asia and Europe. The primary sources of virus spread into disease-free countries are the trade of infected animals and animal products such as wool and hides and the movement of people and goods due to civil war and unrest. The socioeconomic impact of SPP and GTP in the agricultural development and the livelihood of the small ruminant holders should be studied in endemic countries to support for the design of prevention and control strategies, to allocate resources, and to draw policy maker's attention. The implementation of well-organized vaccination campaigns, based on effective vaccines and achieving sufficient coverage, can help to reduce the burden of SPPV and GTPV infections in disease-endemic regions. Monitoring by active surveillance and the genotyping of outbreak viruses can guide for the selection of the most appropriate vaccines. The development of a high-throughput serological test, with enhanced sensitivity and

specificity, and the availability of a safe and effective vaccine allowing for the differentiation of infected from vaccinated animals (DIVA) are highly required.

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