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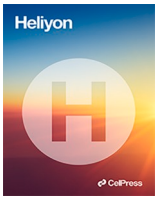

Review article

Morbillivirus: A highly adaptable viral genus

Jane E. Libbey, Robert S. Fujinami *

Department of Pathology, University of Utah School of Medicine, 15 North Medical Drive East, 1100, EEJMRB, Salt Lake City, UT, 84112, USA

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ABSTRACT

Over the course of human history, numerous diseases have been caused by the transmission of viruses from an animal reservoir into the human population. The viruses of the genus *Morbillivirus* are human and animal pathogens that emerged from a primordial ancestor a millennia ago and have been transmitting to new hosts, adapting, and evolving ever since. Through interaction with susceptible individuals, as yet undiscovered morbilliviruses or existing morbilliviruses in animal hosts could cause future zoonotic diseases in humans.

1. Introduction

Morbilliviruses infect and cause disease in both humans and a wide range of animals. Over half of the morbillivirus species were first characterized in the 20th century and viral genomes classified as morbilliviruses have been isolated even more recently in the 21st century. The ongoing discovery of new morbillivirus species in an ever-expanding range of hosts attests to the adaptability of these viruses.

This review will describe the general viral properties and biology of morbilliviruses, followed by an in-depth history of each viral species and a description of the efforts to eradicate the individual viruses for which this has been attempted. The phylogenetic relationship between the morbilliviruses will be explored. Common receptor usage and close antigenic relationship are presented as support for all present-day morbilliviruses having evolved from a common ancestral virus. Finally, the possibility of future morbillivirus emergence and zoonotic disease occurrence are discussed.

2. Morbillivirus

Viruses of the genus *Morbillivirus*, family *Paramyxoviridae*, are important animal and human pathogens resulting in severe and often fatal acute disease [1,2]. Morbillivirus virions are pleomorphic, ranging in size from 100 to 300 nm [3]. The virions are sensitive to heat, sunlight, pH, and lipid-destroying chemicals [2]. These enveloped viruses are characterized by the presence of nonsegmented, linear, single-stranded negative-sense RNA genomes, ranging from 15,040 (representative rodent morbillivirus: longquan berylmys bowersi morbillivirus 1, LBbMV) to 16,050 (*Feline Morbillivirus*, FeMV) nucleotides in size, which encode eight proteins: N (nucleocapsid), P (phosphoprotein), V, C, M (matrix), F (fusion), H (hemagglutinin), and L (large polymerase), six of which are structural and present in the virion (Fig. 1) [[1,4–8], reviewed in Refs. [3,9]]. All morbilliviruses conform to the rule of six whereby each N protein monomer encapsidates six nucleotides of RNA [8]. Morbilliviruses are highly infectious mainly via the respiratory route [1,6,7]. Acute infection can be classified into five distinct stages: incubation, prodromal, mucosal, diarrheic, and convalescent [8]. Upon infection, morbillivirus replicates within the pharyngeal regions followed by viremia and spread to lymphatic and epithelial tissues throughout

* Corresponding author.
E-mail addresses: Jane.Libbey@outlook.com (J.E. Libbey), Robert.Fujinami@hsc.utah.edu (R.S. Fujinami).

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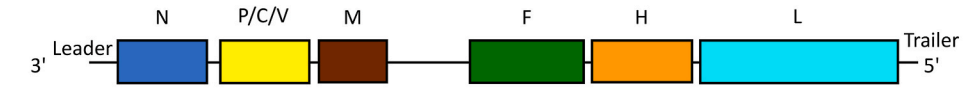
the body [[7], reviewed in Ref. [3]]. Lymphopenia results from morbillivirus infection, in turn causing a profound but transient immunosuppression and susceptibility to secondary, opportunistic bacterial infections, or viral co-infections, with the concomitant increased risk of mortality [8,10,11]. Virus is primarily shed via the oronasal secretions, and this can occur before clinical signs appear [7]. Transmission occurs between infected and susceptible hosts [2]. Respiratory, intestinal, and other signs of morbillivirus infection include fever, depression, nose and eye discharge, pneumonia, gastroenteritis, diarrhea, and dehydration [7,12]. Epidemics occur in previously unexposed populations in which virus disseminates from one acutely infected host to the next [13]. High morbidity and mortality are associated with morbillivirus outbreaks in previously unexposed populations, but survivors develop lifelong immunity [4,6,14,15], and, as a result, are only ever infected once in their lifetime due to this highly immunizing infection. Thus, in populations in which morbilliviruses have become endemic (permanently established within a host population), disease presents/manifests in childhood [6]. Additionally, morbillivirus immunity can provide cross-protection to distantly related morbilliviruses [16,17], acting to restrict spill-over between hosts.

Morbilliviruses are classified as seven species of viruses, to include the measles virus (MeV, species redesignated *Measles Morbillivirus*) which infects humans and other primates [5,6,8,14,18]. The other six viruses in this genus are canine distemper virus (CDV, species redesignated *Canine Morbillivirus*) which infects a wide variety of mammals, to include, but not inclusive, domestic and wild dogs, coyotes, foxes, wolves, pandas, bears, ferrets, skunks, raccoons, and large felines (not domestic cats), as well as seals, walruses, and sea lions, and some primates; FeMV which infects both wild and domestic cats; phocine distemper virus (PDV, species redesignated *Phocine Morbillivirus*) which mainly infects seals; *Cetacean Morbillivirus* (CeMV) which infects dolphins, porpoises, and whales; rinderpest virus (RPV, species redesignated *Rinderpest Morbillivirus*) which infects cattle, domestic buffalo (to include water buffalo), large antelope, deer, giraffes, warthogs, and wildebeests; and pestis-des-petits-ruminants virus (PPRV, species redesignated *Small Ruminant Morbillivirus*) which infects goats, and sheep (Fig. 2) [5,6,8,14,18]. More recently full-length viral genomes have been isolated from domestic pigs (PoMV, *Porcine Morbillivirus*), bats (MBaMV, *Myotitis Bat Morbillivirus*), and rodents (variously named) which have been classified as morbilliviruses (Fig. 2) [reviewed in Ref. [9]]. Reverse transcription/polymerase chain reaction (RT-PCR) morbillivirus primer sets have been produced that can be used to distinguish between RPV and PPRV [19], and sensitive and broadly reactive RT-PCR primer sets have been produced for the detection and identification, through sequencing, of known (MeV, CDV, PDV, CeMV; RPV, PPRV, and FeMV not tested) and novel morbilliviruses [20].

2.1. Human morbillivirus

The development of lifelong immunity following infection, and the fragility of the virions outside of a host, makes it necessary for a constant supply of new susceptible hosts to be present in the population for morbilliviruses to maintain themselves [2,21]. For MeV in the human population, the estimate is at least 300,000 individuals for MeV to maintain itself in circulation as an endemic virus infection with disease prevalence varying from year to year and season to season [[22], reviewed in Ref. [23]]. The prevailing theory is that the human population reached this level when large agricultural communities were established, such as the Middle Eastern River



	Size Range (nucleotides)
Genome	15,040 – 16,050
N	1,548 – 1,578
P	1,476 – 1,530
M	1,008 – 1,014
F	1,632 – 1,995
H	1,788 – 1,830
L	6,552 – 6,609

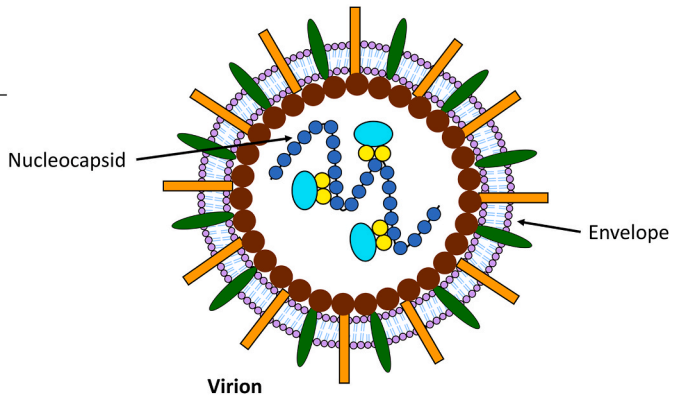


Fig. 1. Schematic representations of the Morbillivirus genome and virion. The size of the genomic RNA varies from 15,040 nucleotides for a representative rodent morbillivirus genome to 16,050 nucleotides for the FeMV genome. The non-structural C protein is encoded by an overlapping open reading frame while the non-structural V protein is encoded through RNA editing. The pleiomorphic virions are approximately 200 nm in diameter. The nucleocapsid of the virion is made up of the genomic RNA and N proteins which associate with the L and P proteins. The envelope is a lipid bilayer, acquired from the host cell, through which the H and F proteins project from the surface of the virion. The M protein interacts with both the nucleocapsid and the envelope H and F proteins. The color of the transcription unit in the genome matches the color of the protein in the virion. FeMV, feline morbillivirus. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

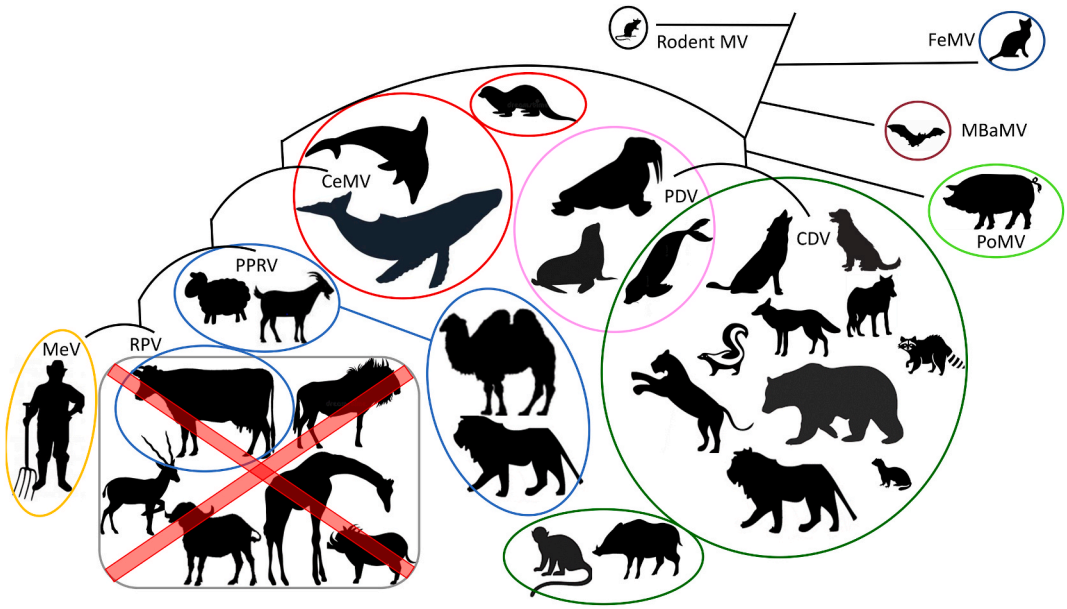


Fig. 2. Host species (not all inclusive) known to be commonly infected (endemic/epidemic/reservoir) with morbilliviruses. Expansion of the host range of PPRV, CeMV, and CDV are indicated by the adjacent/attached bubbles of the same color. RPV has been eradicated as indicated by the red X. The phylogenetic relationships of the morbilliviruses are roughly indicated by the black bars. CDV, canine distemper virus; CeMV, cetacean morbillivirus; FeMV, feline morbillivirus; MBaMV, Myotitis bat morbillivirus; MeV, measles virus; MV, morbillivirus; PDV, phocine distemper virus; PoMV, porcine morbillivirus; PPRV, pestis-des-petits-ruminants virus; RPV, rinderpest virus. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

valley civilizations about 4300 years ago, and that humans contracted a morbillivirus infection from their domesticated cattle, such as an RPV-like bovine morbillivirus, which evolved into MeV [[21], reviewed in Refs. [2–4,23]]. One selection-aware molecular clock modeling study has dated the divergence of MeV from RPV to as early as the 6th century BCE (528 BCE), more than 2500 years ago, roughly coinciding with the rise of large cities in North Africa, India, China, Europe, and the Near East at around 300 BCE [24]. However, the first scientific description of a measles-like disease was by Rhazes of Baghdad in the 9th century CE, and measles epidemics occurring in the human population were not recorded until the 11th and 12th centuries CE [reviewed in Refs. [3,4,25]]. An earlier molecular clock analysis indicated the divergence of MeV from RPV occurred in the 11th or 12th century CE [25]. Therefore, there is a disconnection between the development of human communities of sufficient size to sustain MeV transmission and the first description of measles, the disease [4]. Regardless, a study evaluating the role of agriculture and domestic animals in the evolution of human disease found strong evidence for a domestic-animal origin for measles [26].

There is no animal reservoir for MeV and natural infection occurs only in humans [3]. Infection with MeV is characterized by fever, a macular red skin rash over most of the body, cough, conjunctivitis, photophobia, nausea, and a generalized immune suppression [3, 27]. MeV is highly contagious, with a basic reproductive rate (R0) of 9–18, and transmission can occur prior to the development of symptoms [28]. MeV has a 10–14 day incubation period, from the time of exposure to the appearance of clinical symptoms, a 2–3 day prodrome period with onset of clinical symptoms, followed by the onset of the skin rash which begins to fade within 3–4 days, and individuals are most infectious starting 4–5 days prior to the appearance of the rash continuing through the first 4 days of the rash [3]. Children under 5 years of age are most commonly affected and complications include pneumonia, encephalitis, and death [reviewed in Ref. [29]]. High morbidity and mortality occur upon the introduction of MeV into previously unexposed populations [reviewed in Ref. [3]]. MeV infection can result in acute post infection disseminated encephalomyelitis (ADEM) of the central nervous system, occurring within 2 weeks after the onset of the rash, at a rate of 1 per 1000 infections [3,10]. ADEM is currently understood to be an autoimmune disease induced during MeV infection which more often occurs following MeV infection of children older than 5 years of age [3]. The majority of ADEM survivors have neurologic sequelae [3]. Immunosuppressed individuals can develop measles inclusion body encephalitis upon MeV infection [10]. Immunocompromised adults and children can develop neurological signs 6–12 months after exposure to MeV and rapidly progress to coma and death within weeks to months [3]. Viral persistence in the brain can result in subacute sclerosing panencephalitis (SSPE), a slow, fatal, neurodegenerative disease, occurring at a rate of 1 per 10,000 infections as a late complication, 7–10 years post infection [1,3,10]. Death occurs within months to years of onset [3]. Sequence analysis of viral RNAs isolated from infected tissue of SSPE patients shows frequent mutations in the genes encoding the H, F, and M proteins. SSPE more often occurs following MeV infection of children under 2 years of age when maternal antibodies may still be present and the immune system is immature [3].

A case was first made for the global eradication of measles in 1982 [30], and since then, measles, the disease, has been recommended as a target for worldwide eradication via vaccination [31]. Eradication of MeV is feasible as no animal reservoir exists, highly

effective vaccines are available, and MeV is antigenically stable [reviewed in Refs. [3,32]]. A recent study to assess the genomic stability of vaccine-strain MeV during extended replication found that, despite the lack of a proofreading polymerase, a remarkably small number of mutations accrued over 120 serial passages [33]. MeV had a worldwide distribution, before the introduction of vaccination, having first been described in the Middle East in the 9th century to being endemic in the Americas by the end of the 17th century [reviewed in Ref. [4]]. Although serologically monotypic, MeV genotyping of the genes encoding the H and N viral proteins confirms 8 clades (A-H) which can be further subdivided into at least 23 genotypes [3,29]. Live attenuated MeV vaccine was first licensed in 1963 [3,28–30], and widespread MeV vaccination began first in the U.S. in 1967 [reviewed in Ref. [23]]. The MeV vaccine in current usage is prepared from clade A (Edmonston strain) but is protective against all clades [29]. The live MeV vaccine in current usage (M-M-R II from Merck) was initially approved for use in the U.S. in 1978 [34], but remains effective today against currently circulating wild-type strains due to the stability of the MeV genome. The effect of the widespread vaccination in the U.S. alone was to drop the incidence of infection from an estimated 4 million cases per year prior to the introduction of vaccine to an average of 4500 cases per year during the 1980's [reviewed in Ref. [23]]. In 1974 the World Health Organization introduced its Expanded Program on Immunization [31]. The result was a drop in measles cases and deaths from an estimated 100 million cases and 5.8 million deaths in 1980 to an estimated 44 million cases and 1.1 million deaths in 1995 worldwide. The goal of global measles eradication via vaccination was recommended in 1996 with a target date of 2005–2010 [31]. The Measles Initiative, founded in 2001, contributed to the 78% reduction in global measles deaths between 2000 and 2008 through its support of vaccination activities [35]. The minimum level of vaccination coverage needed for the interruption of MeV transmission is high at 89–94% [28]. Regardless, live attenuated MeV vaccination has led to the end of endemic transmission of MeV in many parts of the developed world [4]. The goal of global eradication has not been reached, however, as over time, due to the less than 100% effectiveness of the vaccine and the lack of universal vaccine coverage, the number of children susceptible to disease increases to a level capable of sustaining MeV transmission, resulting in measles outbreaks, even in areas with high vaccination coverage, when the virus is reintroduced [29,31]. Outbreaks are also occurring in older subjects such as teenagers and young adults [29]. Routine vaccination against MeV is performed at 9 months of age in areas where measles is still prevalent and at 12–15 months of age in areas where there is little evidence of measles, followed later by a second dose [3]. The major obstacles to global measles eradication have been perceptual, political, and financial [31]. Underestimation of the severity of the disease and unsubstantiated links between vaccination and a host of medical conditions have led to reduced vaccination rates and large outbreaks in the developed world [reviewed in Ref. [4]]. Since 2008, declines in donor commitment led to cuts in vaccination activities and a resultant reversal of progress and a global resurgence of measles [35]. More recently, measles deaths annually topped 140,000 in 2018 and 207,500 in 2019 (with a 23 year high in the reported number of cases of measles, and the latest year for which data is available), mostly in the developing world and mostly in children under 5 years of age [36,37]. Poor health care, unsanitary conditions, concurrent bacterial or parasitic infections, and malnutrition all factor into the mortality rate of measles in children [1]. Regardless, global eradication of MeV followed by complete withdrawal of MeV vaccination would result in a human population without MeV-specific immunity, conferred by infection or vaccination, creating a potential opening for infection of humans by closely related animal morbilliviruses [[10,38–40], reviewed in Ref. [32]].

2.2. Animal morbilliviruses

2.2.1. RPV

RPV was recognized as a distinct clinical entity affecting cattle around 376-386 BCE [reviewed in Ref. [41]]. Having arisen in Asia [reviewed in Ref. [42]], RPV first appeared in Europe in 1711, having traveled from Asia to Italy [1]. It was a devastating disease that killed an estimated 200 million cattle in Europe in the 1700's alone [reviewed in Ref. [41]]. RPV was prevalent in Europe, Asia, the Middle East, and Africa, but it never spread to the Americas [4]. Highly virulent strains of RPV can result in near 100% mortality [2]. RPV is not neurovirulent [reviewed in Ref. [42]]. Control measures, including the slaughter of infected animals and the use of antiserum, rigorously applied, led to Europe (except Turkey) being free of RPV by 1930 [reviewed in Ref. [41]]. Outbreaks in areas free of RPV were found to be due to the introduction of live, infected animals. Control of the spread of disease following reintroduction involved quarantine, movement restriction, slaughter, antiserum, and vaccination. Lifelong immunity in adult cattle, due to past infection or vaccination, and the protection of calves for 9–10 months via maternal antibody, led to RPV becoming a disease of yearlings. The presence of large populations of susceptible wild animals, especially in Africa, could help to maintain and disperse RPV [reviewed in Ref. [41]].

In 2011 RPV was declared globally eradicated by targeted vaccination [6]. As early as the 1960's, the Plowright tissue culture attenuated vaccine, a cheap and easily produced vaccine that was safe and not shed by the animal following vaccination, was employed to control RPV [7]. One barrier to the eradication of RPV was the presence of very mild field strains that were difficult to detect and diagnose [7,43]. In contrast to MeV, RPV strain differences give rise to different pathogenicities and different cattle breeds differ in susceptibility [1]. Another barrier to eradication of RPV was the ability of small ruminants to be carriers of RPV without themselves becoming sick [[44,45], reviewed in Ref. [41]]. RPV can infect sheep and goats, often resulting in acute disease and death, but certain strains of RPV have also been observed to cause only a mild or inapparent infection in these animals, due possibly to reduced pathogenicity of the virus strain or genetic differences of the host animal. Exposure of susceptible cattle to these infected sheep or goats resulted in outbreaks of overt disease in the cattle [44,45]. The experimental infection of goats, however, played an important early role in the eradication of RPV as they provided the first attenuated virus, developed in the 1930's, for use as vaccine and the means for large scale production of that vaccine [reviewed in Refs. [2,41]].

2.2.2. CDV

In work published in 1792, from time spent in South America as a member of the 1735 French Geodesic Mission to measure the equator, Antonio de Ulloa y de la Torre-Giral clearly described disease symptoms and characteristics typical of endemic canine distemper in dogs, a disease unknown in Europe up to that time [reviewed in Refs. [4,42,46,47]]. CDV first appeared in Europe in the 1760's, having traveled from South America (Peru) to Spain, from whence the epidemic spread throughout Europe and the rest of the world [reviewed in Refs. [4,46,47]]. The appearance of CDV in the Americas in the early 1700's was well after the introduction of MeV to the Americas in the late 1400's and early 1500's, more than 200 years after [reviewed in Refs. [42,48]]. Distemper-like epidemics were not reported in important hunting and war dogs imported to the Americas by European explorers and conquistadors between 1500 and 1700, as would occur upon exposure of a previously unexposed dog population, suggesting that CDV was not present in the Americas prior to the arrival of Europeans carrying MeV [reviewed in Ref. [42]]. Also, widespread measles epidemics in the Americas shortly before the description of distemper in dogs suggests that dogs may have been infected with MeV which then evolved into CDV [reviewed in Refs. [42,47,48]]. The examination of codon usage bias, or the frequency of codon use, of CDV demonstrates that the codon usage of CDV is closer to human codon usage bias than to canine codon usage bias, suggesting that CDV or its progenitor was originally adapted to and infected humans, supporting the theory that MeV from humans infected dogs and then evolved into CDV in dogs [[42], reviewed in Ref. [47]]. Examination of nucleotide sequences of one of the surface proteins (H) suggested that the current CDV strains circulating worldwide emerged in the 1880's, from a most recent common ancestor, in the USA [49]; however, this result, suggesting a later time and different place of CDV emergence, may be in question due to the limited availability of sequences used in the study and the loss of many original ancestral sequences [reviewed in Refs. [42,47]]. An alternative explanation would be that MeV infected dogs at multiple times in history and the most recent CDV strains were derived from the most recent incursion into dogs. CDV infection is known to occur in urban, rural, wildlife and aquatic environments worldwide [7,50]. The broad host range of CDV includes at least eight orders and over 20 families of mammals which are known to be susceptible to infection with CDV [51,52]. CDV may be unique among the morbilliviruses as it appears that individual host species living in low density populations can sustain the virus [6]. However, CDV has a propensity for host-switching, thus the unusually wide range of species which it can infect allows for many alternative hosts, meaning that CDV persistence likely involves a larger, multi-host community [21,50]. CDV mainly infects carnivores, however, infections of non-carnivorous javelina in the deserts of southern Arizona and non-human primates (Japanese macaques, rhesus monkeys, and long-tailed or cynomolgus macaques) in Japan and China have been recorded [reviewed in Refs. [38,53]]. The ability of CDV to infect a wide range of dog and non-dog species worldwide suggests that it is a potential conservation threat to endangered wildlife and a potential zoonotic threat to humans, especially following MeV eradication and complete withdrawal of MeV vaccination [[10,51,54,55], reviewed in Refs. [47,52]]. A recent in silico and in vitro examination of the cross-species transmission potential of this multi-host pathogen demonstrated, based on H protein interaction with cellular receptors, that there is cause for concern that CDV (specifically the Colombian strain) may be transmitted from dogs to humans [52]. CDV strains can be grouped into as many as 17–18 distinct lineages [51,52,54], and, like RPV, CDV strain differences give rise to varying virulence [7]. Signs of disease include fever, eye and nose discharge, hyperkeratosis (hardening) of the paw pads, in addition to digestive (vomiting, diarrhea), respiratory (coughing), and neurological signs [46,56,57]. CDV is also highly neurovirulent [6]. Chronic distemper meningoencephalitis can arise years following CDV infection due to viral persistence in the brain of dogs [1].

The first morbillivirus vaccine was developed, between 1922 and 1933 in Britain, against CDV, using both dogs and ferrets as experimental animals [reviewed in Refs. [4,56]]. The original vaccine involved a two-step process whereby dogs were vaccinated with a killed virus produced in dogs and treated with formalin, followed 14 days later with inoculation of live virus produced in ferrets [56]. Domestic dogs are now routinely vaccinated against CDV at 6–8 weeks, 10–12 weeks, 16–18 weeks, 12–16 months, and then every 1–2 years [58]. However, the complexity of CDV persistence within a large, multi-host community, containing a wildlife-domestic animal interface, poses a substantial challenge to control and/or elimination of this virus [[50], reviewed in Ref. [53]].

2.2.3. PPRV

PPRV has been recognized as an entity causing disease in sheep and goats (small domestic ruminants) since 1942, when it was first isolated in West Africa [1,41,59]. PPRV was long thought to be a variant of RPV until the late 1970's when it was recognized as a distinct morbillivirus [[60], reviewed in Refs. [59,61]]. PPRV may have emerged from RPV through natural passage of RPV in sheep and goats [reviewed in Ref. [59]]. There is only one serotype of PPRV and the current circulating strains of PPRV can be grouped into 4 lineages, reflective of their geographical origins [[11], reviewed in Refs. [59,62]]. The most recent common ancestor of these PPRV strains emerged in Nigeria in Western Africa in 1904, based on complete genome sequence [63]. Symptoms of infection include apathy, loss of appetite, fever, ocular and nasal discharge, cough, and diarrhea, and mortality, which can occur within 5–10 days following onset of clinical signs, can exceed 90% in previously unexposed populations [reviewed in Refs. [59,61]]. Goats are more susceptible than sheep which have a higher recovery rate [reviewed in Ref. [59]]. PPRV is not associated with neurological complications [reviewed in Ref. [59]]. Since the eradication of RPV, PPRV has expanded in both the hosts it infects, now also cattle, camels, and water buffalo (large domestic ruminants), and in its geographical range, with outbreaks in Morocco, Georgia, Mongolia, and Bulgaria [reviewed in Refs. [4,11,40,61,62]]. PPRV has also recently been found to infect some large and small wild ruminant species, to include African buffalo, dorcas gazelles, saiga antelope, Nubian ibex, gemsbok, among others [[11,64], reviewed in Ref. [62]]. PPRV was also found in tissues from a dead Asiatic lion in India, possibly indicating an expansion in host range [65]. A live attenuated PPRV vaccine was developed in the late 1980's for use in sheep and goats which confers life-long immunity following a single injection [reviewed in Refs. [11,61]]. This vaccine confers no unwanted side effects, is not transmitted from challenge animals to in-contact animals, and is effective against strains from all 4 PPRV lineages [11]. Following the eradication of RPV, PPRV has been proposed as a candidate for eradication, but its wide distribution (west, southern, and northern Africa, central and far-east Asia) and multiple,

highly mobile (local and regional trade, and seasonal movement of flocks between grazing areas) host species contribute to the difficulty expected to reach this goal [reviewed in Ref. [61]]. In 2015, a goal of eradicating PPRV through vaccination by 2030 was set by the international community [62]. A marked drop in the number of global outbreaks of PPRV was seen between 2015, with 3688 outbreaks, and 2019, with 1218 outbreaks. A post-vaccination level of 70% immunity is sought in order to break the endemic maintenance and spread of the virus within a small ruminant population. Currently, 21 countries with no new cases in five years, out of 66 countries with infection, can be declared PPRV free [62].

2.2.4. PDV

PDV was first recognized as a disease-causing agent affecting seals (pinnipeds) in 1988 when it caused the death of 20,000 harbor seals in northwestern Europe along the coasts of the North, Baltic, and Irish seas [[66], reviewed in Ref. [67]]. Gray seals were also affected by PDV, but mortality was low. The virus was thought to have been transmitted to these species of seals from arctic harp seals, the best candidate species to act as a viral reservoir in the North Atlantic, during a southward migration [[66], reviewed in Ref. [67]]. PVD most likely evolved from CDV following contact and viral transmission between artic seals and CDV-infected wolves, foxes, or other terrestrial carnivores [[2,21], reviewed in Ref. [48]]. However, a recent comprehensive time-calibrated phylogenetic analysis of CDV and PDV found that the two viruses diverged in the early seventeenth century with a most recent common ancestor dating to 1614 [68], putting into question the proposed evolution of CDV from MeV in the early 1700's as described above. There is now conclusive evidence of PDV also existing in the North Pacific and Western Arctic regions as well, and serologic evidence is accumulating for the existence of PDV in southern oceans [reviewed in Refs. [48,67]], and the spread of PDV from the North Atlantic to these other oceans appears to have occurred in the late twentieth and early twenty-first centuries [68]. Signs of disease include fever, eye and nose discharge, diarrhea, cough, weight loss, abortion, and neurological signs such as lethargy, head tremors, convulsions, and seizures [reviewed in Refs. [53,67]]. Severe infection can result in increased buoyancy which impedes normal swimming and diving [reviewed in Ref. [67]]. Focal thickening of the epidermis on the dorsal surface of flippers, head, trunk, and tail, similar to the hardening of paw pads in dogs infected with CDV, have been documented in harp and hooded seals infected with PVD. PVD can also infect sea otters (fissipeds) in addition to seals. Vaccination is being considered as a means to protect the endangered and potentially vulnerable Hawaiian monk seal from PDV infection, however vaccination of free-living marine mammals will be logistically challenging [reviewed in Ref. [67]].

2.2.5. CeMV

CeMV was recognized as a disease-causing agent affecting porpoises and dolphins between 1988 and 1992 [66]. It has since been found to infect whales, to include toothed and baleen whales, and the threatened and endangered species of fin and sperm whales [reviewed in Refs. [69,70]]. CeMV has now been found to have a worldwide distribution [reviewed in Refs. [4,70,71]]. Currently, there are 5 known strains of CeMV which are neither host nor location specific [70]. Typical signs of disease include pneumonia, lymphocyte depletion associated with immunosuppression, and encephalitis, as CeMV is neurotropic [[70], reviewed in Ref. [71]]. CeMV is most closely related to RPV, and as such is thought to have made the transition from land-to-sea, as occurred with its cetacean hosts which share a common terrestrial ancestor with ruminants [reviewed in Ref. [69]]. The discovery of the ability of CeMV to infect common seals and Eurasian otters, both of which display a mixed aquatic-terrestrial ecology, suggests that CeMV may be making the reverse transition from sea-to-land [reviewed in Ref. [69]].

2.2.6. FeMV

FeMV was first found in domestic cats in Hong Kong, mainland China, Japan, and the U.S., but has since been reported worldwide, and it is suggested to be associated with a chronic infection of the kidneys, tubulointerstitial nephritis [[72], reviewed in Refs. [4,9,73,74]]. FeMV is phylogenetically divergent from the other six species of morbilliviruses [reviewed in Ref. [9]]. The current strains of FeMV are classified into two genotypes [reviewed in Refs. [9,74]]. The gene organization (N–P/V/C-M-F-H-L) of FeMV is the same as the other morbillivirus species and it conforms to the rule of six, but its molecular biological and pathological features differ from the other morbilliviruses [[72], reviewed in Refs. [67,73,74]]. The 5′ trailer sequence of FeMV is 400 nucleotides in length, 10-fold longer than all other morbilliviruses [[72], reviewed in Ref. [74]]. Whereas the other morbilliviruses are spread via the respiratory route, the routes of infection for FeMV are unknown [reviewed in Ref. [9]]. To date, the pathogenesis of FeMV is not yet well understood, but persistent infection with FeMV has been suggested [reviewed in Refs. [73,74]]. Additionally, cross-species infections are thought to be possible based on the high genetic diversity of FeMV [reviewed in Ref. [73]].

2.2.7. Other morbilliviruses

Of the more recently isolated full-length morbillivirus genomes, morbillivirus genomes circulating in bats in Brazil may share a common ancestor with CDV in South America [[75], reviewed in Refs. [9,47]]. The gene organization of this virus is N–P/V/C-M-F-H-L, matching the other morbilliviruses [reviewed in Ref. [9]]. Additionally, newly identified viral species in domestic pigs (*Porcine Morbillivirus*, PoMV) and rodents (variously named), for which the pathogenicity and infectivity remain unknown, have been classified as morbilliviruses [[76], reviewed in Ref. [9]]. PoMV genome was isolated in Northern Mexico while viral genomes from rodents have been isolated in China, Belgium, Chile, and Argentina. PoMV is most closely related to CDV and PDV, while the rodent morbilliviruses are phylogenetically divergent. The gene organization of PoMV is the classical N–P/V/C-M-F-H-L, whereas the gene organization of the rodent morbilliviruses includes a hypothetical protein coding region between the F and H genes [[76], reviewed in Ref. [9]]. This hypothetical protein coding region did not show homology to any known proteins but did contain a transmembrane domain [76].

2.3. Relationship between the morbilliviruses

These seven-plus morbillivirus species likely evolved from a common ancestral virus through interspecies transmission and adaptation (Fig. 3) [reviewed in Refs. [32,38]]. Homologous recombination may also have played a role as potential recombination events have been proposed to have occurred between CDV strains [13,77]. Both RPV and MeV were described millennia ago, CDV was described centuries ago, while all other morbilliviruses were described in the 20th century, therefore, RPV, MeV, and CDV are the primordial members of the genus [reviewed in Ref. [42]]. A progenitor bovine morbillivirus, like RPV, is thought to be the likely progenitor of both RPV and MeV, as RPV and MeV are closely related to each other and only distantly related to CDV [8,21]. Comparative analysis of the deduced amino acid sequence of the homologous morbillivirus proteins shows a close relationship between MeV and RPV, and between CDV and PVD [13]. In fact, PVD and CDV are the most closely related of all the morbilliviruses [[2, 21], reviewed in Ref. [48]]. For the two morbilliviruses that infect aquatic mammals, PVD is most closely related to CDV, and CeMV is most closely related to RPV and PPRV, which parallels the phylogenetic relationship of their respective host species as seals are more closely related to terrestrial carnivores and the cetaceans are more closely related to the ruminants [14,16,78].

In support of the theory that all morbilliviruses evolved from a single common ancestral virus is the fact that the morbilliviruses use shared cell entry receptors, which are major determinants of the host range and tissue tropism of viruses [[12,39,40], reviewed in Ref. [10]]. Two cell surface receptors have been identified to which the H glycoproteins of morbilliviruses bind and viruses gain entry into cells: CD150 (SLAM/F1, signaling lymphocyte activation molecule F1) on immune cells, and PVRL4 (poliovirus receptor-like 4; nectin cell adhesion molecule 4, Nectin 4) on epithelial cells [[6,8,12,27,39,79–81], reviewed in Refs. [4,32,40]]. SLAM is thought to be the receptor that is key for entry of the virus into the host and establishment of infection through infection of lymphocytes, and Nectin 4 is thought to be the receptor that is key for exit of the virus from the host through infection of epithelial cells lining the lung and shedding of the virus into the lumen of the lung [[8,40], reviewed in Ref. [82]]. Nectin-4 is highly conserved across species while SLAM varies significantly [reviewed in Ref. [32]]. Although Nectin 4 expression has been demonstrated in the central nervous system of dogs (but not humans) and may therefore contribute to the neurotropism of CDV, the receptor used by CDV to infect astrocytes [83], and the receptor used by the other neurotropic morbilliviruses (MeV, PDV, CeMV) to infect the brain is unknown [reviewed in Ref. [82]]. Additionally, the cell entry mechanisms of FeMV are unknown, but due to differences in cell specificity, it has been suggested that non-SLAM receptors are utilized [reviewed in Refs. [9,73,74]]. Unlike FeMV, the bat morbillivirus (MBaMV) can use both SLAM and Nectin-4 as receptors [reviewed in Ref. [9]]. The beta4-beta5 hydrophobic groove of the H protein binds Nectin 4 while the beta5 and beta6 propeller blades of the H protein bind SLAM [84]. A number of interaction sites in the H protein specific for SLAM (D501, D503, D526, S528, R529, P550, Y520, and Y539) and/or Nectin 4 (Y520, Y537, Y539, Y454, L460, G461, and D501) (amino acid position is based on CeMV) have been identified which are conserved among all morbilliviruses [70]. It has been demonstrated that morbilliviruses can use SLAM and Nectin 4 receptors of non-host species, although with lower efficiency than that of the host species, without adaptation [[12,39], reviewed in Ref. [32]] or with minimal adaptation [40,55]. No adaptive alteration was required for CDV to utilize human Nectin 4 and only a single amino acid change/mutation in the CDV H protein receptor binding domain was

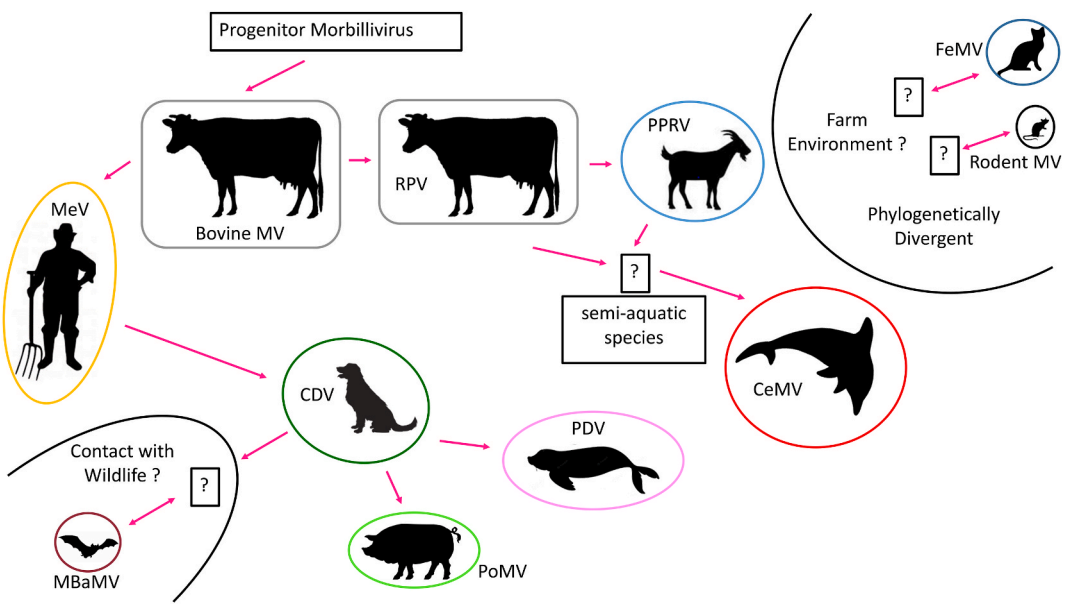


Fig. 3. Possible path of interspecies transmission for the morbilliviruses. An RPV-like bovine morbillivirus may be the archevirus of the entire viral group, having evolved from a progenitor morbillivirus. This figure does not reflect the fact that interspecies transmission of virus may have occurred multiple times, and may have occurred in both directions, in the process of virus establishing itself within a new host species. CDV, canine distemper virus; CeMV, cetacean morbillivirus; FeMV, feline morbillivirus; MBaMV, Myotitis bat morbillivirus; MeV, measles virus; MV, morbillivirus; PDV, phocine distemper virus; PoMV, porcine morbillivirus; PPRV, pestis-des-petits-ruminants virus; RPV, rinderpest virus.

required for CDV to utilize human SLAM [[55,57,85], reviewed in Ref. [48]]. Of note, CDV strains isolated using marmoset B95a cells may already be adapted to utilize human SLAM, as it is almost identical to marmoset SLAM [55,57]. Additionally, although infection of human cells by PPRV was found to be restricted due to deficient interactions (charge incompatibility and steric hindrance) with human SLAM, a single amino acid change/mutation in the PPRV H protein receptor binding domain was able to overcome this restriction [40]. Molecular evolution studies on complete sequences of CDV isolated from dog and non-dog hosts demonstrated that adaptations at two key H protein amino acid residues, located on the beta5 propeller blade and involved in binding SLAM, are associated with CDV host switching and spread to novel non-dog hosts [[86], reviewed in Refs. [48,67]]. Thus, the use of orthologous species-specific proteins as shared cell entry receptors across host species by the morbilliviruses increases the risk of future zoonotic morbillivirus infections and sharing two cell entry receptors may increase the risk even further [10,39].

Morbilliviruses are closely related antigenically; in general, antiserum to one morbillivirus will precipitate proteins from another morbillivirus (cross-reaction), and some cross-neutralization occurs [1,17]. Based on examination of the antigenic relationship between MeV, CDV and RPV using cross-reacting monoclonal antibodies, it has been suggested that RPV may be the progenitor, or archevirus, of the entire viral group, as it reacts with a wider range of monoclonal antibodies raised against other morbilliviruses [87]. Serum antibodies raised against one morbillivirus species will neutralize that homologous viral species to a higher titer than the other heterologous morbillivirus species, allowing for the determination of the morbillivirus species responsible for infection [reviewed in Ref. [67]]. Most neutralizing antibodies are directed against the H protein [3,8,11]. The generation of cross-neutralizing antibodies in a host may contribute to protection from zoonotic transfer [40]. Cross-protection from infection/vaccination is known to occur between more or less distantly related morbilliviruses but is dependent on the virus strains and the animals used [16,17]. Virulent MeV inoculation can protect dogs against CDV challenge [88]. Live attenuated MeV vaccination can protect dogs against severe CDV disease and death [1,15]. Conversely, human children inoculated with live egg-adapted CDV demonstrated no clinical reactions or signs of illness, developed variable levels of neutralizing antibody against CDV, and, in children under 5 years of age, showed a statistically significant three-fold reduction, compared to unvaccinated control children, in the subsequent incidence of measles acquired during a measles epidemic in Panama in 1959 [89,90]. Similarly, patients of a state hospital inoculated with live egg-adapted CDV in November of 1952 showed a three-fold reduction, compared to unvaccinated controls, in the subsequent incidence of measles acquired during a measles epidemic in 1955 [91]. The close similarity of one of the surface proteins (F) between MeV and CDV may explain this partial protection against disease induced by heterotypic vaccination [15]. Virulent CeMV inoculation protects dogs against virulent CDV challenge [16]. Virulent RPV inoculation can protect dogs against CDV challenge [88]. Virulent RPV inoculation and live attenuated RPV vaccination both protect ferrets against CDV challenge [reviewed in Ref. [41]]. Attenuated RPV vaccination can protect goats against PPRV challenge; the immunity lasted at least 12 months and the vaccinated animals were unable to transmit the challenge virus to susceptible, cohoused goats [92]. Conversely, attenuated PPRV vaccination can protect goats against virulent RPV challenge with vaccinated animals being unable to transmit the challenge virus to susceptible, co-housed goats and cattle [93]. Virulent PPRV inoculation, but not attenuated PPRV vaccination, can protect cattle against virulent RPV challenge [17]. Inactivated CDV vaccination can protect harbor seals against virulent PDV challenge [94,95]. Virulent CDV inoculation can protect cattle against RPV challenge [reviewed in Ref. [41]]. However, infection of cattle with virulent MeV failed to protect from subsequent RPV challenge [[88], reviewed in Ref. [41]]. More importantly, live attenuated MeV vaccination can partially protect cynomolgus macaques against virulent CDV challenge, demonstrated as restricted virus shedding, suggesting that continued MeV vaccination in humans could control potential future zoonotic morbillivirus infections [38].

2.4. Possible future morbillivirus emergence and zoonotic disease

Morbilliviruses are but one of many viruses that have, over the course of human history, emerged from an animal reservoir into the human population and resulted in disease. The emergence and disappearance of viral diseases is dependent on a complex interaction between the virus, the hosts (animals and humans), and the environment [reviewed in Refs. [96,97]]. Humans can become infected with viruses that emerge from other animal hosts resulting in disease in the human [reviewed in Ref. [97]]. Disease emergence may be determined by host and environmental factors with no requirement for alteration/adaptation in the virus [reviewed in Ref. [96]]. Two important factors in the emergence of new diseases and the spread of disease in humans are human population growth and movement [reviewed in Ref. [97]]. Both the entry of hosts into new habitats and the invasion of existing host habitats by new viruses may result in the emergence of new viral diseases [reviewed in Ref. [96]]. Disease emergence may occur following exposure of a highly susceptible host population to a virus which up until then had long been absent. For humans, improvements in personal hygiene and public sanitation have resulted in delaying the time of initial infection beyond when infants are passively protected by maternal antibody to an age with increased risk of developing clinical disease, thus causing the emergence of “new” viral diseases within an existing host population [reviewed in Ref. [96]]. Human activities that directly affect the environment, such as water storage practices, land management practices, road building, deforestation, environmental degradation, and war, also factor into the emergence of new diseases [reviewed in Ref. [97]]. Alternatively, evolution of the viral genome may also result in the emergence of a viral disease, either within the existing host population or within a newly exposed host species [reviewed in Ref. [96]]. Re-emergence of established viral diseases occur if evolution of the viral genome increases transmissibility or pathogenicity of the virus [reviewed in Ref. [97]]. Disappearance of a viral disease may be due to naturally occurring immunity resulting in fadeout of a virus, evolution of the viral genome resulting in waning of disease, or immunization [reviewed in Ref. [96]].

A study evaluating the role of agriculture and domestic animals in the evolution of human disease suggested that a broader force for human pathogen evolution could be ecological change, such as anthropogenic modification of the environment, resulting in novel interactions between humans and wildlife [26]. This is supported by the evidence that many current emerging infectious diseases are

associated with human modification of the environment [26,68]. Additionally, domesticated animals may function as a conduit for the transmission of disease from wildlife to humans rather than as a viral reservoir [26]. Increased rates of contact between wildlife and domestic animals, deterioration of natural habitats and loss of biodiversity, as well as globalization likely all contribute to the emergence and spread of zoonotic diseases [68].

The establishment of large, dense interacting populations of humans and animals set the stage for endemic morbillivirus infections within a species and for interspecies transmission of virus [42]. Interspecies transmission of virus may have occurred multiple times, and may have occurred in both directions, in the process of virus establishing itself within a new host species [42]. Interspecies transmission of morbilliviruses appears to be due to their intrinsic ability to adapt to new phylogenetically related host species with biological similarities [14]. Although it has recently been suggested that host-switching of virus may be opportunity driven in that given enough chances any virus can adapt to infect a new host [reviewed in Ref. [97]]. Experimentally, it has been demonstrated that morbilliviruses are typically less pathogenic in a new host, compared to the natural host, following cross-species infection, however, if the morbillivirus is transmissible within the new host, then adaptation of the virus can result in full virulence [reviewed in Ref. [38]]. This adaptation may take the form of mutations in the P/V/C proteins [48]. The new host may present challenges at the level of virus entry into cells, virus replication, and virus transmission from the new host [54], as well as challenging the virus to counteract the new hosts innate immune response [reviewed in Ref. [32]]. Two counteracting forces are in play during the adaptation of a virus to a new host species following interspecies transmission: the conservation of certain defined structures to preserve essential viral functions, and variations that favor attachment, cell entry, and replication within cells of the new species [13].

Currently, humans are effectively protected from contracting disease caused by animal morbilliviruses through vaccination against MeV. The stability of the MeV genome allows for this effective vaccination, and effective vaccination may someday allow for the eradication of MeV. At the point of eradication of MeV and the cessation of ongoing vaccination against MeV, the human population would again become susceptible to possible infection with animal morbilliviruses. If the infecting animal morbillivirus is able to adapt to replicate within humans and be transmitted human-to-human, then once again humans would be facing epidemics caused by a newly emergent morbillivirus. There is precedent for a related morbillivirus filling a vacated niche in the host expansion of PPRV into cattle from which RPV has been eradicated [reviewed in Refs. [4,11,40,61,62]]. The probability that the progenitor of CDV infected humans [[42], reviewed in Ref. [47]], the adaptability of CDV to infect a large multi-host community [21,50], and the ability of CDV to infect non-human primates (Japanese macaques, rhesus monkeys, and long-tailed or cynomolgus macaques) [reviewed in Refs. [38, 53]] suggest that CDV could once again become a human morbillivirus given the opportunity. Finally, newly discovered morbillivirus genomes in bats, pigs, and rodents suggests that there are as yet undiscovered morbilliviruses in animal hosts that, through interaction with susceptible individuals, could cause zoonotic disease in humans.

3. Summary

Morbilliviruses are highly adaptable viruses that have infected both humans and a wild range of animals. They are nonsegmented, linear, single-stranded negative-sense RNA viruses with an envelope. They are mainly transmitted via the respiratory route and their emergence depended on large, dense interacting populations of humans and animals. CDV may be unique among the morbilliviruses as its persistence likely involves a larger, multi-host community made up of individual host species living in low density populations through which transmission may more efficiently occur via direct contact. Although the animal reservoir from which the primordial morbillivirus emerged is unknown, a bovine morbillivirus, like RPV, may be the progenitor, or archevirus, of the entire morbilliviral group from which morbilliviruses spread worldwide. Our knowledge of morbilliviruses continues to expand with the discovery and classification of new morbilliviruses in new host species. Morbilliviruses are likely to continue to spread as they are transmitted to new hosts in which they are able to adapt and evolve and establish infection. An understanding of the spread of morbilliviruses from animal to animal, from animals to humans, and from humans to animals (Fig. 3) may give humanity a handle on how to prevent the emergence of future morbillivirus-caused zoonotic diseases.

Author contribution statement

All authors listed have significantly contributed to the development and the writing of this article.

Data availability statement

No data was used for the research described in the article.

Additional information

No additional information is available for this paper.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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