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Review

Morbillivirus vaccines: Recent successes and future hopes

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ABSTRACT

The impact of morbilliviruses on both human and animal populations is well documented in the history of mankind. Indeed, prior to the development of vaccines for these diseases, morbilliviruses plagued both humans and their livestock that were heavily relied upon for food and motor power within communities. Measles virus (MeV) was responsible for the death of millions of people annually across the world and those fortunate enough to escape the disease often faced starvation where their livestock had died following infection with rinderpest virus (RPV) or peste des petits ruminants virus (PPRV). Canine distemper virus has affected dog populations for centuries and in the past few decades appears to have jumped species, now causing disease in a number of non-canid species, some of which are been pushed to the brink of extinction by the virus. During the age of vaccination, the introduction and successful application of vaccines against rinderpest and measles has led to the eradication of the former and the greater control of the latter. Vaccines against PPR and canine distemper have also been generated; however, the diseases still pose a threat to susceptible species. Here we review the currently available vaccines against these four morbilliviruses and discuss the prospects for the development of new generation vaccines.

1. Introduction

Morbilliviruses form a separate genus within the *Paramyxovirinae* sub-family of the *Paramyxoviridae* family, Order *Mononegavirales*. Currently there are six viruses classified within the morbillivirus genus: rinderpest virus (RPV), measles virus (MeV), canine distemper virus (CDV), peste des petits ruminants (PPRV), phocine distemper virus (PDV) and cetacean morbillivirus (CeMV). Until recently, rinderpest was considered the archetypal morbillivirus with a predicted existence of 9000 years [1]. In 2010, however, it was proposed that rinderpest and measles diverged from the same ancestral virus in more recent times during the 11th or 12th century [2]. MeV caused millions of human deaths annually before vaccines became available in the early 1960s. CDV, which affects many terrestrial and marine carnivores and PPRV which infects small ruminants and some large ruminants, can be as deadly to those they infect as rinderpest was to large bovids. Two other morbilliviruses, phocine distemper virus (PDV), affecting seals and other pinnipeds and cetacean morbillivirus (CeMV), threatening dolphins and porpoises, have also caused mass die-offs of these marine species.

All viruses within the Order *Mononegavirales* contain a negative-sense single-stranded RNA genome that consists of six open reading frames encoding six structural and two non-structural proteins. The nucleocapsid (N), phosphoprotein (P) and large polymerase protein (L), in tandem with the viral RNA, form the ribonucleoprotein complex (RNP). The matrix (M) protein forms a link between the RNP and the host cell derived plasma membrane, covered evenly with distinctive spikes of the viral glycoproteins, the haemagglutinin (H) and fusion proteins (F). The interaction between the H and F proteins governs the virus entry into a host cell. Here we review existing vaccines for morbilliviruses and discuss ideas for future vaccine development and potential eradication.

2. Rinderpest virus: the template for morbillivirus eradication

Early experiments with rinderpest virus (RPV) shifted a focus in animal disease management that altered the direction of veterinary science and had important ramifications for medical science. Indeed, repeated attempts to cure rinderpest virus led to the observation that serum obtained from recovered individuals was protective when administered to naïve animals that were subsequently exposed. This altered the scientific emphasis from treatment to prevention and heralded the new dawn of vaccinology. For RPV, early techniques aimed at preventing disease involved

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the inoculation of both virus and immune serum from convalescent animals and the preparation of hyperimmune serum in goats [3]. This was progressed with the application of immune serum, given either alone or in combination with infected blood where Robert Koch demonstrated that the combination of immune serum with virulent blood induced an active immunity. This approach was termed the "serum-virus simultaneous" method and was applied widely across Africa and India to protect livestock against rinderpest. Whilst highly effective the method suffered drawbacks including the induction of disease in both young and immunosuppressed animals (including pregnant animals), the infectious nature of vaccinees, instability of the preparations and the potential for transmission of piroplasms present within the inoculated preparation.

The next stage in vaccine generation against RPV involved the development of inactivated RPV vaccines. Early inactivated vaccine preparations were produced from infected bovine tissues that were chemically inactivated. Field application of inactivated RPV vaccines successfully cleared RPV from several countries including Iran, the Philippines, Sri Lanka, Thailand and Russia [4]. However, the immunity produced by these inactivated vaccines was short lived and as such, live attenuated vaccine strains were developed following multiple passage in different hosts. The first were produced in goats, and were cheap, efficacious, did not transmit piroplasms and one of them, the Kabete Attenuated Goat (KAG) vaccine was shown to induce a long lived neutralising antibody response to RPV [5,6]. A lapinized version of the RPV vaccine was developed in Korea and Japan, due to a shortage of goats, and was found to be better suited to Asiatic breeds of cattle [7]. Limitations in the number of doses produced in a single rabbit led to further passage in goats and then sheep, with the final product being used to eradicate RPV from China [4]. In Japan and Korea a vaccine was also generated *in ovo* to overcome adverse reactions seen with the lapinized vaccine in highly susceptible cattle. This development of attenuated vaccines in different host species reduced the labour required for mass production of vaccines but it was not until the development of tissue culture based vaccines that mass production at an economically viable cost could be achieved.

Early attempts to passage attenuate viruses in tissue culture were hampered by a lack of susceptible cell line for virus culture. Attempts using adapted strains of rinderpest in chicken embryo fibroblasts (CEFs), bovine kidney (BK) cells or bovine embryonic kidney cells were unsuccessful. Then, during the late 1950s [8,9], a virulent strain of RPV was successfully attenuated in primary calf kidney cultures. Initial passage resulted in an increased virulence but, following further passage, virulence was reduced until the virus was deemed to be completely attenuated and unable to cause disease even in the most susceptible breeds of cattle [10]. This vaccine, termed the tissue culture rinderpest vaccine (TCRV), was able to elicit a long term neutralising antibody response with protection from challenge several years post-vaccination without causing any adverse reactions.

The TCRV was used extensively across the developing World to vaccinate cattle against RPV and proved to be a highly effective tool in the eradication of rinderpest. There was, however, one drawback related to the TCRV: the serological signature of vaccinated animals was identical to that developed by animals infected naturally in the field that had survived infection. Since there was only one serotype of RPV, this meant that the OIE "gold standard" competitive ELISA based on an anti-H monoclonal antibody, was unable to fulfil 'DIVA' requirements – the ability to differentiate between the serological signature of naturally infected and vaccinated animals. Had the Global Rinderpest Eradication Programme not been successful then several candidate DIVA vaccines that had been developed may have found utility. Recombinant RPV vaccines that expressed foreign genes, such as GFP and HA, were developed although the serological response to the foreign genes was not sufficient [11]. A further development was a recombinant RPV that had the RPV N protein swapped with that from the closely related PPRV. Cattle vaccinated with this recombinant vaccine were protected from RPV challenge, and a companion ELISA test was developed to accompany the vaccine, enabling DIVA, although the successful eradication of RPV precluded its use [12]. A further alternative approach that showed promise for vaccine development for morbilliviruses in general was that of negative marking of vaccines by epitope deletion [13]. Such novel DIVA initiatives may find utility in the development of a DIVA vaccine for other morbilliviruses.

The successful global eradication of smallpox announced in 1980 was the incentive for OIE and FAO to examine the feasibility of setting the same goal for rinderpest by the year 2010 [14]. Several factors related to the virulence, pathology and epidemiology of rinderpest were recognised as favouring the Global Rinderpest Eradication Plan (GREP), including the limited geographical distribution of the disease, no latency or persistence of the virus in infected animals, the short infectious period and the requirement for direct or close indirect contact for transmission of the virus. The availability of the TCRV and highly sensitive and specific companion diagnostic tests was crucial for eradication campaign [14]. The economic significance of large ruminants across the developing world gave political and economic impetus to drive the eradication campaign to completion [3,15]. Finally in the year 2011, after a long campaign launched in 1994, the OIE announced RPV as only the second pathogen successfully eradicated from the world by human effort [16].

3. Measles virus

The development of vaccines against measles was facilitated by the isolation of the virus from human and monkey renal cells exposed to whole blood and throat washings obtained from patients infected with measles [17,18]. The cultivation of isolated viral material in chick embryo fibroblasts (CEFs) led to the generation of the first attenuated measles vaccine, the Edmonston-B strain [19], which was licensed for use in 1963. Although this vaccine was effective in preventing measles infection, it had to be administered with human gamma globulin as it commonly caused adverse reactions in vaccinees including fever and rash. In an attempt to ameliorate these side effects, studies were carried out on the development of an inactivated virus vaccine but the new formulation not only offered no protection against the disease, but also caused an atypical form of measles in those patients, who were exposed to wild-type virus post-vaccination [20,21]. In 1965, Maurice Hilleman propagated the Edmonston-B vaccine strain for a further forty passages in CEF cells to increase attenuation of the virus resulting in the generation of the Moraten strain (More Attenuated Enders) [22]. This new live attenuated version did not cause the side effects which accompanied the Enders vaccine, but was equally effective and as such it was licensed for human vaccination in 1968. In 1971 Stokes et al. published the results of studies on the trivalent vaccine against measles, mumps and rubella viruses [23]. The vaccine, known as the MMR, is a cocktail of three live attenuated viruses and has been shown to protect 96%, 95% and 94% of vaccinated individuals from measles, mumps and rubella, respectively. Since being licensed it has been used to vaccinate over 600 million people in over 60 countries across the world. It was originally administered as one-dose vaccine, but in 1989 a second dose was introduced to produce immunity high enough to disrupt measles transmission in a vaccinated population [24,25].

In 1998 a link between the MMR vaccine and the occurrence of autism and bowel disease was made by Wakefield et al. based on a study involving twelve children [26]. The publication sparked a

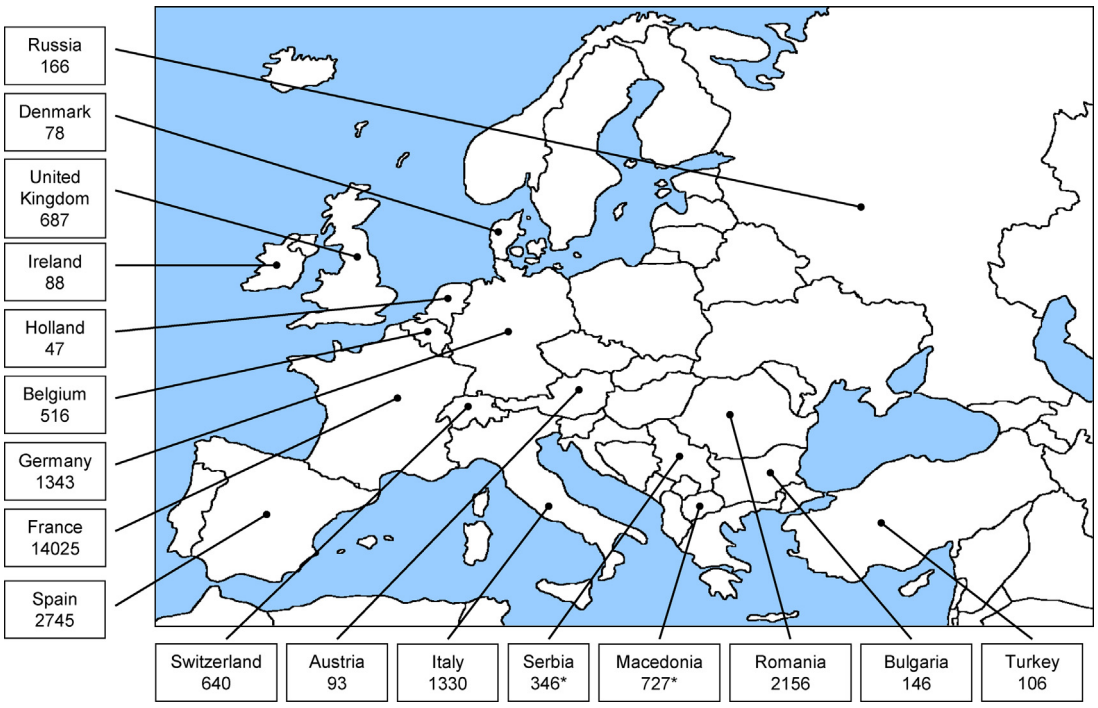

Fig. 1. Suspected measles cases reported by selected member states of the WHO European Region between January and August 2011 (33). During this period, a total number of 26,025 suspected measles cases were reported. The figure equals a nearly threefold increase compared to the same period in 2007. *Accumulated number of cases from 2010 and 2011.

lasting controversy despite the fact that subsequent reports extensively discredited the initial data [27,28]. Although the Wakefield publication has been fully retracted on both ethical and methodological grounds [29], the article and the public concern caused by the accompanying press coverage had a profound effect on the uptake of MMR vaccine [30]. A study on vaccine uptake in UK revealed that a conscious parental choice was a reason for 75% parents refusing to vaccinate their children with MMR, with the safety concerns being in part the legacy of the original publication [31,32]. A significant impact of this has been the re-emergence of measles virus in populations across Europe where it was previously absent, following a drop in immunity afforded where vaccination coverage drops below a level required to prevent virus spread within a community [33] (Fig. 1). In the UK, where the MMR controversy originally started, the measles vaccination coverage has also dropped and consequently a significant increase in the disease cases has been observed [30]. Between April and June 1995, 92.5% of children received their first dose of MMR before 2 years of age, compared to only 78.9% in the same period in 2003 [34,35]. The importance of vaccination was shown by a study summarising measles incidents in five European countries in which the majority of cases were unvaccinated or incompletely vaccinated children [36].

Despite the public concerns, MMR is regarded in the scientific community as one of the safest vaccines available. Moreover, measles virus possesses several features which make it an ideal candidate for recombinant vaccine development. The development of reverse genetics techniques has made manipulation and subsequent generation of recombinant live viruses possible and these tools have been extensively utilised for the morbilliviruses. Contrary to what might have been expected of an RNA virus, MeV recombinants are genetically stable and have been shown to not revert back to the virulent phenotypes. The risk of accumulation of mutations in neutralising epitopes in order to escape the protective immune responses conferred by vaccine strain could be theoretically expected, but despite high degree of mutations in RNA viruses, no such event has ever been observed not only in measles [37], but also in other morbilliviruses, such as RPV and PPRV. These assets along with the capability to accept up to 6 kb of foreign sequences, induction of both humoral and cellular immune responses, and the good safety record of vaccine strains and their low production costs have made MeV a vector of choice for the development of vaccines against numerous foreign pathogens and recombinant oncolytic viruses [38]. To date, surface proteins of several heterologous viruses have been expressed in MeV backbone and the immunogenicity of the foreign proteins examined in susceptible hosts (Table 1 and references therein).

The successful global elimination of rinderpest virus has highlighted the feasibility of measles eradication. Several factors related to the biology of the virus and the characteristics of the current vaccine favour the eradication idea. The measles virus, like all other morbilliviruses, is antigenically monotypic, which means that seroconversion after recovery from the disease or successful vaccination protects from all strains of the virus. There are no known animal reservoirs of the virus and where infected, susceptible non-human primates are considered “dead end” hosts due to insufficient population size [49]. Therefore the risk of “spill over” events is unlikely to hamper control efforts. For MeV, licensed vaccines are safe and can be administered to immunocompromised individuals safely with the two doses of vaccine, administered around 9–15 months and then 4–5 years of age, being able to induce protection for at least several decades if not for a life time [50]. The existing diagnostic assays are of great utility although point-of-care tests would improve diagnostic potential, similar to those being developed for ruminant morbilliviruses [51]. Limitations to MeV vaccines do, however, exist including the potential for interference from maternal antibodies in young children preventing a strong protective response [49]. This factor has led to the re-emergence of MeV in highly vaccinated populations [52]. Efforts to overcome the poor paediatric vaccine efficacy by using high titre vaccine were attempted but were stopped when mortalities were reported [53]. DNA vaccines have been proposed as an alternative vaccination

Table 1
Selected recombinant viruses generated in recent years with the use of MeV replication complex [39–48].

Foreign pathogen expressed	Animal model	Effect of modification	Reference
Nipah virus (NiV) glycoprotein	Hamster, African green monkey	Protection of both animal species against NiV challenge	Yoneda et al. [39]
Combination of hepatitis C virus (HCV) structural proteins	Mouse	Induction of neutralising antibodies to MV and HCV	Reyes-del Valle et al. [40]
Fusion protein consisting of human immunodeficiency virus 1 (HIV-1) Clade B p17, p24, RT and Nef antigens	Cynomolgus macaque	T cell response to the fusion protein in 6 out of 8 vaccinated animals	Stebbings et al. [41]
Secreted form of the envelope glycoprotein of West Nile virus (WNV)	Squirrel monkey	Induction of neutralising antibodies to WNV; reduced viraemia in vaccinated animals	Brandler et al. [42]
Tetravalent antigen composed of envelope domain III from four dengue virus (DV) serotypes	Mouse	Induction of neutralising antibodies to all four serotypes of DV; strong memory neutralising response after challenge with DV	Brandler et al. [43]
Hepatitis B surface antigen (HBsAg) protein	Rhesus monkey	Induction of protective anti-HBs responses after three-dose vaccination with the recombinant virus followed by a single dose of HBsAg	Reyes-del Valle et al. [44]
Single and multiple antigens of HIV-1	Mouse	Protection from pseudochallenge with recombinant vaccinia virus after vaccination with recombinant MeV-HIV expressing Gag protein	Liniger et al. [45]
Canine distemper virus (CDV) envelope proteins	Ferret	Protection from challenge with a lethal dose of CDV	Rouxel et al. [46]
Spike glycoprotein (S) or nucleocapsid protein (N) of severe acute respiratory syndrome associated coronavirus (SARS-CoV)	Mouse	Induction of anti-SARS-CoV neutralising antibodies and cellular immunity by recombinant virus expressing SARS-S and –N proteins, respectively	Liniger et al. [47]
Single or multiple antigens of simian immunodeficiency virus (SIV)	Mouse	Induction of strong humoral responses in vaccinated animals.	Zuniga et al. [48]

strategy for infants and *in vivo* studies have demonstrated their potential utility [54–56]. Alternative viral vectors for delivery of MeV glycoproteins are also being assessed [57,58].

4. Canine distemper virus

A disease with clinical manifestations matching those of canine distemper has been reported for centuries plaguing animals across the globe [59]. Canine distemper virus (CDV), was initially thought to being restricted to infection of members of the *Canidae*, although in the last 20 years it has caused outbreaks in a wide variety of hosts including members of *Felidae*, *Hyaenidae*, *Mustelidae*, *Procyonidae*, *Ursidae*, *Viverridae*, *Ailuridae* and *Mephitidae* [60,61]. Alongside this CDV has been implicated as the cause of extensive distemper outbreaks in seal populations [60,62].

In naïve dogs, CDV is generally associated with a pessimistic prognosis but its dramatic impact on wildlife species has increased awareness of the disease. In 1992, outbreaks of CDV killed several tigers, lions and leopards kept in American zoos [63], an unexpected event since CDV was not previously thought to cause clinical disease in Felids. Two years later, in 1994, a large outbreak decimated a population of lions in Serengeti National Park in Tanzania, reducing their population by an estimated 30% [64]. During this outbreak several hyena cubs also fell victim to CDV [65]. In Tanzania, a recent outbreak among African wild dogs (*Lycaon pictus*) killed 49 out of 52 breeding animals in just 2 months [66]. In fact, distemper outbreaks in domesticated and wildlife species are regularly reported across the world [67–69]. Moreover, recently the virus has expanded its host range to non-human primates, namely rhesus monkeys (*Macaca mulatta*) in China and cynomolgus monkeys (*Macaca fascicularis*) in Japan [70–72]. These reports have highlighted the need for a CDV vaccine that can be used for the control of the disease in both domesticated and wild animals.

Until recently only modified live vaccines (MLV) against CDV, both introduced in early 1960s, were available. The first, the Onderstepoort vaccine, was developed from a natural isolate which was passaged in ferrets and then adapted to chicken embryos, later replaced with chicken cell culture [73]. The other was generated by adaptation of the Rockborn strain to canine kidney cell culture [74]. The canine adapted cell culture vaccine offers protection to almost 100% vaccinated dogs, but on rare occasions can cause post-vaccination encephalitis [75]. These two vaccines have significantly reduced CDV infection in domestic dog populations, although these vaccines have found to be insufficiently attenuated for use in wildlife species. For example, a CDV vaccine generated in canine cells causes disease in grey foxes (*Urocyon cinereoargenteus*) and ferrets (*Mustela nigripes*) [76,77], while an avian attenuated vaccine can be fatal for European mink (*Mustela lutreola*) and ferrets [77,78]. Generally, the avian cell adapted vaccine is considered safer for wildlife species, and is tolerated by both grey and red foxes (*Vulpes vulpes*), bush dogs (*Speothos venaticus*), maned wolves (*Chrysocyon brachyurus*) and fennec foxes (*Vulpes zerda*) [76,79–81]. In contrast, out of all of the above mentioned species, the canine cell adapted vaccine was found safe for red foxes only [76]. Problems associated with MLV vaccines, especially their unsuitability for many endangered species, are the incentive for development of recombinant vaccines, which could be safely used for all species. The recently generated recombinant CDV vaccine, incorporating the fusion (F) and haemagglutinin (H) proteins of CDV in a strain of canarypox virus was shown to be safe to all susceptible species tested to date, including dogs, European ferrets (*Mustela putorius furo*), giant pandas (*Ailuropoda melanoleuca*), fennec foxes, meerkats (*Suricata suricatta*) and Siberian polecats (*Mustela eversmanni*) [82–85]. Whilst the canarypox vectored vaccine is safe in numerous target species, it is replication incompetent and as such induces a milder immunological response than MLV vaccines [86]. However, this latter feature highlights its applicability for immunisation of young animals in the presence of maternal antibodies [87].

Other attempts have employed reverse genetics techniques for the study and development of novel vaccines. The concept of rational attenuation of the CDV by modifying the viral RNA polymerase (L protein) to express a reporter gene from within its open reading frame led to viral attenuation, as previously demonstrated for other morbilliviruses [88,89]. Similarly, depleting the CDV H protein of N-linked glycosylation sites has produced a virus with an attenuated phenotype, that was no longer able to cause disease in ferrets [90]. A further approach centred around generating a chimeric virus combining the replication complex of the MeV Moraten strain with the glycoproteins of wild-type CDV [46]. The resulting recombinant did not cause any clinical signs or immunosuppression in vaccinated animals and induced protective immunity from lethal challenge. DNA vaccines, formulated in a similar way to those described for MeV have also shown promise inducing a strong protective humoral and cell-mediated immune response [91,92]. Although effective and relatively inexpensive to produce, problems

associated with vaccination regime and delivery routes must be resolved to make DNA vaccines an effective alternative to MLVs for wildlife populations. Finally, CDV has recently been used as a vector to express the rabies virus glycoprotein. The chimeric virus induced a strong rabies neutralising antibody response and protected mice from a lethal dose of rabies [93]. The virus was shown to be safe for both mice and dogs, and although no CDV challenge experiments were carried out, the vaccine resulted in production of long-lasting neutralising antibodies against both CDV and rabies and demonstrate the potential for multivalent morbillivirus vaccines.

5. Peste des petits ruminants virus

Peste des petits ruminants (PPR) was first identified in 1942 as a disease of small ruminants distinct from RPV [94]. In 1979 it was classified as the fourth member of the morbillivirus genus alongside RPV, MeV and CDV [95]. PPR is endemic across large parts of Africa and Asia [96]. For many years the TCRV was used effectively to protect sheep and goats from PPRV with the cross-neutralising antibody response affording protection for at least 12 months [97]. However, the need to stop vaccinating animals with the TCRV during the rinderpest eradication meant that a homologous PPRV vaccine, Nigeria 75/1, was required. This vaccine was generated by serial passages of a virulent PPRV strain in cell culture [98] and was reported to be able to protect goats and sheep from challenge with wild-type PPRV isolates for at least 3 years post-vaccination. The existence of only one serotype of PPRV means that this vaccine protects against challenge with viruses from all four PPRV lineages. Currently, several PPRV vaccines are licensed alongside the Nigeria 75/1 vaccine, with live attenuated Sungri/96, Arasur/87 and Coimbatore/97 vaccines being used in India [99].

It is widely accepted that, as with the RPV eradication, a DIVA vaccine would be of great utility in combatting PPRV. However, development of such a vaccine was hampered through the lack of a reverse genetics system for PPRV. This hurdle has been overcome by Murali et al. and Hu et al. [100,101] who have reported the development of recombinant forms of PPRV. Alternatively, with RPV eradicated then TCRV based recombinants could be used for DIVA purposes, as developed by Das et al. who generated a chimeric RPV containing the glycoproteins of PPRV [102]. This recombinant vaccine protected animals from virulent challenge and fulfilled the DIVA principles [103] but a reluctance to utilise a vaccine virus based on RPV in the post-eradication era means that such DIVA tools are unlikely to be used.

Other research groups have proposed a different approach for designing DIVA vaccines by engineering capripox virus to express PPRV H protein. If used in the field, the serological profiles of animals vaccinated with this recombinant vaccine could be distinguished from those of naturally infected animals by the absence of antibodies directed against the viral proteins not included in vaccine formulation. Although initial trials showed that a capripox vaccine protected goats from challenge with a virulent strain of PPRV, no field studies have been carried out [104–106].

Given the similarities between RPV and PPRV, it is believed that any future plans for PPRV elimination will require the implementation of the same principles which led to rinderpest eradication. There are however some requirements, which will have to be met before any coordinated action against PPRV is initiated. Most importantly, our knowledge about the epidemiology of PPRV in wildlife has to be improved. While RPV was regularly reported in wildlife species [107], it was generally accepted that wildlife populations were the victims of virus circulating in domesticated cattle rather than the reservoirs of the infection. The role of wildlife in transmission of PPR remains to be elucidated although several species have been reported as susceptible [108–110]. In addition to gaps in our understanding of PPRV epidemiology, the financial costs of a possible eradication campaign are also unknown and may be difficult to estimate. The shorter life-span of small ruminants compared to cattle means that their turnover rate is higher. This in turn results in the need for more vaccine and trained veterinary services to carry out vaccine administration. All of these factors need addressing before formulating a viable eradication programme.

6. Conclusions

The successful elimination of RPV has highlighted the potential for eradication of other morbilliviruses and initiatives to eradicate MeV are underway coordinated by the WHO [31]. Despite this, MeV is still causing the death of thousands of children in Africa and, due to controversies associated with the MMR vaccine, even developed countries are experiencing extensive epidemics. While regular vaccinations helped to reduce CDV incidence in dogs, the same virus causes mass die-offs in wildlife species, some of them already being on the verge of extinction. In addition, a new morbillivirus, named feline morbillivirus (FmoPV), was recently discovered in domestic cats (*Felis catus*) [111]. As the battle against morbilliviruses continues, further development of vaccines, in particular DIVA vaccines, will aid our ability to disrupt circulation of these viruses and potentially aid eradication of other morbilliviruses in the wake of successful RPV eradication.

References

[1] Scott GR. The Murrain now known as Rinderpest. Newsl Trop Agr Assoc UK 2000;(20):14–6.
[2] Furuse Y, Suzuki A, Oshitani H. Origin of measles virus: divergence from rinderpest virus between the 11th and 12th centuries. Virol J 2010;7:52 [Epub: 2010/03/06].
[3] Roeder PL. Rinderpest: the end of cattle plague. Prevent Vet Med 2011;102(2):98–106 [Epub: 2011/07/26].
[4] Taylor WP, Roeder PL, Rweyemamu MM. History of vaccines and vaccination. In: Barrett T, Pastoret P-P, Taylor WP, editors. Rinderpest and Peste des Petits Ruminants. Virus Plagues of Large and Small Ruminants. Academic Press; 2006. p. 222–47.
[5] Brown RD, Raschid A. The duration of immunity following vaccination with caprinised virus in the field. In: Annual Report. East African Veterinary Research Organisation, 1956–57; 1958. p. 21.
[6] Edwards JT. The uses and limitations of the caprinised virus in the control of rinderpest (cattle plague) among British and Near-Eastern cattle. Br Vet J 1949;105:209–53.
[7] Nakamura J, Fukusho K, Kuroda S. Rinderpest: laboratory experiments on immunisation of Chosen cattle by simultaneous inoculation with immune serum and rabbit virus. Jpn J Vet Sci 1943;5:455–77 [abstracted in Vet Bull 22 (1944) 186–187].
[8] Johnson RH. Rinderpest in tissue culture. I: Methods for virus production. Br Vet J 1962;118:107–16.
[9] Plowright W, Ferris RD. Studies with rinderpest virus in tissue culture. I. Growth and cytopathogenicity. J Comp Pathol 1959;69(2):152–72 [Epub: 1959/04/01].
[10] Plowright W. The application of monolayer tissue culture techniques in rinderpest research. II. The use of attenuated culture virus as a vaccine for cattle. Bull Off Int Epizoot 1962;57(253–276).
[11] Walsh EP, Baron MD, Anderson J, Barrett T. Development of a genetically marked recombinant rinderpest vaccine expressing green fluorescent protein. J Gen Virol 2000;81(Pt. 3):709–18 [Epub: 2000/02/17].
[12] Parida S, Mahapatra M, Kumar S, Das SC, Baron MD, Anderson J, et al. Rescue of a chimeric rinderpest virus with the nucleocapsid protein derived from peste-des-petits-ruminants virus: use as a marker vaccine. J Gen Virol 2007;88(Pt. 7):2019–27 [Epub: 2007/06/08].
[13] Buczkowski H, Parida S, Bailey D, Barrett T, Banyard AC. A novel approach to generating morbillivirus vaccines: negatively marking the rinderpest vaccine. Vaccine 2012;30(11):1927–35 [Epub: 2012/01/24].
[14] FAO. Report of the FAO Expert Consultation on the Strategy for Global Rinderpest Eradication. Held at FAO Headquarters, Rome, 27–29 October 1992. Rome: FAO; 1992.
[15] Rweyemamu MM, Cheneau Y. Strategy for the global rinderpest eradication programme. Vet Microbiol 1995;44(2–4):369–76 [Epub: 1995/05/01].
[16] Normile D. Animal science. Rinderpest, deadly for cattle, joins smallpox as a vanquished disease. Science 2010;330(6003):435 [Epub: 2010/10/23].
[17] Enders JF, Peebles TC. Propagation in tissue cultures of cytopathogenic agents from patients with measles. Proc Soc Exp Biol Med Soc Exp Biol Med 1954;86(2):277–86 [Epub: 1954/06/01].

[18] Enders JF, Peebles TC, McCarthy K, Milovanovic M, Mitus A, Holloway A. Measles virus: a summary of experiments concerned with isolation, properties, and behavior. Am J Publ Health Nation's Health 1957;47(3):275–82 [Epub: 1957/03/01].
[19] Katz SL, Enders JF, Holloway A. The development and evaluation of an attenuated measles virus vaccine. Am J Publ Health Nation's Health 1962;52(2 Suppl.):5–10 [Epub: 1962/02/01].
[20] Karelitz S, Peck Jr FB. Experience with measles immunization. Alum-absorbed killed measles-virus vaccine and live measles-virus vaccine challenge. Am J Dis Child 1962;103:427–9 [Epub: 1962/03/01].
[21] Rauh LW, Schmidt R. Measles immunization with killed virus vaccine. Serum antibody titers and experience with exposure to measles epidemic. Am J Dis Child 1965;109:232–7 [Epub: 1965/03/01].
[22] Hilleman MR, Buynak EB, Weibel RE, Stokes Jr J, Whitman Jr JE, Leagus MB. Development and evaluation of the Moraten measles virus vaccine. JAMA 1968;206(3):587–90 [Epub: 1968/10/14].
[23] Stokes Jr J, Weibel RE, Villarejos VM, Arguedas JA, Buynak EB, Hilleman MR. Trivalent combined measles-mumps-rubella vaccine. Findings in clinical-laboratory studies. JAMA 1971;218(1):57–61 [Epub: 1971/10/04].
[24] Banatvala JE, Brown DW. Rubella Lancet 2004;363(9415):1127–37 [Epub: 2004/04/06].
[25] Gay NJ. The theory of measles elimination: implications for the design of elimination strategies. J Infect Dis 2004;189(Suppl. 1):S27–35 [Epub: 2004/04/24].
[26] Wakefield AJ, Murch SH, Anthony A, Linnell J, Casson DM, Malik M, et al. Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. Lancet 1998;351(9103):637–41 [Epub: 1998/03/21].
[27] Black C, Kaye JA, Jick H. Relation of childhood gastrointestinal disorders to autism: nested case-control study using data from the UK General Practice Research Database. BMJ 2002;325(7361):419–21 [Epub: 2002/08/24].
[28] Uno Y, Uchiyama T, Kurosawa M, Aleksic B, Ozaki N. The combined measles, mumps, and rubella vaccines and the total number of vaccines are not associated with development of autism spectrum disorder: the first case-control study in Asia. Vaccine 2012;30(28):4292–8 [Epub: 2012/04/24].
[29] Retraction–ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. Lancet 2010;375(9713):445 [Epub: 2010/02/09].
[30] Jansen VA, Stollenwerk N, Jensen HJ, Ramsay ME, Edmunds WJ, Rhodes CJ. Measles outbreaks in a population with declining vaccine uptake. Science 2003;301(5634):804 [Epub: 2003/08/09].
[31] Casiday R, Cresswell T, Wilson D, Panter-Brick C. A survey of UK parental attitudes to the MMR vaccine and trust in medical authority. Vaccine 2006;24(2):177–84 [Epub: 2005/09/15].
[32] Pearce A, Law C, Elliman D, Cole TJ, Bedford H, Millennium Cohort Study Child Health G. Factors associated with uptake of measles, mumps, and rubella vaccine (MMR) and use of single antigen vaccines in a contemporary UK cohort: prospective cohort study. BMJ 2008;336(7647):754–7 [Epub: 2008/03/04].
[33] WHO. WHO Epidemiological Brief, vol. 18; 2011. p. 1–6.
[34] http://www.hpa.org.uk/cdr/archives/2003/cdr3903.pdf.
[35] White JM, Rush M, Leon S, Ramsay ME. COVER/Korner 95-1 (April to June 1995). Vaccination Coverage Statistics for Children up to 2 Years Old in the United Kingdom. Communicable Disease Report CDR Review, vol. 5(12); 1995. p. R186–7 [Epub: 1995/11/10].
[36] Muscat M, Bang H, Wohlfahrt J, Glismann S, Molbak K, Group EN. Measles in Europe: an epidemiological assessment. Lancet 2009;373(9661):383–9 [Epub: 2009/01/10].
[37] Tamin A, Rota PA, Wang ZD, Heath JL, Anderson LJ, Bellini WJ. Antigenic analysis of current wild type and vaccine strains of measles virus. J Infect Dis 1994;170(4):795–801 [Epub: 1994/10/01].
[38] Billeter MA, Naim HY, Udem SA. Reverse genetics of measles virus and resulting multivalent recombinant vaccines: applications of recombinant measles viruses. Curr Top Microbiol Immunol 2009;329:129–62 [Epub: 2009/02/10].
[39] Yoneda M, Georges-Courbot MC, Ikeda F, Ishii M, Nagata N, Jacquot F, et al. Recombinant measles virus vaccine expressing the nipah virus glycoprotein protects against lethal nipah virus challenge. PloS ONE 2013;8(3):e58414 [Epub: 2013/03/22].
[40] Reyes-del Valle J, de la Fuente C, Turner MA, Springfeld C, Apte-Sengupta S, Frenzke ME, et al. Broadly neutralizing immune responses against hepatitis C virus induced by vectored measles viruses and a recombinant envelope protein booster. J Virol 2012;86(21):11558–66 [Epub: 2012/08/17].
[41] Stebbings R, Fevrier M, Li B, Lorin C, Koutsoukos M, Mee E, et al. Immunogenicity of a recombinant measles-HIV-1 clade B candidate vaccine. PloS ONE 2012;7(11):e50397 [Epub: 2012/12/12].
[42] Brandler S, Marianneau P, Loth P, Lacote S, Combredet C, Frenkiel MP, et al. Measles vaccine expressing the secreted form of West Nile virus envelope glycoprotein induces protective immunity in squirrel monkeys, a new model of West Nile virus infection. J Infect Dis 2012;206(2):212–9 [Epub: 2012/05/04].
[43] Brandler S, Ruffie C, Najburg V, Frenkiel MP, Bedouelle H, Despres P, et al. Pediatric measles vaccine expressing a dengue tetravalent antigen elicits neutralizing antibodies against all four dengue viruses. Vaccine 2010;28(41):6730–9 [Epub: 2010/08/07].
[44] Reyes-del Valle J, Hodge G, McChesney MB, Cattaneo R. Protective anti-hepatitis B virus responses in rhesus monkeys primed with a vectored measles virus and boosted with a single dose of hepatitis B surface antigen. J Virol 2009;83(17):9013–7 [Epub: 2009/06/19].
[45] Liniger M, Zuniga A, Morin TN, Combardiere B, Marty R, Wiegand M, et al. Recombinant measles viruses expressing single or multiple antigens of human immunodeficiency virus (HIV-1) induce cellular and humoral immune responses. Vaccine 2009;27(25–26):3299–305 [Epub: 2009/02/10].
[46] Rouxel RN, Svitek N, von Messling V. A chimeric measles virus with canine distemper envelope protects ferrets from lethal distemper challenge. Vaccine 2009;27(36):4961–6 [Epub: 2009/06/23].
[47] Liniger M, Zuniga A, Tamin A, Azzouz-Morin TN, Knuchel M, Marty RR, et al. Induction of neutralising antibodies and cellular immune responses against SARS coronavirus by recombinant measles viruses. Vaccine 2008;26(17):2164–74 [Epub: 2008/03/19].
[48] Zuniga A, Wang Z, Liniger M, Hangartner L, Caballero M, Pavlovic J, et al. Attenuated measles virus as a vaccine vector. Vaccine 2007;25(16):2974–83 [Epub: 2007/02/17].
[49] Rima BK. Strategy for measles eradication. In: Barrett T, Pastoret P-P, Taylor WP, editors. Rinderpest and Peste des Petits Ruminants Virus Plagues of Large and Small Ruminants. 2006. p. 284–97.
[50] Dine MS, Hutchins SS, Thomas A, Williams I, Bellini WJ, Redd SC. Persistence of vaccine-induced antibody to measles 26–33 years after vaccination. J Infect Dis 2004;189(Suppl. 1):S123–30 [Epub: 2004/04/24].
[51] Bruning-Richardson A, Akerblom L, Klingeborn B, Anderson J. Improvement and development of rapid chromatographic strip-tests for the diagnosis of rinderpest and peste des petits ruminants viruses. J Virol Methods 2011;174(1–2):42–6 [Epub: 2011/03/23].
[52] Dallaire F, De Serres G, Tremblay FW, Markowski F, Tipples G. Long-lasting measles outbreak affecting several unrelated networks of unvaccinated persons. J Infect Dis 2009;200(10):1602–5 [Epub: 2009/10/16].
[53] Halsey NA. Increased mortality after high-titre measles vaccines: too much of a good thing. Pediatr Infect Dis J 1993;12:462–5.
[54] Polack FP, Lee SH, Permar S, Manyara E, Nousari HG, Jeng Y, et al. Successful DNA immunization against measles: neutralizing antibody against either the hemagglutinin or fusion glycoprotein protects rhesus macaques without evidence of atypical measles. Nat Med 2000;6(7):776–81 [Epub: 2000/07/11].
[55] Premenko-Lanier M, Rota PA, Rhodes GH, Bellini WJ, McChesney MB. Protection against challenge with measles virus (MV) in infant macaques by an MV DNA vaccine administered in the presence of neutralizing antibody. J Infect Dis 2004;189(11):2064–71 [Epub: 2004/05/15].
[56] Pasetti MF, Ramirez K, Resendiz-Albor A, Ulmer J, Barry EM, Levine MM. Sindbis virus-based measles DNA vaccines protect cotton rats against respiratory measles: relevance of antibodies, mucosal and systemic antibody-secreting cells, memory B cells, and Th1-type cytokines as correlates of immunity. J Virol 2009;83(6):2789–94 [Epub: 2009/01/09].
[57] Pan CH, Greer CE, Hauer D, Legg HS, Lee EY, Bergen MJ, et al. A chimeric alphavirus replicon particle vaccine expressing the hemagglutinin and fusion proteins protects juvenile and infant rhesus macaques from measles. J Virol 2010;84(8):3798–807 [Epub: 2010/02/05].
[58] Skiadopoulos MH, Surman SR, Riggs JM, Collins PL, Murphy BR. A chimeric human-bovine parainfluenza virus type 3 expressing measles virus hemagglutinin is attenuated for replication but is still immunogenic in rhesus monkeys. J Virol 2001;75(21):10498–504 [Epub: 2001/10/03].
[59] Blancou J. Dog distemper: imported into Europe from South America? Hist Med Vet 2004;29(2):35–41 [Epub: 2004/09/21].
[60] Appel MJ, Summers BA. Pathogenicity of morbilliviruses for terrestrial carnivores. Vet Microbiol 1995;44(2–4):187–91 [Epub: 1995/05/01].
[61] Woolf A, Gremillion-Smith C, Evans RH. Evidence of canine distemper virus infection in skunks negative for antibody against rabies virus. J Am Vet Med Assoc 1986;189(9):1086–8 [Epub: 1986/11/01].
[62] Osterhaus AD, Groen J, De Vries P, UytdeHaag FG, Klingeborn B, Zarnke R. Canine distemper virus in seals. Nature 1988;335(6189):403–4 [Epub: 1988/09/29].
[63] Harder TC, Kenter M, Vos H, Siebelink K, Huisman W, van Amerongen G, et al. Canine distemper virus from diseased large felids: biological properties and phylogenetic relationships. J Gen Virol 1996;77(Pt 3):397–405 [Epub: 1996/03/01].
[64] Roelke-Parker ME, Munson L, Packer C, Kock R, Cleaveland S, Carpenter M, et al. A canine distemper virus epidemic in Serengeti lions (*Panthera leo*). Nature 1996;379(6564):441–5 [Epub: 1996/02/01].
[65] Haas L, Hofer H, East M, Wohlsein P, Liess B, Barrett T. Canine distemper virus infection in Serengeti spotted hyenas. Vet Microbiol 1996;49(1–2):147–52 [Epub: 1996/03/01].
[66] van de Bildt MW, Kuiken T, Visee AM, Lema S, Fitzjohn TR, Osterhaus AD. Distemper outbreak and its effect on African wild dog conservation. Emerg Infect Dis 2002;8(2):211–3 [Epub: 2002/03/19].
[67] Thomas S. Canine distemper outbreak in ferrets in the UK. Vet Rec 2012;170(1):27 [Epub: 2012/01/10].
[68] Schumaker BA, Miller MM, Grosdidier P, Cavender JL, Montgomery DL, Cornish TE, et al. Canine distemper outbreak in pet store puppies linked to a high-volume dog breeder. J Vet Diagn Invest 2012;24(6):1094–8 [Epub: 2012/09/27].
[69] Nagao Y, Nishio Y, Shiomoda H, Tamaru S, Shimojima M, Goto M, et al. An outbreak of canine distemper virus in tigers (*Panthera tigris*): possible transmission from wild animals to zoo animals. J Vet Med Sci 2012;74(6):699–705 [Epub: 2012/01/05].
[70] Sakai K, Nagata N, Ami Y, Seki F, Suzaki Y, Iwata-Yoshikawa N, et al. Lethal canine distemper virus outbreak in cynomolgus monkeys in Japan in 2008. J Virol 2013;87(2):1105–14 [Epub: 2012/11/09].

[71] Qiu W, Zheng Y, Zhang S, Fan Q, Liu H, Zhang F, et al. Canine distemper outbreak in rhesus monkeys, China. Emerg Infect Dis 2011;17(8):1541–3 [Epub: 2011/08/02].
[72] Yoshikawa Y, Ochikubo F, Matsubara Y, Tsuruoka H, Ishii M, Shirota K, et al. Natural infection with canine distemper virus in a Japanese monkey (*Macaca fuscata*). Vet Microbiol 1989;20(3):193–205 [Epub: 1989/07/01].
[73] Haig DA. Canine distemper-immunization with avianised virus. Onderstepoort J Vet Res 1956;27:19–53.
[74] Rockborn G. An attenuated strain of canine distemper virus in tissue culture. Nature 1959;184(Suppl. 11):822 [Epub: 1959/09/12].
[75] Hartley WJ. A post-vaccinal inclusion body encephalitis in dogs. Vet Pathol 1974;11(4):301–12 [Epub: 1974/01/01].
[76] Halbrooks RD, Swango LJ, Schnurrenberger PR, Mitchell FE, Hill EP. Response of gray foxes to modified live-virus canine distemper vaccines. J Am Vet Med Assoc 1981;179(11):1170–4 [Epub: 1981/12/01].
[77] Carpenter JW, Appel MJ, Erickson RC, Novilla MN. Fatal vaccine-induced canine distemper virus infection in black-footed ferrets. J Am Vet Med Assoc 1976;169(9):961–4 [Epub: 1976/11/01].
[78] Sutherland-Smith MR, Rideout BA, Mikolon AB, Appel MJ, Morris PJ, Shima AL, et al. Vaccine-induced canine distemper in European mink, *Mustela lutreola*. J Zoo Wildl Med Off Publ Am Assoc Zoo Vet 1997;28(3):312–8 [Epub: 1997/11/20].
[79] McInnes EF, Burroughs RE, Duncan NM. Possible vaccine-induced canine distemper in a South American bush dog (*Speothos venaticus*). J Wildl Dis 1992;28(4):614–7 [Epub: 1992/10/01].
[80] Thomas-Baker B. Vaccination-induced distemper in maned wolves, vaccination-induced corneal opacity in a maned wolf. Proc Am Assoc Zoo Vet 1985:53 [Ann. Rep., Scottsdale, Ariz.].
[81] Montali RJ, Bartz CR, Bush M. Canine distemper virus. In: Appel M, editor. Virus Infections of Carnivores. Amsterdam: Elsevier Science Publishers; 1987. p. 437–43.
[82] Stephensen CB, Welter J, Thaker SR, Taylor J, Tartaglia J, Paoletti E. Canine distemper virus (CDV) infection of ferrets as a model for testing Morbillivirus vaccine strategies: NYVAC- and ALVAC-based CDV recombinants protect against symptomatic infection. J Virol 1997;71(2):1506–13 [Epub: 1997/02/01].
[83] Coke RL, Backues KA, Hoover JP, Saliki JT, Ritchey JW, West GD. Serologic responses after vaccination of fennec foxes (*Vulpes zerda*) and meerkats (*Suricata suricatta*) with a live, canarypox-vectored canine distemper virus vaccine. J Zoo Wildl Med Off Publ Am Assoc Zoo Vet 2005;36(2):326–30 [Epub: 2007/02/28].
[84] Bronson E, Deem SL, Sanchez C, Murray S. Serologic response to a canarypox-vectored canine distemper virus vaccine in the giant panda (*Ailuropoda melanoleuca*). J Zoo Wildl Med Off Publ Am Assoc Zoo Vet 2007;38(2):363–6 [Epub: 2007/08/08].
[85] Wimsatt J, Biggins D, Innes K, Taylor B, Garell D. Evaluation of oral and subcutaneous delivery of an experimental canarypox recombinant canine distemper vaccine in the Siberian polecat (*Mustela eversmanni*). J Zoo Wildl Med Off Publ Am Assoc Zoo Vet 2003;34(1):25–35 [Epub: 2003/05/02].
[86] Schultz RD. Duration of immunity for canine and feline vaccines: a review. Vet Microbiol 2006;117(1):75–9 [Epub: 2006/05/19].
[87] Pardo MC, Tanner P, Bauman J, Silver K, Fischer L. Immunization of puppies in the presence of maternally derived antibodies against canine distemper virus. J Compar Pathol 2007;137(Suppl 1):S72–5 [Epub: 2007/06/15].
[88] Silin D, Lyubomska O, Ludlow M, Duprex WP, Rima BK. Development of a challenge-protective vaccine concept by modification of the viral RNA-dependent RNA polymerase of canine distemper virus. J Virol 2007;81(24):13649–58 [Epub: 2007/09/28].
[89] Brown DD, Rima BK, Allen IV, Baron MD, Banyard AC, Barrett T, et al. Rational attenuation of a morbillivirus by modulating the activity of the RNA-dependent RNA polymerase. J Virol 2005;79(22):14330–8 [Epub: 2005/10/29].
[90] Sawatsky B, von Messling V. Canine distemper viruses expressing a hemagglutinin without N-glycans lose virulence but retain immunosuppression. J Virol 2010;84(6):2753–61 [Epub: 2010/01/01].
[91] Jensen TH, Nielsen L, Aasted B, Blixenkrone-Moller M. Early life DNA vaccination with the H gene of Canine distemper virus induces robust protection against distemper. Vaccine 2009;27(38):5178–83 [Epub: 2009/07/15].
[92] Nielsen L, Jensen TH, Kristensen B, Jensen TD, Karlskov-Mortensen P, Lund M, et al. DNA vaccines encoding proteins from wild-type and attenuated canine distemper virus protect equally well against wild-type virus challenge. Acta Virol 2012;157(10):1887–96 [Epub: 2012/06/21].
[93] Wang X, Feng N, Ge J, Shuai L, Peng L, Gao Y, et al. Recombinant canine distemper virus serves as bivalent live vaccine against rabies and canine distemper. Vaccine 2012;30(34):5067–72 [Epub: 2012/06/16].
[94] Gargadennec L, Lalanne A. La peste des petits ruminants. Bull Serv Zootech Epiz Afr Occid Franc 1942;(5):16–21.
[95] Gibbs EPJ, Taylor WP, Lawman MPJ, Bryant J. Classification of peste des petits ruminants virus as the fourth member of the genus morbillivirus. Intervirology 1979;11:268–74.
[96] Dhar P, Sreenivasa BP, Barrett T, Corteyn M, Singh RP, Bandyopadhyay SK. Recent epidemiology of peste des petits ruminants virus (PPRV). Vet Microbiol 2002;88(2):153–9.
[97] Taylor WP. Protection of goats against peste-des-petits-ruminants with attenuated rinderpest virus. Res Vet Sci 1979;27(3):321–4 [Epub: 1979/11/01].
[98] Diallo A, Taylor WP, Lefevre PC, Provost A. Attenuation of a strain of rinderpest virus: potential homologous live vaccine. Revue d'elevage et de medecine veterinaire des pays tropicaux 1989;42(3):311–9 [Epub: 1989/01/01, Attenuation d'une souche de virus de la peste des petits ruminants: candidat pour un vaccin homologue vivant].
[99] Saravanan P, Sen A, Balamurugan V, Rajak KK, Bhanuprakash V, Palaniswami KS, et al. Comparative efficacy of peste des petits ruminants (PPR) vaccines. Biol J Int Assoc Biol Standard 2010;38(4):479–85 [Epub: 2010/03/05].
[100] Hu Q, Chen W, Huang K, Baron MD, Bu Z. Rescue of recombinant peste des petits ruminants virus: creation of a GFP-expressing virus and application in rapid virus neutralization test. Vet Res 2012;43(1):48 [Epub: 2012/06/05].
[101] Muniraju M, Buczkowski H, Banyard AC, Parida S. Development of a reverse genetics system for Peste des petits ruminants virus: the first successful rescue of a recombinant PPRV. In: 1st Meeting of the Global PPRV Eradication Alliance. 2012.
[102] Das SC, Baron MD, Barrett T. Recovery and characterization of a chimeric rinderpest virus with the glycoproteins of peste-des-petits-ruminants virus: homologous F and H proteins are required for virus viability. J Virol 2000;74(19):9039–47.
[103] Anderson J, McKay JA. The detection of antibodies against peste des petits ruminants virus in cattle, sheep and goats and the possible implications to rinderpest control programmes. Epidemiol Infect 1994;112(1):225–31.
[104] Berhe G, Minet C, Le Goff C, Barrett T, Ngangnou A, Grillet C, et al. Development of a dual recombinant vaccine to protect small ruminants against peste-des-petits-ruminants virus and capripoxvirus infections. J Virol 2003;77(2):1571–7 [Epub: 2002/12/28].
[105] Diallo A, Minet C, Berhe G, Le Goff C, Black DN, Fleming M, et al. Goat immune response to capripox vaccine expressing the hemagglutinin protein of peste des petits ruminants. Ann N Y Acad Sci 2002;969:88–91 [Epub: 2002/10/17].
[106] Chen W, Hu S, Qu L, Hu Q, Zhang Q, Zhi H, et al. A goat poxvirus-vectored peste-des-petits-ruminants vaccine induces long-lasting neutralization antibody to high levels in goats and sheep. Vaccine 2010;28(30):4742–50 [Epub: 2010/05/18].
[107] Kock RA, Wambua JM, Mwanzia J, Wamwayi H, Ndungu EK, Barrett T, et al. Rinderpest epidemic in wild ruminants in Kenya 1993–97. Vet Rec 1999;145(10):275–83 [Epub: 1999/12/01].
[108] Kinne J, Kreutzer R, Kreutzer M, Wernery U, Wohlsein P. Peste des petits ruminants in Arabian wildlife. Epidemiol Infect 2010;138(8):1211–4 [Epub: 2010/01/14].
[109] Furley CW, Taylor WP, Obi TU. An outbreak of peste des petits ruminants in a zoological collection. Vet Rec 1987;121(19):443–7 [Epub: 1987/11/07].
[110] Elzein EM, Housawi FM, Bashareek Y, Gameel AA, Al-Afaleq AI, Anderson E. Severe PPR infection in gazelles kept under semi-free range conditions. J Vet Med B Infect Dis Vet Publ Health 2004;51(2):68–71 [Epub: 2004/03/20].
[111] Woo PC, Lau SK, Wong BH, Fan RY, Wong AY, Zhang AJ, et al. Feline morbillivirus, a previously undescribed paramyxovirus associated with tubulointerstitial nephritis in domestic cats. Proc Natl Acad Sci U S A 2012;109(14):5435–40 [Epub: 2012/03/21].