

ILSI Europe  
Report Series

# ANIMAL-BORNE VIRUSES OF RELEVANCE TO THE FOOD INDUSTRY



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REPORT

Commissioned by  
the ILSI Europe Emerging Microbiological Issues Task Force

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***ANIMAL-BORNE VIRUSES OF RELEVANCE  
TO THE FOOD INDUSTRY***

*by ILSI Europe Expert Group on Animal-Borne Viruses*

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REPORT OF AN ILSI EUROPE EXPERT GROUP  
COMMISSIONED BY THE ILSI EUROPE EMERGING MICROBIOLOGICAL ISSUES TASK FORCE

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# 1. EXECUTIVE SUMMARY

**T**his report deals with a number of highly contagious animal viruses, including both known zoonotics and those that have never been recorded in humans. The aim is to facilitate awareness in the food production industries of the basic facts, in a simple compact format. The relevance to human and pet food production is examined for thirteen highly contagious viral diseases of animals. The species considered as pets comprise cats, dogs, horses and birds. The viruses considered were selected and agreed by ILSI Europe after taking advice from all members of the expert group. For humans, potential exposure routes considered include contact with, and ingestion of, meat and other animal-derived foods, and occupational exposure for those working in the food production industry. For pets, potential exposure routes include exposure to animal-derived commercially prepared pet foods as well as contact with, and ingestion of, raw animal-derived products intended for human or pet food, or produced as waste from such food production. In the data section, for all viruses, basic information is given on the virus, host range, transmission routes, the disease in animals (and humans where applicable), current and possible future geographical distribution, persistence in the environment and detection, prevention and control in animals and foods.

Of the thirteen viruses considered, only five are considered to infect humans. Of these, only three have significant clinical effects; hepatitis E virus (HEV), highly pathogenic avian influenza virus (HPAIV) and Rift Valley fever virus (RVFV). Vesicular stomatitis virus (VSV) is considered a 'minor' zoonotic virus, and Newcastle disease virus (NDV) infection in humans is rare and atypical, when it may cause mild conjunctivitis.

Neither VSV nor RVFV has been identified in Europe. However, whilst VSV is only a minor zoonotic, RVFV can be fatal, and can be transmitted through infected carcasses and unpasteurised milk. It is mosquito-borne, and in endemic areas there are cryptic cycles where detection is difficult. If entry to the EU occurred, it could similarly remain undetected for a period after entry, leading to potential exposure for slaughterhouse workers, and via products containing unpasteurised milk. However, due to the susceptibility of previously unexposed European livestock, this undetected period is likely to be short.

Both HPAIV and NDV could be present undetected in poultry products in the EU at an early stage in an outbreak. NDV contaminated food is not considered to be a risk to humans or other mammals, but HPAIV can be fatal and, for undetected infections, the risks to those working directly with the birds, their fresh carcasses or their eggs could be high. There is also an indication that freezing might not inactivate the virus, although properly cooked poultry products (including eggs) are considered not to remain infectious.

The products mainly associated with HEV infection, which can be fatal in up to 25% of pregnant women, are raw or undercooked pig products and, occasionally, shellfish. Although not historically associated with human infection in the EU, the virus is found globally in pigs and there is little information on its survival in the environment or in food production processes. Therefore, the possibility of contamination and transmission must be seriously considered in slaughterhouses and food production processes.

HPAIV and NDV can affect pet birds, but food produced specifically for pet birds is unlikely to contain meat products; however, contamination of pet bird food by wild birds must be avoided. Although VSV does affect horses, their food does not contain meat products, and currently the virus is not found in the EU. Felines are susceptible to HPAIV by ingestion of raw meat, thus raw contaminated poultry products (possibly even after freezing) could potentially infect cats. In addition, antibodies to HEV have been found in cats, possibly associated with food related exposure, although no virus has been detected to date.

Viruses considered not to infect humans are African swine fever virus (ASFV), bluetongue virus (BTV), classical swine fever virus (CSFV), foot and mouth disease virus (FMDV), lumpy skin disease virus (LSDV), peste des petits ruminants virus (PPRV), sheep and goat pox virus (SandGPV) and swine vesicular disease virus (SVDV). However, it should not be forgotten that host and pathogenicity ranges can change over time due, for example, to mutation.

For legal movements of traded food products of animal origin, the majority of the viruses described here are subject to strict controls, greatly reducing the probability of contamination of legally imported products. In particular, all those which are either zoonotic or can infect domestic pets, except for HEV, are subject to legal import requirements. However, imported chicken and duck meats have tested positive for avian influenza viruses, and for HEV no certification is required, thus contamination of imported products with these viruses cannot be discounted. Illegal movement of food and food products obviously avoids all controls and safeguards, and contaminated food waste from ships and aeroplanes has been implicated in the spread of animal infection.

Two major areas of recommended research emerged from the virus experts involved in the production of this report. The first recommendation is for research applied directly to the virus itself, to develop improved diagnostics, especially in the field, as well as advancements in vaccine technology and therapeutic tools. The second is for population-based research, including epidemiological research, research into surveillance methodology, and the utilisation of risk assessment methodology. In addition, for the avian viruses, research into the factors which predispose and/or cause the shift from low to high pathogenicity is also recommended.

A key feature identified as relevant to both governments and animal producers is the requirement for good surveillance, diagnosis and control. These, plus contingency plans and (where feasible) eradication policies both in countries where a particular virus is endemic and in those to which it might spread, are also likely to be enhanced by international collaboration, agreement and initiatives. Increased use of risk assessments and modelling are also suggested as an aid to providing information for policy-making. For HEV, specific recommendations regarding additional testing and surveys are made.

For both livestock and food producers, appropriate hygiene and biosecurity measures should be employed, and food producers are recommended to source their products from known suppliers with appropriate quality standards. Those responsible for animals must be able to recognise and act upon signs of ill health, including production signs and egg drop. Appropriate sourcing and preparation of feed is also important. Also important are pre-slaughter veterinary inspection in the lairage, disinfection and/or proper disposal of slaughterhouse waste, and protection of slaughterhouse workers from aerosols and when handling newly killed carcasses. However, if an infected animal is slaughtered, post-mortem pH changes rapidly reduce the infectivity of HPAI and RVF viruses. Overall, staff awareness and knowledge are key at all stages.

Whilst unpasteurised milk products may be a transmission route for VSV and RVFV, there is a certain demand for such products. Where RVF is endemic, this may be considered unwise; elsewhere, labelling will allow customer choice. In the EU, all eggs from flocks with HPAI infection must be destroyed or heat-treated, and although the incubation period gives a brief window for infected eggs to enter the food chain, the resultant effects of rapid egg drop and abnormal eggs greatly reduces this probability. Little is yet known about the distribution and survival of HEV in food. For pet foods, regardless of the products from which they are manufactured, access by wild birds, rodents etc. to feed storage rooms must be prevented.

For consumers, basic kitchen hygiene, and thorough cooking of meat, meat products, eggs and egg products is generally considered to be the best way of avoiding any zoonotic virus from such products, and the same principles apply to reducing the risks from pet foods. With regard to the consumption of unpasteurised milk, or milk products, consumers living in, or visiting, countries where RVF is present should be advised of the additional risks. Similarly, the risks from the use of raw eggs need to be considered in the light of any prevailing HPAI disease situation. Consumers should be made aware that contaminated kitchen waste and scraps can transmit livestock disease.



## 2. INTRODUCTION

A number of highly contagious animal diseases have been in the news in Europe in recent years. These include highly pathogenic avian influenza, a regular issue over a number of years, bluetongue, after its unexpected emergence in Northern Europe in 2006, and foot and mouth disease, following the outbreak in the UK in 2001 and its spread to other European countries. In addition, there is the prospect of climate change, with the likelihood of increased average temperatures in Europe and of more extreme weather events such as floods and droughts. This has resulted in the scientific community giving thought to how the pattern of infectious diseases, particularly the vector-borne diseases of livestock, will be affected.

Not all highly contagious animal diseases are considered to be zoonotic; many have never been recorded to infect humans. Nevertheless, there is frequently a perception amongst consumers, and perhaps the media, that some non-zoonotic viruses can actually infect humans. A further consideration is that consumers might be expected to prefer their meat, eggs, milk and other dairy products to come from healthy animals, regardless of the zoonotic potential.

This report deals with a number of highly contagious animal viruses, including both known zoonotics and those that have never been recorded in humans. The aim is to facilitate awareness in the food production industries of the basic facts, in a simple compact format. The viruses considered were selected and agreed by ILSI Europe, after taking advice from virologists, and have been selected on the basis of a number of factors. They are either already present in Europe, or it is considered likely that they will be in the future. They cause highly contagious diseases in one or more species of commonly farmed livestock in Europe. Furthermore, they are either known to be zoonotic, or have been (or could become) associated with an incorrect perception in some sectors of society of having zoonotic potential, or with other adverse effects connected with food. On this basis, the following viruses are considered in this report:

- African swine fever virus (ASFV)
- Bluetongue virus (BTV)
- Classical swine fever virus (CSFV)
- Foot and mouth disease virus (FMDV)
- Hepatitis E virus (HEV)
- Highly pathogenic avian influenza virus (HPAIV) (considered together with low pathogenic avian influenza virus (LPAIV) where appropriate)
- Lumpy skin disease virus (LSDV)
- Newcastle disease virus (NDV)
- Peste des petits ruminants virus (PPRV)
- Rift Valley fever virus (RVFV)
- Sheep pox and goat pox virus (SandGPV)
- Swine vesicular disease virus (SVDV)
- Vesicular stomatitis virus (VSV)

The potential routes of exposure are considered for each virus. For humans, these routes include contact with, and ingestion of, meat and other animal-derived foods, and occupational exposure of those working in the food production industry. For pets, the potential exposure routes include exposure to animal-derived, commercially prepared pet foods and contact with, or ingestion of, raw animal-derived products intended for human or pet consumption, or produced as waste from such food production.

The remainder of the report comprises a data section, giving relevant information on each virus plus a section describing the conclusions reached by the viral experts regarding the implications for human and pet food, for trade and for a number of other connected issues.

### 3. DATA

#### 3.1 A brief description of the viruses

This section comprises, in tabular form, a brief description of each of the viruses under consideration. The description includes taxonomy and general characteristics, the major transmission methods, and the disease characteristics in the usual animal host range.

Table 1: African swine fever virus (ASFV)

Property	Information	References
Taxonomy and general characteristics	The only member in the family <i>Asfviridae</i> , genus <i>Asfivirus</i> . A complex icosahedral double-stranded linear DNA of ~170 kbp. The virus particle has an average diameter of 200 nm and is formed by several concentric structures with an external hexagonal membrane.	Murphy (1995); Montgomery (1921); Breese and DeBoer (1966); Blasco <i>et al.</i> (1989)
Animal host range	Natural hosts: African wild swine (Warthogs and Bush pigs). Domestic and wild pigs are the only naturally susceptible species. Vectors: soft ticks of <i>Ornithodoros</i> species ( <i>O. Moubata</i> and <i>O. Erraticus</i> ).	Sanchez Botija (1963); Plowright <i>et al.</i> (1970)
Transmission routes in animals	Natural infection: mainly oral and nasal from direct contact with infected pigs or contaminated materials, and infected tick bites (soft ticks of the genus <i>Ornithodoros</i> ). Experimental infection: other routes reported (e.g. cutaneous scarification and intramuscular, intravenous, subcutaneous or intraperitoneal injection).	Plowright <i>et al.</i> (1969); OIE (2002); McVicar (1984)
The disease in animals	Incubation periods vary widely from 2 to 19 days. Clinical signs, lesions and mortality, ranging from an acute form to a subacute, chronic and/or inapparent form. May resemble other swine haemorrhagic diseases e.g. hog cholera and erysipelas.	Mebus <i>et al.</i> (1983)

Table 2: *Bluetongue virus (BTV)*

Property	Information	References
Taxonomy and general characteristics	Family <i>Reoviridae</i> ; genus <i>Orbivirus</i> . A double-stranded RNA virus; 24 serotypes recognised to date; numerous strains.	ICTV (2008); Institute of Animal Health (2002)
Animal host range	All ruminants (i.e. sheep, goats, cattle, buffaloes, camels, antelopes, deer). Antibodies detected in some wild carnivores in Africa.	OIE (2002); Institute of Animal Health (2002)
Transmission routes in animals	Transmitted by vectors: biting midges of <i>Culicoides</i> species (competency varies with species). Transmission only at times when climate favourable to adult vector activity. (Rarely transmitted directly from vertebrate to vertebrate).	OIE (2002); Institute of Animal Health (2002)
The disease in animals	Sheep: incubation period is 4–8 days. Variable signs, acute to sub-clinical. Acute: fever, hyperaemia of mouth and nose mucosae, swelling of face and tongue, possible foot lesions, bloody diarrhoea, lesion necrosis. Mortality up to 70%. Goats: similar but usually milder. Cattle: infection usually sub-clinical.	OIE (2002); Institute of Animal Health (2002)

Table 3: *Classical swine fever virus (CSFV)*

Property	Information	References
Taxonomy and general characteristics	Family <i>Flaviviridae</i> , genus <i>Pestivirus</i> . Small single-stranded RNA virus, enveloped.	Moennig (2000); Beer <i>et al.</i> (2007)
Animal host range	<i>Suidae</i> only.	Liess (1981); Loan and Storm (1968)
Transmission routes in animals	Direct oro–nasal, and fomite contamination considered main routes. Initial introduction is often by feeding of unheated kitchen wastes (swill) containing infected pork. Transmission by semen is possible.	Ribbens <i>et al.</i> (2004); Kaden <i>et al.</i> (2003); Terpstra (1987)
The disease in animals	Incubation period: 3–10 days. A febrile, multi-systemic disease with extremely variable symptoms dependent on age (more severe in younger animals; often much harder to detect in fatteners and breeders) and virus strain. Acute, chronic and prenatal forms. Fever (>40°C), anorexia, conjunctivitis, respiratory and gastrointestinal symptoms, petechial bleeding (skin, mucous membranes), CNS signs (ataxia, convulsions), stillbirth, abortion, congenital tremor in piglets, high morbidity and mortality, secondary infections. Carriers: chronic infection – shedding for several months; persistent infection – permanent shedding.	Moennig <i>et al.</i> (2003); Paton and Greiser-Wilke (2003)

Table 4: Foot and mouth disease virus (FMDV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Picornaviridae</i> , genus <i>Aphthovirus</i> . Non-enveloped virus, capsid of 22 nm diameter. Genome is a single linear molecule of single-stranded RNA ~7.2–8.4 kbase in size. Seven immunologically distinct serotypes identified: O, A, C, SAT1, SAT2, SAT3, and Asia 1.	Racaniello (2001)
Animal host range	Cloven-hoofed animals, especially cattle, pigs, sheep, goats, buffalo.	Racaniello (2001)
Transmission routes in animals	Transmission usually by direct contact with infected animal. More rarely, by exposure to the excretions and secretions of acutely infected animals. Virus entry mainly through superior respiratory tract.	Olascoaga <i>et al.</i> (1999); OIE (2008)
The disease in animals	Incubation period: 2–14 days. Clinical signs vary from mild to severe. Typically, a vesicular condition of feet, buccal mucosa and, in females, mammary glands. Mortality low in adults; often high in young due to myocarditis. Pigs may develop severe foot lesions, while lesions in sheep and goats are less pronounced. Persistent infection possible (but not in pigs); infectious virus detected in oropharyngeal fluids after >28 days of infection, but transmission from cattle never demonstrated. Circumstantial evidence indicates persistently infected African buffaloes can, occasionally, infect animals in close contact. The carrier state in cattle is usually <6 months, but occasionally up to 3 years.	Olascoaga <i>et al.</i> (1999); OIE (2008)

Table 5: Hepatitis E virus (HEV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Hepeviridae</i> , genus: <i>Hepevirus</i> ; single-stranded RNA, 7.2 kbase. Four major genotypes, 1,2,3 and 4. Genotypes 1 and 2 infect humans only; 3 and 4 infect humans plus other species, and have high to very high sequence similarity to geographically related human strains.	Khuroo (1980); Bradley <i>et al.</i> (1987); ICTV (2005); Schlauder and Mushawar (2001); Banks <i>et al.</i> (2004)
Animal host range	HEV confirmed in pigs (including wild boar), Sika deer, mongooses. Antibodies detected in cattle, sheep, rats, cats; no virus yet detected in any of these species. (Two chicken HEV viruses are related but different from human/swine HEV; not considered further)	Clayson <i>et al.</i> (1995a); Tei <i>et al.</i> (2003); Takahashi <i>et al.</i> (2004); Nakamura <i>et al.</i> (2006)
Transmission routes in animals	Pigs: faecal–oral route. No information available on other routes.	Kasorndorkbua <i>et al.</i> (2004)
The disease in animals	No recorded disease in animals.	Sun <i>et al.</i> (2004)

Table 6: Highly pathogenic avian influenza virus (HPAIV)<sup>1</sup>

Property	Information	References
Taxonomy and general characteristics	Family <i>Orthomyxoviridae</i> , genus <i>Influenzavirus A</i> . Single-stranded RNA viruses. Subtypes classified by 16 antigenically distinct haemagglutinin (HA) antigens (H1 to H16), and 9 neuraminidase (NA) antigens (N1 to N9). HPAIV is restricted to subtypes H5 and H7 but low pathogenicity AI viruses (LPAIV) may include all virus subtypes, including H5 and H7, so HPAIV and LPAIV must be considered together.	Alexander (2007)
Animal host range	Many species of wild and domestic birds. Some mammals also susceptible and occasionally infected, e.g. pigs, cats, wild felines held in captivity, dogs.	OIE (2002); G. Cattoli (personal communication)
Transmission routes in animals	Bird to bird: usually via faecal–oral route (may be via fomites, e.g. litter, drinking water) or, possibly, by faecal–cloacal route. Excretion also possible via upper respiratory route. Bird to cat: believed due to ingestion of infected birds.	Webster <i>et al.</i> (1978); Alexander (2007); Kuiken <i>et al.</i> (2004)
The disease in animals	Domestic poultry: incubation period is 3–5 days HPAI: acute respiratory and nervous signs, with flock mortality up to 100% within a few days. Rapid drop in egg production with eggs often soft-shelled or misshapen. LPAI: spectrum from no signs to a milder disease, primarily respiratory and enteric signs, often plus rapid egg production drop/soft shells/misshapen eggs. Wild birds: with two exceptions (both involving H5 HPAIV), <i>virulent</i> viruses have never been detected in wild birds. Felines: Severe clinical signs; high mortality (natural and experimental infections, H5N1 HPAIV).	Capua and Alexander (2004); Rimmelzwan <i>et al.</i> (2006); OIE (2002)

1. Plus specific information on LPAIV where relevant, as is also the case in all other HPAIV tables in this report

Table 7: Lumpy skin disease virus (LSDV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Poxviridae</i> , sub-family <i>Chordovirinae</i> , genus <i>Capripoxvirus</i> (also includes sheep and goat pox). A slightly rounded, brick-shaped double-stranded DNA virus measuring ~320 × 260 nm. The external coat, made of tubular protein subunits or filaments, is surrounded by a double membrane.	Fenner <i>et al.</i> (1974); ICTV (2008)
Animal host range	Cattle and domestic buffalo ( <i>Bubalus</i> spp.); thin skinned European breeds are particularly susceptible. Experimentally, the giraffe ( <i>Giraffa camelopardalis</i> ) and impala ( <i>Aepyceros melampus</i> ) are susceptible. Lesions have also been seen in naturally infected impala.	Young <i>et al.</i> (1970); Weiss (1968)
Transmission routes in animals	Thought to be due to mechanical transmission by biting flies (virus isolated from <i>Stomoxys</i> and <i>Musca</i> spp) feeding on skin nodules, as fly blood titre is low. Experimental transmission using <i>Aedes aegypti</i> . Transmission to calves via milk has been documented. Direct contact transmission is inefficient.	Chihota <i>et al.</i> (2003); Carn and Kitching (1995a)
The disease in animals	Incubation period: approximately 10 days. A febrile, debilitating disease characterised by lachrymation and generalised lymphadenopathy. There may be salivation, nasal discharge, subcutaneous oedema of legs, and papular skin lesions. Erosions may be seen on mucosae of nose, mouth, respiratory tract and reproductive organs. Mortality up to 10%; morbidity 5–45%. Mastitis, temporary or permanent infertility, prolonged debility and severe damage to hides result in economic losses.	Haig (1957); Carn and Kitching (1995b); OIE (2002)

Table 8: Newcastle disease virus (NDV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Paramyxoviridae</i> , genus <i>Avulavirus</i> ; avian paramyxovirus serotype 1 (APMV-1) viruses. Single-stranded RNA genome with nucleocapsid.	Al-Garib <i>et al.</i> (2003); Lamb <i>et al.</i> (2000)
Animal host range	Many types (from many orders) of birds including feral birds (often low pathogenicity) and domestic poultry (often high pathogenicity). Reported to non-reproductively infect animals other than birds, including reptiles (clinical signs would be atypical)	Alexander (2001); Al-Garib <i>et al.</i> (2003)
Transmission routes in animals	Inhalation and ingestion. Direct contact with infected birds or carcasses and indirect contact (via fomites) due to contamination (especially with faeces, also mucus) from infected birds. Ingestion of infected poultry products (including frozen products) used as food (e.g. in 'swill') has historically caused infection in poultry. Airborne spread may be possible over short distances.	Alexander (2001); Al-Garib <i>et al.</i> (2003)
The disease in animals	Incubation period: 4–6 days. Strains divide into 5 pathotypes with a wide variation in clinical signs, depending on virulence of strain, and on host species. They are asymptomatic, lentogenic (mild respiratory signs), mesogenic (moderate respiratory signs, high mortality in young birds), neurotropic velogenic (nvND; acute neurological signs, respiratory distress often with high mortality), velogenic (vvND; acute lethal infection, sudden death may be first sign). Mortality may be up to 60%.	Alexander (2001); Al-Garib <i>et al.</i> (2003), OIE (2002)

Table 9: Peste des petits ruminants virus (PPRV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Paramyxoviridae</i> , genus <i>Morbillivirus</i> . Single-stranded RNA virus, antigenically close to rinderpest virus.	Lefèvre and Diallo (1990); ICTV (2008); Gibbs <i>et al.</i> (1979)
Animal host range	Small ruminants (especially goats with breed-linked susceptibility), captive wild ungulates, such as <i>Gazellinae</i> (dorcass gazelle), <i>Caprinae</i> (Nubian ibex and Laristan sheep), <i>Hippotraginae</i> (gemsbok). Cattle and pigs: sub-clinical infections.	Lefèvre and Diallo (1990); Diop <i>et al.</i> (2005); Furley <i>et al.</i> (1987); Mornet <i>et al.</i> (1956)
Transmission routes in animals	Nasal and oral by aerosol or feed after direct contact, via the epithelium of the upper and lower respiratory tract. No carrier state.	Couacy-Hymann <i>et al.</i> (2007); Opasina and Putt (1985)
The disease in animals	Incubation period: 3–10 days. Fever (40–41°C), serous ocular and nasal discharge becoming mucopurulent, respiratory distress, necrosis on the visible mucous membranes, severe diarrhoea, bronchopneumonia, hypothermia, death within 5–10 days. Morbidity 10–80%; mortality 0–50%. Outbreaks more frequent in rainy, and dry, cold seasons.	Hamdy <i>et al.</i> (1975); Lefèvre and Diallo (1990); Roeder <i>et al.</i> (1994); OIE (2002)

Table 10: Rift Valley fever virus (RVFV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Bunyaviridae</i> , genus <i>Phlebovirus</i> . One of five genera, which includes the sand fly fevers. An enveloped spherical virus up to 120 nm diameter; single-stranded RNA genome.	Daubney <i>et al.</i> (1931); Bishop <i>et al.</i> (1980); ICTV (2008)
Animal host range	Domestic ruminants including sheep, goats, cattle and camels as well as wild African buffalo ( <i>Syncerus caffer</i> ) are affected. In general, European breeds are highly susceptible. Experimental aerosol infection of puppies and kittens.	OIE (2002); Keefer <i>et al.</i> (1972)
Transmission routes in animals	Arthropod transmission, biological and mechanical. Main vectors, mosquitoes; in Africa 23 species from five genera involved. Mechanical transmission by ticks, midges and biting flies. Transovarially infected floodwater-breeding <i>Aedes</i> mosquitoes are primary cycling hosts; maintenance cycles involve other mosquito species. Contact transmission of little importance.	Gargan <i>et al.</i> (1988); McIntosh (1972)
The disease in animals	Incubation period: lambs as short as 12 hr, calves and older lambs up to 72 hr. Clinical disease peracute to inapparent; age- or breed-dependent. Clinical signs: fever to 42°C, anorexia, abdominal pain, jaundice, nasal discharge, haemorrhagic diarrhoea and recumbency. Abortions, from 40% to 100%. Neonatal mortality in lambs and kids, 70–100%. Slightly more resistant calves develop icterus before death. Wild ruminants experience viraemia. The African buffalo ( <i>Syncerus caffer</i> ) may also abort.	Gerdes (2002)

Table 11: Sheep pox virus and goat pox virus (SandGPV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Poxviridae</i> , sub-family <i>Chordopoxvirinae</i> , genus <i>Capripoxvirus</i> . Slightly rounded brick-shaped (~290–320 × 260–280 nm), enveloped with complex symmetry (tubular protein surrounded by double membrane); double-stranded DNA genome ~150 kbp.	Tulman <i>et al.</i> (2002); Murphy <i>et al.</i> (1999); Kitching and Smale (1986); Ghaboussi (1978)
Animal host range	Sheep and goats. Isolates highly host-specific, but this varies from isolate to isolate. Kenyan and Yemeni isolates and an Omani sheep isolate infect sheep and goats equally readily. However, isolates from the Middle East, India and Nigeria are host-specific in sheep or goats.	Kitching and Taylor (1985); Kitching <i>et al.</i> (1986a, 1989); Soman <i>et al.</i> (1985); Rafiyi and Ramyar (1959); Bakos and Bragg (1957)
Transmission routes in animals	Commonly, respiratory tract via aerosol during direct or close contact, also via skin abrasion. Virus found in all secretions, excretions, and scabs. Mechanically by biting insects e.g. <i>Stomoxys calcitrans</i> , tsetse fly. Experimentally by inoculation.	Geering <i>et al.</i> (1995); Munz and Dumbell (1994); Kitching and Mellor (1986); Webbs <i>et al.</i> (1980); Bakos and Bragg (1957)
The disease in animals	Incubation period: 8–13 days. Fever >40°C, dyspnoea, depression, anorexia, conjunctivitis, rhinitis. Enlargement of all superficial lymph nodes and lung necrotic lesions. In severe cases, pneumonia, enlargement of udder, abortion and death. Skin lesions progress through macular, papular, vesicular, pustular, scabby stages and scars. Morbidity 75–100%, mortality 10–58%.	OIE (2002); Rao and Bandyopadhyav (2000); Jubb <i>et al.</i> (1993)

Table 12: Swine vesicular disease virus (SVDV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Picornaviridae</i> , genus <i>Enterovirus</i> . Single-stranded RNA virus with a single serotype having four antigenic or genomic lineages.	Nardelli <i>et al.</i> (1968); King <i>et al.</i> (2000); Brocchi <i>et al.</i> (1997); ICTV (2008)
Animal host range	Swine are the only susceptible species.	OIE (2002)
Transmission routes in animals	Direct contact with infected pigs or indirect contact via contaminated materials, environment, fomites. Transmission route: oral (main), skin and mucosal lesions. Infectious sources: faeces (major), vesicular fluid, contaminated meat scraps and swill.	Lin and Kitching (2000); Dekker (2000)
The disease in animals	Incubation period 2–7 days. Signs may be mild or even asymptomatic. Young animals more severely affected. Temperature increase ~2–4°C. Sudden lameness in several animals in group with limping, arched back, and refusal to move even for food. Vesicles on snout, coronary band, interdigital and (rarely) buccal cavity, tongue, teats (similar to those due to FMDV). Vesicle rupture leading to erosions. Foot-pads may loosen, particularly in young stock, which may lose the horny hoof. Morbidity in herds may be low but high in pen/contact groups. Recovery usually within 1 week, maximum 3 weeks. No mortality, no carrier state.	Loxam and Hedger (1983); Lin and Kitching (2000); Dekker (2000)



Table 13: Vesicular stomatitis virus (VSV)

Property	Information	References
Taxonomy and general characteristics	Family <i>Rhabdoviridae</i> , genus <i>Vesiculovirus</i> . The genome is a single linear, negative-sense molecule of single-stranded RNA; ~11–15 kb in size. Two serotypes: New Jersey (NJ), Indiana (Ind).	Rose and Whitt (2001)
Animal host range	Mainly horses, cattle and pigs. Sheep, goats and many other wild species can also be infected.	Rose and Whitt (2001); OIE (2002)
Transmission routes in animals	Transmitted directly by transcutaneous or transmucosal routes. Virus also isolated from sand flies and mosquitoes, indicating that it could be insect-borne.	Rose and Whitt (2001); OIE (2002)
The disease in animals	Incubation period: 2–21 days. Usually a fever. Excessive salivation, characteristic vesicles may be on lips, nostrils, hooves, teats, in mouth. Vesicles swell and rupture leading to painful ulcers and erosions, which can cause anorexia, refusal to drink, and lameness. Dairy cattle with teat lesions may develop mastitis from secondary infections. Morbidity 5–90%. Recovery generally ~2 weeks. Most cases occur in adults; uncommon in young cattle and horses under a year of age. Mortality close to zero; higher mortality rates have been seen in some pigs infected with NJ strain.	Panaftosa (2006); OIE (2002)

### 3.2 Human susceptibility and the disease in humans

Although all the above viruses are considered to be highly contagious in animals, not all are considered to be zoonotic. Table 14 lists the five viruses that are considered in this report to be zoonotic, along with the relevant transmission routes and the clinical picture of infection in humans. Table 15 lists the eight viruses that are not considered to be zoonotic.

Table 14: Those viruses that are considered by the authors of this report to be zoonotic, their transmission routes, and the clinical picture of infection in humans

Property	Information	References
<i>Hepatitis E virus (HEV)</i>		
Transmission routes	Animal-human: ingestion of infected meat and probably shellfish, or contaminated water. Human-human: faecal-oral is the major route, also vertical transmission, blood transfusion, secondary/intra-familial spread.	Bile et al. (1994); Chan (1995); Tei et al. (2003); Khuroo et al. (2004); Arankalle et al. (2000)
The disease in humans	Incubation period 2–9 weeks. Acute icteric hepatitis; virus shed into faeces. Mortality 0.5–2% (but pregnant women up to 25%). Most cases in developed regions acquired by travel to endemic region but increasing numbers of autochthonously acquired disease reported from developed regions.	Clayson et al. (1995b); Appleton et al. (2007); Ijaz et al. (2005); Tei et al. (2003); Takahashi et al. (2004)
<i>Highly pathogenic avian influenza virus (HPAIV)</i>		
Transmission routes	Direct exposure to live birds or carcasses, e.g. during culling and disposal in outbreak, probably by inhalation. Transmission via handling or ingestion of raw and undercooked poultry products has also been suggested.	Perdue and Swayne (2005); Van Reeth (2007)

Property	Information	References
The disease in humans	H5N1/H7N7 HPAI: Severe flu-like syndrome with high fever, acute respiratory signs and enteric signs also reported. Mortality rates can be up to 60%. LPAIV (H7 and H9 subtypes) can also infect humans to give conjunctivitis and a mild flu-like syndrome.	Van Reeth (2007) WHO (2009)
<i>Newcastle disease virus (NDV)</i>		
Transmission routes	Reported to infect humans non-reproductively, but rare atypical event.	I. Brown (personal communication)
The disease in humans	Rarely results in clinical signs. Occasional mild conjunctivitis.	
<i>Rift Valley fever virus (RVFV)</i>		
Transmission routes	Mosquito bites. Contact with infected animals and aborted foetuses through abraded skin and mucous membranes; aerosols during ritual slaughter. Raw meat fallen below pH 6 is safe (virus destroyed). Transmission through raw milk is possible.	Meegan <i>et al.</i> (1979)
The disease in humans	Incubation 3–7 days. Ranges from fever, chills, joint pains, nausea with recovery, to haemorrhagic fever, liver necrosis, icterus, bleeding and death. Complications include encephalitis, coma with residual damage, retinitis, retinal haemorrhage and macular oedema with residual damage.	Peters and Meegan (1994)
<i>Vesicular stomatitis virus (VSV)</i>		
Transmission routes	Transmission is poorly understood, but could occur via contact with animals. There are reports of accidental infection of workers, while performing necropsies or in the laboratory.	Rose and Whitt (2001)
The disease in humans	Considered a 'minor' zoonosis. The incubation period in humans is usually 24–48 hr or as long as six days. Acute influenza-like symptoms (e.g. fever, muscle aches, headaches, malaise), lesions rare but resemble herpes virus when they occur. Disease course is 4–7 days. No mortality.	Rose and Whitt (2001); OIE (2002)

Table 15: Those viruses which are not considered by the authors of this report to be zoonotic

African swine fever virus <sup>1</sup> (ASFV)	Lumpy skin disease virus (LSDV)
Bluetongue virus (BTV)	Peste des petits ruminants virus (PPRV)
Classical swine fever virus (CSFV)	Sheep pox and goat pox virus (SandGPV)
Foot and mouth disease virus <sup>2</sup> (FMDV)	Swine vesicular disease virus (SVDV)

1. First described in 1921; to date (2006) no human cases reported (Sánchez-Vizcaino, 2006).

2. Not classed as zoonotic by the authors of this report; humans not considered natural hosts. Another virus (enterovirus 71) causes hand, foot and mouth disease in humans (ICTV, 2008), sometimes confused with foot and mouth disease (Olascoaga *et al.*, 1999).

### 3.3 Geographical distribution and ecological factors

This section describes the current known geographical distribution, any current trends in distribution and any factors considered likely to affect distribution in the future. In addition, information is given on the persistence of the virus in the environment. Table 16 gives the information for those viruses known to be zoonotic. Table 17 gives briefer details for those not considered zoonotic.

Table 16: Geographical distribution and related information for those viruses considered as zoonotic

Property	Information	References
<i>Hepatitis E virus (HEV)</i>		
Current geographical distribution	Endemic in humans in many developing regions of the world including Indian sub-continent, Asia, Africa and South and Central America. High human prevalence (~10%) in several developed regions also suggests endemnicity. Globally ubiquitous amongst pigs — no country has demonstrated freedom.	Labrique <i>et al.</i> (1999); Ijaz <i>et al.</i> (2005)
Current and recent changes of distribution	For human infection, no widespread trends reported in endemic regions; epidemics often occur in conflict zones with compromised water supplies. Increased diagnosis in several industrialised countries over last 2–3 years, but unclear how much is due to improved surveillance. No trends recorded in pigs.	Appleton <i>et al.</i> (2007); Ijaz <i>et al.</i> (2005); Dalton <i>et al.</i> (2007)
Persistence of virus in the environment	Environmental survival is unknown, largely due to the difficulty in cultivation of the virus in vitro. One study shows ~95% of virus inactivated after 1 hr at 60°C.	Emerson <i>et al.</i> (2005)
Factors likely to affect distribution in the future	Conflict in developing regions. Several vaccines are undergoing stage III clinical trials and may have a dramatic impact in endemic regions.	Purcell <i>et al.</i> (2003)
<i>Highly pathogenic avian influenza virus (HPAIV)</i>		
Current geographical distribution	Worldwide. HPAIV (and LPAIV) isolated in domestic poultry in Asia, Europe, Africa, Australia and the Americas.	OIE (2002)
Current and recent changes of distribution	Increasing outbreaks with potentially zoonotic impact (HPAIV and H9 LPAIV), and an expanding geographical distribution.	Capua and Alexander (2004); Alexander (2007)
Persistence of virus in the environment	Infectivity can remain in lake and pond water, duration temperature-dependant. Infective viruses detected after 102 and 207 days at 28°C and 17°C, respectively; persistence of H5 and H7 inversely proportional to salinity. In faeces, survival possible for >44 days.	Webster <i>et al.</i> (1978); Stallknecht <i>et al.</i> (1990); Alexander (2007); Brown <i>et al.</i> (2007)
Factors likely to affect distribution in the future	Change in wild birds' movements due to e.g. climate changes. Change in poultry production systems, e.g. poultry density changes, free range.	

Property	Information	References
<i>Newcastle disease virus (NDV)</i>		
Current geographical distribution	Endemic in many areas of the world (e.g. Africa, Asia, Central and South America). Sporadic incursions in most non-endemic countries, including the EU.	Alexander (2001)
Current and recent changes of distribution	First described in 1926; 3 pandemics identified since then. Possibility of recurrence. Spread probably due to global trade and increases in travel.	Alexander (2001); Doyle (1927); Alexander (1988a,b,c)
Persistence of virus in the environment	Data scarce. Can survive in frozen poultry carcasses, and at refrigerator temperatures, for 4 months in bone marrow and muscle. Can survive in eggs for months at room temperature, and >1 year at 4°C. Similar survival likely for faeces. Can be transmitted by fomite contamination.	Alexander (2001); Lancaster (1966)
Factors likely to affect distribution in the future	Increased trade in live poultry, live bird markets, legal and illegal trade in exotic birds, use of poultry manure as fertiliser, increase in free-range birds. Virus widespread in pigeons, which may affect domestic poultry. Affected by local vaccination policy.	Alexander (2001)
<i>Rift Valley fever virus (RVFV)</i>		
Current geographical distribution	Sub-Saharan Africa, mostly East and South coasts, also Egypt and West coast of Senegal and Mauritania. Game involved in cryptic cycles. Distribution follows the great Rift Valley floor. Madagascar and recently Saudi Arabia and Yemen.	Daubney <i>et al.</i> (1931); Meegan <i>et al.</i> (1979); Zeller <i>et al.</i> (1997)
Current and recent changes of distribution	Activity is cyclical in Africa in sub-tropical and tropical areas depending on rainfall. Last documented extension of range was from Horn of Africa to Arabian peninsula in 2000 following flooding in Africa.	EFSA (2005)
Persistence of virus in the environment	Virus dormant in transovarially infected <i>Aedes</i> eggs in dried mud; activation requires persistent rainfall and sustained flooding.	Davies <i>et al.</i> (1985)
Factors likely to affect distribution in the future	Countries with irrigation schemes, dams or large river delta systems are under threat from wind borne vectors or trade in viraemic animals. Climate change, resulting in extremes of weather and globalisation of trade, likely to expand range.	Porter (1999)
<i>Vesicular stomatitis virus (VSV)</i>		
Current geographical distribution	Restricted to the Americas, mainly to North, Central, and the northern part of South America.	Panaftosa (2006); OIE (2002)
Current and recent changes of distribution	None	Panaftosa (2006)
Persistence of virus in the environment	Survives for long periods at low temperatures; stable between pH 4.0 and 10.0. The presence of antibodies in several species of wild animals suggests a natural reservoir.	Panaftosa (2006); OIE (2002); AusVetPlan (2000)
Factors likely to affect distribution in the future	Climatic and ecological changes could have an effect.	

Table 17: Geographical distribution and related information for those viruses not considered as zoonotic

Property	Information	References
<i>African swine fever virus (ASFV)</i>		
Current geographical distribution, trends and factors which may affect this	Africa; endemic in many sub-Saharan countries, and spreading into new areas, e.g. Congo, Gambia, Ghana, Madagascar, Senegal. In Europe, endemic in Sardinia (Italy) with outbreaks increasing; eradicated elsewhere. Incursions have occurred in several countries outside of Africa, and may continue.	OIE (2002); OIE (2008)
Persistence of virus in the environment	Very resistant to heat or pH; inactivation by 60°C for 20 min; inactivation at pH <3.9 or >11.5, or by chemical treatment.	OIE (2002); Stone and Hess (1973)
<i>Bluetongue virus (BTV)</i>		
Current geographical distribution, trends and factors which may affect this	Originally thought only to be in Africa. Over last 50 years, area has increased. Now, the Middle East, Indian sub-continent, China, USA, Mexico, the Mediterranean basin and, most recently, northern Europe (Belgium, UK etc.). Climate change likely to affect pattern and distribution (via vector lifecycle) and possible expansion of competent vector range once infected.	OIE (2002); IAH (2002); Defra (2007)
Persistence of virus in the environment	Virus can persist, even over winter, in vectors if conditions are suitable for survival of the adult active vector.	
<i>Classical swine fever virus (CSFV)</i>		
Current geographical distribution, trends and factors which may affect this	Present in parts of Africa, Central and Southern America, Asia and Europe. 'Hemispheric Plan' to eradicate the disease by 2020 from the Americas. Occasional recurrence in industrialised countries; attempts made to eradicate from EU. Improvement of socio-economic factors facilitates control in non-professional pig holdings (backyard pigs).	Edwards <i>et al.</i> (2000); OIE (2005, 2007a)
Persistence of virus in the environment	Survives well in cold conditions, and is partially resistant to moderate heat (up to 56°C). Inactivated by pH <3.0 or >11.0.	OIE (2002)
<i>Foot and mouth disease virus (FMDV)</i>		
Current geographical distribution, trends and factors which may affect this	Historically a global disease, which has affected most regions and is still present in many parts of the world. Some countries are traditionally free, e.g. Australia, New Zealand, Japan. Control strategies have led to eradication from many countries, e.g. North America, Europe. In South America, a control programme has greatly reduced the number of outbreaks; many countries free with or without vaccination. Distribution is likely to diminish with the spread of eradication programmes.	Olascoaga <i>et al.</i> (1999)
Persistence of virus in the environment	Can persist in contaminated fodder and the environment for one month or more, depending on climatic conditions. Inactivation; above 50°C; pH <6.0 or >9.0; chemical treatment.	Olascoaga <i>et al.</i> (1999); Cottral (1969)

Property	Information	References
<i>Lumpy skin disease virus (LSDV)</i>		
Current geographical distribution, tends and factors which may affect this	Africa: established south of the Sahara, North Africa and the Middle East; spread to Madagascar and Mauritius. An outbreak in Israel in 1989 (wind-borne vectors from Egypt suspected). Outbreaks associated with high rainfall and insect activity. Spread linked to trade, animal- and wind-borne vector movement. Experimentally <50% of animals develop visible disease so movement of infected cattle that appear normal is possible. Climatic conditions that favour increased vector activity could lead to spread.	Von Backstrom (1945); Yeruham <i>et al.</i> (1995); Woods (1988)
Persistence of virus in the environment	Stable in the environment between pH 6.6 and 8.6; resistant to heat and, when dry, withstands 100°C for 10 min. Persists in hides at room temperature for 18 days.	Weiss (1968)
<i>Peste des petits ruminants virus (PPRV)</i>		
Current geographical distribution, tends and factors which may affect this	Africa, spreading over the equator and the Sahara, the Arabian Peninsula, most Middle East countries, India and South West Asia. Present in the European part of Turkey since 2004. PPRV can be spread via unofficial trade. Domestic animals are a reservoir; wildlife act as sentinels.	Taylor (1984); Shaila <i>et al.</i> (1989); Diallo (2003); FAO (2005)
Persistence of virus in the environment	Resists 56°C for 60 min or 60°C for 30 min. Stable between pH 4.0 and 10.0. Survives for long periods in chilled and frozen tissues.	OIE (2002)
<i>Sheep pox and goat pox virus (SandGPV)</i>		
Current geographical distribution, tends and factors which may affect this	Endemic in North and Central Africa, South West and Central Asia, Indian subcontinent, parts of People's Republic of China (2002, 2003). Frequent outbreaks in past 10 years; Southern Europe (Bulgaria, 1996; Greece, 2000), Russia (2002, 2003), Vietnam (2005), due to increase in livestock trade. Future spread likely due to climate changes (increasing temperature; decreasing humidity and rainfall) as drought increases virus survival and livestock migration, and vectors may spread.	Esposito and Fenner (2001); Carn (1993), OIE (2005, 2007b); Bhanuprakas <i>et al.</i> (2005); Kitching and Mellor (1986)
Persistence of virus in the environment	Resistant to desiccation, persistent for up to 2-3 months on wool and 3 months in dry scabs. Sensitive to direct sunlight, but may persist for up to 6 months in a cool and dark environment. Sensitive to high humidity.	Bhanuprakas <i>et al.</i> (2005); Kitching (2000); Geering <i>et al.</i> (1995); Jubb <i>et al.</i> (1993)
<i>Swine vesicular disease virus (SVDV)</i>		
Current geographical distribution, tends and factors which may affect this	Currently declared only in Italy (Europe), on the basis of results of sero-surveillance and virus surveillance. SVD-free status only based on absence of clinical evidence in most of the world. Difficult to predict changes in distribution, as it is frequently subclinical and there is an absence of surveillance (serological and virological) in most countries. In the EU few member states have SVDV surveillance.	Bellini <i>et al.</i> (2006); Brocchi <i>et al.</i> (1997)
Persistence of virus in the environment	Highly resistant in the environment, also in absence of susceptible animals. Resistant at pH 2-12, preserved by refrigeration and freezing, resistant to fermentation and smoking processes. Inactivated at 56°C for 1 hr.	Mann (1981); Lin and Kitching (2000)

### **3.4 Detection, prevention and control in animals**

#### **3.4.1 Detection and identification of diseases and viruses**

Detection of diseases in animals, and identification of the causes, will vary depending upon a number of factors. The first indication is the observation of a change from the normal condition, which may be in the animal itself, or in some production factor (e.g. milk yield, egg yield, egg shape). The likelihood of this observation depends on the standard of husbandry, which in turn depends upon the knowledge of the stockperson, and the management system in place. Further investigation of the cause will depend upon the resources available locally and their affordability (e.g. veterinary surgeon, laboratory etc.), and the value of the animals concerned. In some areas, there may be targeted surveillance of particular viruses, or herd health schemes, making early detection of some viruses more likely.

Livestock for legal importation into the EU must comply with a variety of regulatory requirements, including certification of freedom from specific viruses at the herd/flock, region, or country level as appropriate. This system is subject to inspection by the EU Food and Veterinary Office (FVO) inspectors. Illegal importation, by definition, is not subject to such safeguards.

For confirmation of the presence of specific viruses, either for disease diagnosis, or for routine surveillance, laboratory tests are generally used. The diagnostic efficacy of a laboratory test depends upon its sensitivity and specificity, and for some tests there is data available on these values. Diagnostic sensitivity is defined as the proportion of truly infected animals that test positive, and diagnostic specificity is defined as the proportion of truly uninfected animals that test negative (Saah and Hoover, 1997).

Tests fall into two broad categories, those that detect antibodies produced by the host, and those that directly detect the presence of viral antigen or whole virus. Many of the immunologically based tests can detect either antigen or antibody, depending upon the specific method used, and there are a wide variety of types of immunological test, making this a complex area. Table 18 gives brief details of the diagnostic tests generally used and, where available, numerical values for diagnostic sensitivity and specificity are given, but it is not the intention of this report to describe these in detail. For all the viruses described except HEV, further details of diagnostic tests available, and their methods, can be found on the website of the World Organisation for Animal Health (Office International des Epizooties, OIE) (OIE 2008). However, all numerical estimates of test sensitivity and specificity, where available, are from other references indicated in the table.

Table 18: Overview of diagnostic tests commonly used plus diagnostic sensitivity and specificity where available

Virus	Test	Sensitivity <sup>1</sup> (%)	Specificity (%)	References
<i>Zoonotic group</i>				
HEV	ELISA <sup>2</sup> VN PCR	~90 Unknown Unknown	~90 Unknown Unknown	Mast <i>et al.</i> (1998); Emerson <i>et al.</i> (2006)
HPAIV	Ag-immunoassay (unspecified) PCR VI AGID	<90  90–96 (Gold standard)	Unknown  Unknown (Gold standard)	Cattoli and Capua (2006); Woolcock and Cardona (2005); OIE (2002)
NDV	ELISA VN PCR	~90 Unknown Unknown	~90 Unknown Unknown	Mast <i>et al.</i> (1998); Emerson <i>et al.</i> (2006)
RVFV	Ab-ELISA PCR VI AGID	97.3 Unknown Unknown Unknown	97.4 Unknown Unknown Unknown	Paweska <i>et al.</i> (1995); OIE (2008)
VSV	Ab-ELISA VN Ag-CFT Ag-ELISA PCR VI	>98 >98 Up to 100 Up to ~100 ~90 Unknown	>98 >98 ~100 ~100 ~100 Unknown	Allende <i>et al.</i> (1992); Allende and Germano (1993); Gomes <i>et al.</i> (1989); Sepulveda <i>et al.</i> (2007)
<i>Non-zoonotic group</i>				
ASFV	Ab-ELISA Haemadsorbtion Ag and Ab immuno- fluorescence PCR Immunoblotting	~97.4 ~97–98 ~90  Unknown ~98	87.3–92.8 99.9 95  99.9 99	Sánchez-Vizcaino (1987, 2006); OIE (2008); Agüero <i>et al.</i> (2003, 2004)
BTV	Ab and Ag ELISA AGID VI VN PCR	Unknown Unknown Unknown Unknown Unknown	Unknown Less than ELISA Unknown Unknown Unknown	OIE (2002); IAH (2002); FAO (2008)
CSFV	Ab-ELISA VN Ag-ELISA Ab immuno- fluorescence PCR VI	97–99 Unknown Moderate Moderate to ~99  Unknown (Gold standard)	97–99 Unknown ~95 ~99  Unknown ~99	W. Loeffen <i>et al.</i> (personal commu- nication); Bouma <i>et al.</i> (2001); Blome <i>et al.</i> (2006); Hoffmann <i>et al.</i> (2005); OIE (2008); Risatti <i>et al.</i> (2005); Greiser-Wil- ke <i>et al.</i> (2006)
FMDV	Ab-ELISA VN Ab-indirect ELISA Ag-ELISA VI CFT PCR	~100 ~100 ~100 Up to 100 Unknown Up to >98 >95	~98 ~98 >99 ~100 Unknown ~100 ~100	Mackay <i>et al.</i> (2001); FAO/IAEA (1994, 1997); Bergmann <i>et al.</i> (2000); Alonso <i>et al.</i> (1992); Reid <i>et al.</i> (2002); King <i>et al.</i> (2006)



Virus	Test	Sensitivity <sup>1</sup> (%)	Specificity (%)	References
LSDV	Ab-ELISA VN AGID PCR VI EM (rapid)	Unknown Unknown Unknown Unknown Unknown	Unknown Unknown Unknown Unknown Unknown	Ireland and Binopal (1998); Heine <i>et al.</i> (1999); Carn <i>et al.</i> (1994)
PPRV	ELISA VN PCR	94.5 Unknown Unknown	99.4 Unknown Unknown	OIE (2008), Libeau <i>et al.</i> (1994), Couacy-Hymann <i>et al.</i> (2002), Libeau <i>et al.</i> (1995)
SandGPV	ELISA VN VI PCR AGID IFA EM (rapid)	Unknown Unknown Unknown Unknown Unknown Unknown	Unknown Unknown Unknown Unknown Unknown Unknown	OIE (2008); Heine <i>et al.</i> (1999); Ireland and Binopal (1998); Carn <i>et al.</i> (1994); Carn (1995); Chand <i>et al.</i> (1994); Kitching <i>et al.</i> (1986b); Kitching and Smale (1996); Davies and Atema (1978)
SVDV	Ab-ELISA VN Ag-ELISA VI PCR	100 Unknown Low High High	99.5 99.9 100 100 100	Brocchi <i>et al.</i> (1995); Chenard <i>et al.</i> (1998); Golding <i>et al.</i> (1976); Núñez <i>et al.</i> (1998); Fallacara <i>et al.</i> (2000); Reid <i>et al.</i> (2004); OIE (2008)

1. Diagnostic sensitivity and specificity data given where available

2. Abbreviations used: Ab, antibody; Ag, antigen; AGID, agar gel immunodiffusion test; CFT, complement fixation test; ELISA, enzyme-linked immunosorbant assay; EM, electron microscopy; PCR, polymerase chain reaction (detects virus/viral fragments); VI, virus isolation; VN, virus neutralisation.

It will be noted that quantitative values for sensitivity and specificity are lacking for many test and virus combinations. The result also requires biological interpretation. For example, antibody production may not always indicate exposure to field virus, or it may identify historical exposure, with no infectious virus remaining. Detection of the viral genome (for example in PCR) does not necessarily mean that the virus is still viable and infectious. However, there are a variety of tests for each virus that, if used as recommended and in appropriate sequences and combinations, are likely to give a high overall sensitivity to laboratory diagnosis when actually undertaken for an infected animal. With respect to surveillance testing, ability to detect depends upon many other factors, including prevalence of infection and sampling regime, and this should also be considered when interpreting results.

### 3.4.2 Prevention and control of diseases in animals

The efficacy of the prevention and control of disease will depend upon many of the same factors as does detection. These factors include knowledge, experience, resources available and the economics and value of the animals. The management system will directly affect the level of biosecurity possible, in particular whether the animals are kept indoors or outside. Whether vaccines are available, economic, efficacious under field use, and allowed, will affect prevention and control, as will the availability of effective disinfectants. Table 19 gives brief details of the vaccines available, and table 20 gives an indicative, rather than an exhaustive, list of the susceptibility of the viruses to disinfectants and other chemicals. For both vaccines and disinfectants, and for all viruses except HEV, further details can again be found on the OIE website (OIE 2008).

Table 19: Animal vaccines available, type, and an indication of efficacy where this information is available

Virus	Vaccine(s) and efficacy (where available)	References
<i>Zoonotic group</i>		
HEV	Under development.	M. Banks (personal communication)
HPAIV	Inactivated whole virus vaccines: effective but need multiple injections. Recombinant vectored (live) vaccines: pre-existing immunity to the vector can impair efficacy.	EFSA (2007a)
NDV	Inactivated vaccines and live vaccines (e.g. Hitchner B1; La Sota – protective response transient, multiple vaccines required).	Alexander (2001)
RVFV	Inactivated bovine strain, use in pregnant animals. Attenuated Smithburn neurotropic strain, 'effective' but abortigenic. No efficacy values published. Experimental: e.g. MP12 and R566 strains.	Barnard and Botha (1977); Barnard (1979)
VSV	Attenuated; used in the field. Inactivated; available commercially.	Panaftosa (2006); OIE (2008)
<i>Non-zoonotic group</i>		
ASFV	None	OIE (2002)
BTV	Attenuated vaccines used widely in Africa, US, Israel. Some use in Mediterranean basin. Usually serotype specific, but some multi-valents used in South Africa. In Europe, a move has recently been made to use inactivated vaccines.	OIE (2002); IAH (2002) Bhanuprakash <i>et al.</i> (2009)
CSFV	Attenuated (e.g. Chinese (C), Thiverval, and GPE-) strains widely used where endemic; 'highly' efficacious. Immunity often life-long, but efficacy in field and lab conditions may be very different. For wild boar, an oral formulation is available. Two subunit vaccines now licensed.	Beer <i>et al.</i> (2007); Biront <i>et al.</i> (1987); Blome <i>et al.</i> (2006); European Commission – Food Safety (2003); Greiser-Wilke and Moenig (2004); Van Oirschot (2003); Bouma <i>et al.</i> (1999, 2000); de Gante <i>et al.</i> (2003); de Smit <i>et al.</i> (2000)
FMDV	Inactivated: viral type/subtype varies as appropriate. Expected efficacy (clinical symptoms) >80%.	OIE (2008)
LSDV	Attenuated Neethling strain: antibodies found for 2–3 years. Recombinant vaccines: under development.	Ngichabe <i>et al.</i> (2002); Aspiden <i>et al.</i> (2002); Wallace <i>et al.</i> (2006); Plowright and Ferris (1959)
PPRV	Attenuated: PR 75-1. 'Strong' immunity.	Diallo <i>et al.</i> (1989); Martrenchar <i>et al.</i> (1997); OIE (2002)
SandGPV	Attenuated: several strains used, e.g. Romanian and RM-65 (sheep), Mysore and Gorgan (goats), 0240 Kenya (sheep and goats).	Kitching <i>et al.</i> (1986c); Kitching and Taylor (1985); Kitching (1983); Davies and Mbugwa (1985); Ramyar <i>et al.</i> (1974); Ramyar and Hessami (1970, 1967)
SVDV	None	OIE (2002)

Table 20: Disinfectants available and their efficacy (where information is available)

Virus	Disinfectant(s) and efficacy	References
<i>Zoonotic group</i>		
HEV	None used or tested.	M. Banks (personal communication)
HPAIV	Oxidizing agents, aldehydes, phenolic compounds, chlorine and derivatives. Efficacy said to be good for all.	De Benedictis <i>et al.</i> (2007)
NDV	Alkalis (e.g. sodium hydroxide), halogens (e.g. chlorine), phenolic compounds, glutaraldehyde; all 'effective'.	G. Kock (personal communication)
RVFV	Inactivated by lipid solvents, ionic and non-ionic detergents, pH <6.	Porterfield <i>et al.</i> (1975/76)
VSV	Inactivated by formalin 1%, sodium carbonate 4%, sodium hydroxide 2%, sodium or calcium hypochlorite 2–3%, hydrochloric acid 2%, citric acid 0.2%. Sensitive to ether and other organic solvents.	Panaftosa (2006); OIE (2002); AusVetPlan (2000)
<i>Non-zoonotic group</i>		
ASFV	Inactivated by sodium hydroxide, hypochlorites, ortho-phenylphenol 3%, all with 'very high efficacy'.	Stone and Hess (1973); OIE (2002)
BTV	Iodophores and phenolic compounds.	OIE (2002)
CSFV	Easily inactivated by a wide range of organic solvents or detergents and UV-radiation.	Moennig (1988); Edwards (2000)
FMDV	Labile to pH <6. Inactivated by UV, phenol 5%, sodium hydroxide 2%, sodium carbonate 4%, citric acid 0.2%. Resistant to iodophores, quaternary ammonium compounds, hypochlorite and phenol.	OIE (2002, 2008)
LSDV	Susceptible to ether and chloroform. Inactivated by sodium hydroxide 1%, formalin 2%, quaternary ammonium compounds 0.5%.	OIE (2002); T. Gerdes (personal communication)
PPRV	Most disinfectants 'effective', e.g. phenol, sodium hydroxide 2%.	
SandGPV	Inactivated by sodium hypochlorite 0.1–1%, hydrochloric acid 2%, phenol 2%, citric acid, aldehydes, alcohols, iodophors, detergents containing sodium dodecyl sulphate (SDS), ether, chloroform, formalin.	DPIE (1996); Murphy <i>et al.</i> (1995); Tantawi and Al-Falluji (1979); Dardiri (1978)
SVDV	Inactivated in the environment by sodium hydroxide, potassium hydroxide, formaldehyde, and on personnel in absence of organic matter, iodophors and citric acid.	Herniman <i>et al.</i> (1973)

A variety of vaccines are available for the majority of the viruses considered here, but few quantitative data are available for their efficacy in the field. Problems in their use often include the necessity for multiple vaccinations, strain variation in the virus and the subsequent inability to differentiate vaccinated animals from those who have received a field virus challenge (DIVA – differentiating infected from vaccinated animals). They are therefore only one aspect of virus control.

A variety of disinfectants are effective against many of these viruses. Alkalis, acids, organic solvents and UV irradiation are commonly described as effective, although again few quantitative data on efficacy are available. The point has been made by a number of disease experts that prior cleansing to remove organic matter is the most important decontamination step, as most disinfectants are inactivated by remaining organic matter. They do, however, represent a further important aspect of virus control and biosecurity, particularly where contamination is suspected or likely.

### ***3.5 Potential for contamination and survival of viruses in foods***

Food ingredients may be contaminated with highly contagious viruses of animals in a number of ways. The most obvious method is where they are derived from infected animals. In this case, if the tissues that constitute the ingredient are those in which the virus is normally found during infection, then those tissues will be contaminated; specific tissues likely to be contaminated will depend upon the specific viral tissue tropism. The second route is cross-contamination, where the tissues have come into contact with the virus during slaughter and processing, for example from secretions, excretions, aerosols, faeces, tissue scraps etc., from infected animals. Finally, indirect contamination due to environmental and water contamination, fomite transfer (including via plant personnel) and similar means may be responsible.

Given a specific original level of contamination, there are a number of factors that will affect the burden of infectious virus remaining on or in a food ingredient. Food ingredients derived from animals (including birds), and used in human, pet or other foods include meat, offal, blood, bone, other tissues, milk and eggs. One factor is the fragility of the virus under different environmental conditions, for example its susceptibility to temperature, pH, humidity etc. Another relevant factor is time; a virus cannot multiply except in living tissue and therefore, as soon as it is separated from its host, numbers will begin to decline, the specific rate depending upon those conditions. The range of specific processes through which the food ingredient passes before consumption, including chilling, freezing, smoking, drying, mixing, diluting, cooking etc., will all affect the viral burden. Dilution of the viral burden by washing or mixing is possible. Food may be presented at retail as fresh, raw chilled, raw frozen, preserved by drying, smoking, salting or similar, cooked-chilled, cooked-frozen, tinned, made into cheeses etc. Given the same contaminated starting ingredient, each final product will have a different likelihood of contamination and infectious viral burden. In general, appropriate heat treatment is likely to reduce any infectious burden present. This may be accomplished by a heat processing stage during manufacture, or by thorough cooking of a product shortly before consumption. As there is much complexity in producing food products, so there is much complexity in attempting to identify the products most at risk of contamination.

One other related issue is that of slaughterhouse and processing plant effluents. Where infected animals are present, effluents from lairage, stunning and slaughter areas, trimmed discarded tissues and any other effluents or production waste represent a potential source of viral escape, and thus need to be considered in food production.

Table 21 gives information on the potential for contamination and survival of known zoonotic viruses in food, and Table 22 gives the same information for non-zoonotic viruses.

Table 21: The potential for contamination and survival of the virus in food (including food intended for pets) for those viruses considered to be zoonotic

Property	Information	References
<i>Hepatitis E virus (HEV)</i>		
Tissue tropism	Pig liver; has also been detected in meat, offal, blood.	Williams <i>et al.</i> (2001)
Potential for contamination of food or during food production	Few data available, one study shows ~5% HEV will survive simulated cooking conditions of 1 hr at 60°C; 1.9% and 11% of retail pig liver in Japan and USA, respectively, contained detectable HEV. No direct information available on contamination of food preparation and processing environments with the exception of effluent from pig slaughterhouses in Spain.	Emerson <i>et al.</i> (2005); Pina <i>et al.</i> (2000); Yazaki <i>et al.</i> (2003); Feagins <i>et al.</i> (2007)
<i>Highly pathogenic avian influenza virus (HPAIV)</i>		
Tissue tropism	Muscle meat, blood, liver, bone, lung. Eggs: HPAIV on surface and in contents (for LPAIV, up to 57% may contain virus; no equivalent detail for HPAIV).	EFSA (2005); Toffan <i>et al.</i> (2007); Tumpey <i>et al.</i> (2003); Mase <i>et al.</i> (2005); Cappuccini <i>et al.</i> (1985)
Potential for contamination of food or during food production	Inactivation of H5N1 HPAIV in chicken meat after cooking assumed at internal temperature of 73.9°C, or 70°C for 5 s. One report that high hydrostatic pressure can inactivate HPAIV. Viable for long periods in tissues, faeces, water. Tigers, leopards and chickens infected after eating raw chicken infected with H5N1 HPAIV. Live virus detected in frozen duck lungs. Heat treatment (as required by EU after detection of infection) validated to kill all residual virus in eggs.	Keawcharoen <i>et al.</i> (2004); Swayne and Beck (2005); Thomas and Swayne (2007); Isbarn <i>et al.</i> (2007); Beato <i>et al.</i> (2007); OIE (2002, 2008)
<i>Newcastle disease virus (NDV)</i>		
Tissue tropism	Lentogenic strains: detected in respiratory tract only. Virulent strains: most organs and tissues including eggs.	Al-Garib <i>et al.</i> (2003); Alexander (2001); Lancaster (1966)
Potential for contamination of food or during food production	Survives for long periods at ambient temperature especially in faeces. Transmission to birds via ingested food; can survive freezing. Processing of eggs and poultry meat (3 min at 80°C) will kill the virus.	Alexander (2001); AusVetPlan (1996); OIE (2002)
<i>Rift Valley fever virus (RVFV)</i>		
Tissue tropism	Primarily hepatotropic with high titres present in blood. Low concentrations of virus in milk and body fluids.	Mundel and Gear (1951)
Potential for contamination of food or during food production	Ritual slaughter produces blood aerosol infection; abattoir workers have been found to be seropositive. Contamination of meat unlikely as virus inactivated by heat, drying and acid conditions. When meat is matured and chilled for 24 hr, acid pH in meat inactivates virus. Circumstantial evidence that unpasteurised milk is infectious.	Chambers and Swanepoel (1980)
<i>Vesicular stomatitis virus (VSV)</i>		
Tissue tropism	Epithelial tissue covering the vesicles, and vesicular fluid.	OIE (2002)
Potential for contamination of food or during food production	Survives for long periods at low temperatures. Virus not found in any part of the edible carcass of infected animals. It has been found in raw milk, but does not survive pasteurisation.	AusVetPlan (2000); OIE (2002)

Table 22: Information available on the potential for contamination and survival of the virus in food (including food intended for pets) for those viruses not considered to be zoonotic

Property	Information	References
<i>African swine fever virus (ASFV)</i>		
Tissue tropism	Blood, lymph nodes, bone marrow, fat, muscle.	Mebus <i>et al.</i> (1993)
Potential for contamination of food or during food production	Virus may persist for several weeks or months in blood (18 months), fresh (105 days) or frozen (1000 days) meat. In products prepared by curing, e.g. Parma ham, viral infectivity was not demonstrated at 300 days; in typical Spanish dry cured meat products e.g. Serrano hams, Iberian hams, the virus survives for 112 days to 140 days. As it is very resistant in blood, it can easily contaminate effluent.	McKercher <i>et al.</i> (1987); Mebus <i>et al.</i> (1993); Quintero <i>et al.</i> (1986)
<i>Bluetongue virus (BTV)</i>		
Tissue tropism	Lymph nodes, blood.	OIE (2002); IAH (2002)
Potential for contamination of food or during food production	Has survived for years in blood stored at 20°C. Animal carcasses and products (e.g. meat, milk, wool) are stated not to be a method of spread (sensitive to pH <6 or >8).	OIE (2002); IAH (2002)
<i>Classical swine fever virus (CSFV)</i>		
Tissue tropism	All tissues and body fluids (e.g. blood, urine, semen, saliva) may contain high titres.	
Potential for contamination of food or during food production	May remain infectious under favourable conditions, e.g. survives in frozen pork >4 years and >85 days in chilled fresh pork. Salt-curing and smoking of meats has no direct affect; critical factors are storage time and temperature e.g. survival in smoked and cured products <17–188 days, Parma ham <313 days, Serrano and Iberian hams <140–252 days. Pasteurisation or cooking readily inactivates virus, e.g. meat for 30 min at 65°C or 1 min at 71°C; blood for 60 min at 66°C, 45 min at 68°C or 30 min at 69°C. Relatively easily inactivated in environment. Survival in abattoir waste at 20°C is <4 days. Cleaning and disinfection should be sufficient to inactivate. Pigs' milk unlikely to be used.	Edwards (2000); Helwig and Keast (1966)
<i>Foot and mouth disease virus (FMDV)</i>		
Tissue tropism	Milk may contain virus at low titre up to 4 days before clinical signs. During acute phase, blood may contain high titres; epithelial tissues and vesicular fluids likely to contain high titres. Muscle not a target tissue, but contamination likely due to viraemia.	OIE (2008).
Potential for contamination of food or during food production	Meat matured between 2–7°C for 24–76 hr (reaching pH <6) then deboned is unlikely to contain infectious virus. Can persist in environment for up to 1 month, depending upon temperature and pH. Variable titre can remain in milk, but dilution and processing will reduce this considerably. Milk from pigs not commonly used in human food products.	OIE (2007b); OIE (2002)

Property	Information	References
<i>Lumpy skin disease virus (LSDV)</i>		
Tissue tropism	Both epitheliotropic and systemic (mainly respiratory and reproductive organs). Milk may contain virus.	Weiss (1968)
Potential for contamination of food or during food production	Heating at 56°C for 30 min or 60°C for 10 min inactivates virus in milk for suckling calves. As an extremely debilitating disease, meat is unlikely to be used as food, either eaten fresh or processed, thus food contamination is unlikely to occur. However, dried virus in the environment can withstand 100°C for 10 min, and environmental contamination for several months in scab material is possible.	OIE (2002), T. Gerdes (personal communication)
<i>Peste des petits ruminants virus (PPRV)</i>		
Tissue tropism	Respiratory system (nasal mucosa, epiglottis, trachea, lung). Lymphatic system (thymus, lymph nodes, spleen, lymphatic fluid).	Bundza <i>et al.</i> (1988); Brown <i>et al.</i> (1991); Kumar <i>et al.</i> (2004); Galbraith <i>et al.</i> (2002)
Potential for contamination of food or during food production	Survives for long periods in chilled and frozen tissues; destroyed at 60°C for 60 min.	OIE (2002)
<i>Sheep pox and goat pox virus (SandGPV)</i>		
Tissue tropism	Skin papules and superficial lymph nodes. Papules also found on the tongue, palate, trachea, oesophagus, and in digestive tract. Virus in blood and spleen, possibly also in kidney, liver, lung and testicles. Possibly in meat and milk.	OIE (2002); Rao and Bandyopadhyay (2000); Munz and Dumbell (1994); Jubb <i>et al.</i> (1993); Davies (1991); Isloor <i>et al.</i> (1991); Saha <i>et al.</i> (1991); Kirk (1981); Bennet <i>et al.</i> (1944)
Potential for contamination of food or during food production	Stable in freezing and thawing. Loss of infectivity at pH 3 for 2 hr. Less susceptible to alkali. Heat sensitivity varies with strain, some not detectable after treatment at 55°C for 1 hr. Infectivity in meat and milk considered to be a low probability, but the low pH of cheese cannot be relied upon for complete inactivation. Virus remains viable in wool for 2 months and in premises for up to 6 months; skins, hides and wool may contain infectious virus and are thus associated with higher risks.	Rao and Bandyopadhyay (2000); AQIS (1999); Geering <i>et al.</i> (1995); Datta and Solman (1991); Sharma <i>et al.</i> (1986); Dardiri (1978); Koylu and Nitzschke (1968); AFFA (2003); MacDiarmid and Thomson (1997); Munz and Dumbell (1994); Kirk (1981); OIE (2002)
<i>Swine vesicular disease virus (SVDV)</i>		
Tissue tropism	All tissues, secretions and excretions. Virus has tropism for epithelial tissue (skin and mucosa of digestive tract), lymphoid tissue, CNS.	Lai <i>et al.</i> (1979); Mann (1980); Dekker <i>et al.</i> (1995); Lenghaus <i>et al.</i> (1976); OIE (2002); Lin and Kitching (2000)
Potential for contamination of food or during food production	Resistant to fermentation and smoking; may remain in hams for 180 days, dried sausages for >1 year and processed intestinal casings for >2 years. Inactivated by 56°C for 1 hr. The potential for contamination is very high since the virus is extremely resistant in the environment and stable in the range pH 2–12.	OIE (2002); McKercher <i>et al.</i> (1974, 1980, 1985)

Information on tissue tropism, the potential for food contamination and the resistance of the virus to environmental effects is used when estimating the risks from particular animal products, when a susceptible host species is exposed and thus potentially infected. There is considerable variety in the tropism and resistance of the different viruses; therefore, each must be considered separately in this context. The possible exception is the effect of heat; pasteurisation or thorough cooking is generally considered to be effective for decontamination of food products.

### ***3.6 Detection, prevention and control in foods, the food production environment and waste products***

For all the viruses considered, the possible methods of detection in foods (for both human and animal use), and environmental and effluent contamination, are essentially the same as those used to detect infection in animals, modified as appropriate for the specific sample and food product matrix. The use of meat juice ELISAs would be one example of a potential modification. Virus isolation and PCR methods can also be used.

Prevention of viral contamination of the food product, the food production environment and waste products depends primarily on ensuring the animals from which food is derived are uninfected. Recommendations and guidelines for the regulation of trade in animals and products of animal origin are given through the OIE Terrestrial Animal Health Code (OIE, 2007b) and the Codex Alimentarius Commission (CAC) (CAC, 2008), and are designed primarily to ensure that trade does not include infected animals or contaminated animal products. For imported animals and their products, entry into the EU is highly regulated and includes, for example, certification of disease freedom for live animals, border inspection and/or documentation checks for both live animals and product consignments at Border Inspection Posts (BIPs), and specific pre-treatment requirements for particular products. These requirements vary with region of origin, product and the species from which it is obtained. For example, the maturation and de-boning of meat from certain countries may be specified to reduce the probability of contamination with viable FMDV (OIE, 2007b) or heat treatment required to ensure the destruction of the PPRV for certain products (OIE, 2007b). Full details on regulations and directives can be found from the EU website (EC, 2008). Thus, the probability of legally imported animals and their products containing an infective virus on entry to the EU can, for most viruses, generally be discounted (also see section 4.2).

For local sourcing of food-producing animals for meat, milk and eggs, the major preventive measure against viral contamination of foods, the food production environment and waste products is again ensuring that the animals from which the food originates are not infected, and the effectiveness depends upon the sensitivity of detection of the particular virus, including clinical detection. This varies with the particular virus (see Sect. 3.4.1). With regard to eggs, birds with both NDV and HPAIV infection are likely both to produce abnormal eggs and, especially for clinical HPAI, to cease laying, reducing the likelihood that such eggs will enter the food chain.

Control measures at the slaughterhouse include pre- and post-mortem examination, and general hygiene measures to prevent cross-contamination, including disinfection (see Sect 3.4.2 for efficacy of disinfectants against specific viruses). For poultry, complete and clean evisceration of the carcass will limit contamination with LPAIV. The maturation of meat (thus reducing pH to levels at which many viruses are susceptible), the pasteurisation of milk and the production of cheese from pasteurised milk are all measures that will reduce the probability of viral contamination of the relevant foods. These measures are all considered effective against the range of viruses, and there are generally no detection regimes or control measures in place during food production in the EU against any single specific virus considered in this document.



## 4. CONCLUSIONS

### ***4.1 Implications for human (and pet) health from contaminated foods (including fresh and raw foods), and their production processes***

#### ***4.1.1 Implications for human health from foods contaminated with zoonotic viruses and their production processes***

Few of the viruses reviewed in this document are considered to be zoonotic.

Vesicular stomatitis virus is considered only to be a minor zoonotic, with acute flu-like symptoms in humans and no mortality. Its transmission routes are poorly understood, but reports of accidental infection of workers while performing post-mortems suggest a theoretical possibility of unprotected slaughterhouse workers becoming infected. There is no evidence to indicate transmission via food; the virus is not found in any part of the edible carcass of infected animals. It has been found in raw milk, but does not survive pasteurisation. Currently, vesicular stomatitis virus is restricted to the Americas, and thus any animals or food products likely to be infected would be subject to EU import safeguards. Within the EU, therefore, there are currently considered to be no implications for human health from food products of animal origin.

Rift Valley fever is a vector- (mosquito-) borne disease with a climatic dependency, requiring rainfall and floods. The virus has historically been present in parts of Africa and more recently, following flooding, spread into the Middle East. Currently it is not present in the EU, therefore any animals or food products likely to be infected would be subject to EU import safeguards. However, climate change resulting in extremes of weather, coupled with movement of wind-borne vectors could lead to an expansion of its present range, even into parts of Europe. The virus also has, within endemic areas, cycles of between 5 and 35 years between epidemics, between which cryptic cycling occurs and detection is difficult. If entry to the EU occurred, it could similarly remain undetected for a period after entry, although due to the susceptibility of previously unexposed European stock, this undetected period is considered likely to be short. In addition, human infection via aerosols during ritual slaughter, or the handling of fresh carcasses during food preparation is possible, although matured meat below pH 6 is considered to be non-infectious. Consumption of raw milk has also been implicated as a transmission route. Therefore, currently, any implications for human health are unlikely from food products produced in the EU. However, if the virus does spread into the EU, there would be implications for local slaughterhouse workers, especially for those undertaking ritual slaughter, and for products containing unpasteurised milk.

Highly pathogenic avian influenza virus, if present in poultry, could be a source of infection for those coming into close contact with infected birds and newly slaughtered carcasses; for example abattoir workers. The finding of live AI virus in frozen duck lung suggests that AI viruses are not necessarily inactivated by freezing, and they may also survive in fluids for some time if conditions are right. Therefore, raw poultry products and their environment may be contaminated and can be a source of infection. Eggs may also be contaminated with, or contain, virus. Thus, in the event of undetected HPAIV infection of poultry (for example, at an early stage in the outbreak), the risks to those working directly with the birds or their eggs could be high, particularly for slaughterhouse workers. However, thorough cooking will inactivate both LPAI and HPAI viruses, and properly cooked poultry products (including eggs) are considered not to remain infectious.

Newcastle disease virus contaminated food is not considered to be a risk for mammals, including humans.

Hepatitis E virus is transmitted by ingestion of infected meat, and found globally in pigs. The products considered most likely to be implicated in infection are raw or undercooked pig meat or liver. Shellfish have also been implicated in outbreaks. Little information is available on the survival of HEV in the environment or in food production processes; one study shows 95% virus inactivation after cooking at 60°C for 1 hr. Although most disease has historically been acquired in regions with human endemicity, this picture is changing, although the exact role of food products is unknown. Nevertheless, the possibility of contamination and transmission must be seriously considered in slaughterhouse and food production processes.

#### ***4.1.2 Implications for human health from foods contaminated with non-zoonotic viruses***

For any virus to which humans are truly not susceptible, there are no human health implications from foods contaminated with these viruses. However, it is likely that people would in general prefer to eat foods produced from healthy animals and, on occasion, there has also been media concern over such infections (or vaccinations) in animals, without scientific basis. This can lead to a perception of lack of safety, which may also affect consumption of a particular product.

In addition, sometimes the host range or pathogenicity of a particular virus can change, due for example to a mutation, and this should always be borne in mind.

Viruses reviewed in this document and considered not to be zoonotic are African swine fever virus, classical swine fever virus, foot and mouth disease virus, lumpy skin disease virus, bluetongue virus, peste des petits ruminants virus, sheep pox and goat pox virus, and swine vesicular disease virus.

#### ***4.1.3 Implications for pets, pet foods and other products eaten by pets, and associated production processes, if contaminated with the viruses considered in this report***

Any species of animal, including livestock, may theoretically (and sometimes in fact) be kept as a pet, including those susceptible to the viruses described in this report. Of note in this regard are backyard pigs and mini-pigs, which may be kept as pets more frequently than other livestock species. Pigs are omnivores and susceptible to a number of these viruses and, therefore, could be infected by food products or abattoir waste containing infectious doses of relevant viruses.

The main focus of this section is those species commonly kept as pets or companion animals in the EU, namely dogs, cats, horses and birds. Dogs and cats are both carnivorous species for which pet foods are routinely manufactured using livestock carcasses. In addition, fresh meat and milk is sometimes bought for, or fed to, these pet species. However, neither these species, nor horses or birds, are considered to be susceptible to the majority of the viruses included in this report. Possible exceptions are now indicated.

Antibodies to HEV have been recorded in cats, indicating possible exposure. If the source of this exposure is food-associated, then the products considered most likely are raw or undercooked pig meat or liver (as the virus is found ubiquitously in pigs) and, possibly, shellfish. As contaminated water is also considered to be a possible transmission route, abattoir effluent may also be a source of exposure. Little information is available on the survival of HEV in the environment or in food production processes. Although there are no recorded cases of disease in animals, and no virus yet detected in cats, these possibilities cannot be dismissed.

RVFV has been shown experimentally to infect puppies and kittens via aerosols, but not by ingestion. However, in the EU it would not be normal to allow pets in an abattoir, exposed to possible aerosols produced by the slaughter process, even if the virus were to enter the EU.

Whilst VSV does affect horses, food produced for horses would not generally contain meat products, and currently the virus is not found in the EU. Similarly, although HPAIV and NDV could affect pet birds, food produced specifically for pet birds is usually unlikely to contain meat products. There are therefore no particular implications for commercially produced food for horses or, in the majority of cases, pet birds in the EU.

NDV however, if present undetected in avian material (including frozen carcasses), has been shown to be capable of infecting other birds to which it has been fed. In addition, if sub-clinical or low virulence strains are present in source material, they may be undetected. Thus, there is a theoretical route by which pet birds might become infected if their food contains, or is contaminated by, such material. In addition, contamination of bird food production and storage facilities by faeces from wild birds might also lead to exposure and infection. NDV-contaminated bird food, that has not subsequently been heat-treated, would be a major risk if given to susceptible poultry or pet birds.

HPAI viruses have been shown to infect felines after ingestion. Therefore, in the event of undetected HPAIV in the source material, pet foods made from raw contaminated poultry products (possibly even after freezing) could potentially infect cats. As for human food, thorough cooking would inactivate the virus.

To summarise, the only viruses considered here with possible implications for pet food manufactured in the EU are HEV and, in the event of an undetected infection or contamination, NDV and HPAIV. However, just as for human food products, pet owners might be expected to prefer to feed their pets with foods produced from healthy livestock.

## ***4.2 Implications for trade in food products of animal origin (including fresh and raw products)***

Trade in food produced from animals may be undertaken for a variety of reasons: imported food (including pet food) may be cheaper; it may be impossible to find certain foods produced locally (especially those associated with particular cultures and cuisines); certain countries may have particularly high reputations for quality; and so on. As travel increases, people become exposed to a variety of new foods that they want to experience at home. Thus, trade in these products tends to increase. Movement of meat and animal products around the world may be undertaken legally, or illegally.

For legal movement, as indicated in Sect. 3.6, the majority of the viruses described here are subject to strict controls, both of livestock and their products. Known presence of the infection in the country of origin will result in appropriate movement restrictions, the requirement for certification of freedom before trade can recommence and/or, for food produced from such animals, specific viral reduction processes such as maturation, de-boning or pasteurisation. ASFV, BTV, CSFV, FMDV, HPAIV, NDV, PPRV, RVFV, SVDV and VSV are the subject of such controls; however, for LSDV, import controls refer only to semen, not to food products, and no specific trading requirements exist for HEV. If a specific product is in short supply because a major source country has an outbreak that disrupts normal trading patterns, the lack of that product may indirectly affect food or pet-food supply and production. There are likely also to be economic implications for the infected country.

Illegal movement of food and food products obviously avoids all controls and safeguards, and, if there is illegal trade in infected animals or contaminated animal products, runs the risk of bringing infection to new regions of the world. In addition, contaminated food waste from ships and aeroplanes has been implicated in the spread of animal infection.

When considering the implications for human and pet health with regard to traded food products of animal origin, if a contaminated product is successfully imported, then the risks are similar to those produced from locally reared livestock. Only contamination with those viruses that are zoonotic, or can infect domestic pets, will have direct implications. Only five of the viruses considered fall into this class: VSV, RVFV, HPAIV, NDV and HEV. All these viruses except hepatitis E virus are subject to legal import requirements making the probability of contamination of legally imported products extremely low. In addition, maturation of the meat with associated pH changes causes the viral titre in even fresh meat to decrease rapidly, and this would be the case for both legally and illegally imported products. Thus for VSV, and RVFV there are considered to be no zoonotic or pet-food related implications for meat products or for pasteurised milk. However, imported chicken and duck meats have tested positive for H9N2 (LPAIV) and H5N1 (HPAIV) respectively (Mase et al. 2007; Tumpey et al. 2003) and, historically, trade and import of poultry meat has been implicated in the spread of Newcastle disease. Trade and, in particular, illegal movements of poultry products may therefore have the potential to disseminate these viruses in areas previously free of avian influenza and Newcastle disease. For hepatitis E virus, the disease is occult and virtually apathogenic in pigs, and no import/export certification is required. Given that the virus has been found globally and is present in fresh animal products at retail, the possibility of contamination of traded pig and possibly shellfish products cannot be discounted.

### ***4.3 Areas for research and recommendations for research***

Two major areas of recommended research emerged from the suggestions of the virus experts involved in this report.

The first concerns research applied directly to the virus itself, with the aim of developing improved diagnostics, especially in the field, plus advancements in vaccine technology and therapeutic tools. Further research into one or more of these aspects is specifically recommended for ASFV, CSFV, FMDV, HPAIV, NDV, PPRV, RVFV and VSV. One particular aspect of vaccine research involves the problem of differentiating infected from vaccinated animals (DIVA), and the development of vaccines (and associated diagnostic tests) that allow it. This requirement was noted particularly for CSFV and is also important in HPAIV. New approaches for such DIVA vaccines include DNA, vector vaccines and chimaeric viruses. For RVFV, research to enable improved availability of human vaccines, as well as for non-abortigenic vaccines for livestock has been recommended.

The second major area of recommended research involves epidemiological research, research into surveillance methodology and the utilisation of risk assessment methodology. Work on one or more of these aspects is specifically recommended for ASFV, CSFV, FMDV, HEV, PPRV, SandGPV and VSV. For HEV the basic epidemiology is still poorly understood, and studies are recommended on sources of infection, transmission routes, prevalence (including prevalence in retail pig products), environmental contamination potential (including effluent etc.) and differences in susceptibility amongst and within populations. For SandGPV, further epidemiological research into potential human infection and possible transmission routes is recommended as well as studies on semen contamination and viral activity when diluted in the environment. Although the more basic epidemiology may be known for other viruses, significant epidemiological knowledge gaps are still present. For CSFV, for example, eradication strategies that work in an industrialised setting do not necessarily work in rural settings with different management

systems; therefore, research into better control systems is necessary. Improved surveillance of foot and mouth disease in endemic regions is recommended, to allow early identification and the following of the evolution of new variants. This would also allow the checking of the efficacy of existing diagnostics and vaccines. Research into measuring the effectiveness of surveillance and control is also suggested specifically for PPRV. Research into the specific competencies of different vector species would be helpful in predicting the spread of bluetongue. The data required to enable meaningful risk assessments of import risk and food infectivity, are frequently not being collected or, in some cases, even identified. It is recommended that work to identify and collect such data be undertaken. In particular, survival of viruses in a variety of environmental and food matrix conditions needs researching. Currently, many such risk assessments must employ a large amount of expert opinion to fill data gaps.

In addition, for the avian influenza viruses, research into resistance and persistence in different environments is specifically recommended, as well as research into their infectivity and pathogenesis in different host species. This would include research into the factors that predispose and/or cause the shift from low to high pathogenicity, and this point would also apply to Newcastle disease.

#### ***4.4 Considerations for governments, animal producers, food manufacturers (including pet food and water providers) and consumers***

##### ***4.4.1 Major points governments should consider with respect to these viruses***

For the majority of these viruses, one key requirement mentioned by the virus experts was the necessity to ensure good surveillance, diagnosis, control, contingency and (where feasible) eradication policies, both in the countries where the particular virus is endemic and in countries to which it might spread. These should be based on the specific epidemiological and biological characteristics of the virus being considered. Implicit in this is the necessity to undertake relevant research, as indicated in Sect. 4.3, and to raise awareness and knowledge amongst farmers and veterinarians to aid rapid diagnosis. Underpinning these activities requires the availability of funding, a suitable veterinary infrastructure and, ideally, co-operation between industry, government and international bodies. In fact, international initiatives have specifically been suggested as the way forward for a number of viruses, and the point has often been made that the most effective management and control policies, for the reduction of opportunities for persistence and spread, would be policies that are agreed and are consistent between national regulatory authorities.

For vector-associated viruses, government and industrial initiatives involving civil engineering in vector-breeding areas could be considered. For example, to best control RVFV, the impact assessment of major irrigation schemes and dam constructions should consider the effect on this virus. In Egypt and Mauritania, epidemics of Rift Valley fever followed construction of the Aswan dam on the Nile, and of the Diama dam on the Senegal river.

With respect to import and export issues, import risk assessments to establish the probable level of risk for each animal product (including both food and non-food products, e.g. semen, hides etc.) can aid government decision-making. Similarly, risk assessments, including modelling of spread within a country or region, can assist in decisions on vaccination policy in countries currently free without vaccination, for specific viruses, as a part of contingency planning. Once policy decisions are made, appropriate controls, including border controls where relevant, need to be in place. In addition, it is necessary to ensure public awareness of the risks of illegal importation.

Specifically for HEV, as well as the basic epidemiological research indicated in Sect. 4.3, testing all non-A, non-B acute hepatitis cases within the EU for hepatitis E, regardless of whether or not they have recently undertaken foreign travel, would assist in elucidating possible infection sources, as would surveys to establish the level of HEV contamination in retail pig products.

#### *4.4.2 Major points animal producers should consider with respect to these viruses*

Once again appropriate surveillance, identification, diagnosis and control measures are the major issue for all animal viruses considered here. Producers should ensure staff utilise hygiene and biosecurity measures appropriate to the management system in use, and are aware of the major signs of ill-health generally, and these viruses in particular, in the animal species for which they are responsible. Of note in this respect are HPAIV/LPAIV, as they both have low pathogenic variants likely to be much harder to detect clinically, although an additional sign may be egg production drop or abnormal eggs. If any of these viruses are suspected, the veterinarian should be contacted immediately and their instructions followed.

Where a routine vaccination policy is in use, it must be kept up to date. For LSDV and RVFV, this is particularly important in endemic areas when the weather is, or is forecast to be, abnormally wet.

A number of these viruses could be transmitted to pigs via swill, if improperly prepared; NDV could be transmitted to poultry in a similar way. This has led to the banning of the feeding of swill in many countries, but where it is still allowed and used, it is essential to ensure it is thoroughly cooked. To reduce risks even further, waste from international ships and aeroplanes should never be used in swill.

Within the EU, in areas where CSFV is endemic in wild boar, it is advised either that hunters do not keep pigs or, if they do, that they avoid entering the pigs accommodation for at least three days after having been in contact with wild boar.

As there are no clinical signs of HEV in pigs, there are no specific considerations for animal producers relevant to this virus at present; however, further research may alter this advice.

#### *4.4.3 Major points food producers (including pet food producers and water providers) should consider with respect to these viruses*

It is recommended that food producers ensure they source their products from known suppliers with appropriate quality standards. It might be expected that, in general and whether or not a virus is a known zoonotic, consumers would prefer their food to be sourced from healthy animals. It should also be apparent that to reduce risks of exotic diseases to the minimum, imported livestock and raw materials should all be obtained from legally imported sources. Nevertheless, awareness of the international situation, including new outbreaks and patterns of spread, would be an additional safeguard. In addition, proper veterinary inspection in the lairage pre-slaughter will reduce further the likelihood of any infected animal being admitted for routine slaughter, with its additional risks of aerosol and effluent contamination.

As most of the viruses considered are not zoonotic, there are no specific considerations for them with regard to human health, either for the production workers or for the eventual consumers. Nevertheless, good practice requires adherence to the general principles of food production hygiene. In particular, to avoid the spread of an animal virus to other animals, appropriate disinfection and/or disposal of waste (including, for example, feathers) and effluent from production premises (e.g. slaughterhouses) must be undertaken.

With regard to those viruses that are known to be zoonotic or that could affect domestic pets including birds (VSV, HPAIV, RVFV, NDV, HEV), careful sourcing and (for zoonoses) proper protection of slaughterhouse workers, particularly from aerosols and unprotected handling of newly killed carcasses, is the major consideration. If an infected animal has managed to get undetected into the slaughter chain, post-mortem pH changes rapidly reduce the infectivity of HPAI and RVF viruses. However, one particular virus should be mentioned and that is HEV. Until more is known about the distribution and survival of HEV in food, considerations for food manufacturers are difficult to specify.

It has already been noted that unpasteurised milk products maybe a transmission route for VSV and RVFV to humans and, for RVFV, to pets. However, there is a demand in some areas for such products. Obviously, these present a higher risk in areas where such viruses are endemic and it may be considered unwise to fail to pasteurise such milk. In other areas, labelling unpasteurised milk and cheese is the usual way to ensure customers can make their own choice. Indeed, informative labelling including source, contents and instructions for storage and preparation is now a common customer requirement, is good practice for all packaged or processed products of animal origin, and, in some cases, is a legal requirement.

In the EU there is a requirement to destroy, or heat-treat, all eggs from flocks diagnosed with HPAI, although there is a brief window in which eggs produced during the incubation period of the disease might enter the food chain. However, it is considered that most commercially produced eggs can be traced back for destruction if necessary. With HPAI, rapid egg drop and abnormal eggs are typical signs, thus the probability of eggs remaining in the food chain is considered lower than for LPAI.

With respect to contamination of pet foods, regardless of the products from which they are manufactured, it is vital to prevent access by wild birds, rodents etc. to feed storage rooms. In particular, wild birds accessing feeds intended for pet birds may lead to contamination with NDV or AIV.

#### *4.4.4 Major points consumers should consider with respect to these viruses*

Basic kitchen hygiene and thorough cooking of meat, meat products, eggs and egg products are generally considered to be the best ways of reducing the risk of exposure to, and subsequent infection by, any zoonotic virus from such products; the same principles apply to reducing the risks from pet foods. Adherence to these principles is best achieved by ensuring consumer awareness. Whilst, in general, campaigns in this area feature the risks from pathogens such as Salmonella and Campylobacter, this advice applies equally well to, for example, HEV.

With regard to the consumption of unpasteurised milk, or milk products, this is considered unlikely to be a significant risk within the EU, at present, for the exotic viruses of VSV and RVF. However, consumers living in, or visiting, countries where these viruses are present should be aware of the additional risks. Similar risks may also arise with food items imported illegally into the EU and consumers should consider this if they are offered such products. Raw eggs are traditionally used in some recipes, mayonnaise for example, and the risks from this practice need to be considered in the light of any prevailing HPAI disease.

To reduce transmission risks, consumers should avoid allowing pet birds (including backyard chickens) access to any uncooked poultry products and scraps. In addition, consumers could be made more aware of the, perhaps remote but nevertheless possible, risk of infecting farmed livestock with exotic diseases by leaving such things as sandwich scraps and kitchen waste in areas where they can be accessed by farm livestock, pigs in particular. Fly-tipping appears to be an increasing activity in some places, even though often illegal.

## 5. FOOTNOTE ON H1N1 INFLUENZA A VIRUS

**G**eneral information on the genus *Influenzavirus A*, its subtypes (of which H1N1 is just one), and its host range, has already been given in the section on highly pathogenic avian influenza viruses. In April 2009, a previously unrecognised strain of an H1N1 influenza A virus was reported, initially in Mexico. This was identified in people, but due to similarities with a subtype found in pigs, was initially called 'swine flu'. On 11 June 2009, the World Health Organisation (WHO) designated it a pandemic. Due to the relationship between the previously recognised swine flu viruses in swine and this novel strain found in humans it was decided to include a brief overview of the H1N1 influenza A virus in swine and humans.

Table 23: *Influenza A virus in pigs*

Property	Information	References
The virus	Subtypes H1N1, H1N2, H3N3 are all found in pigs, but there is significant genetic and antigenic variability within each subtype.	Irvine and Brown (2009)
Transmission routes in pigs	Usually by aerosols produced by coughing and sneezing. Also by direct and indirect contact (i.e. via fomites) and asymptomatic infected pigs.	Irvine and Brown (2009)
The disease in pigs	Signs range from asymptomatic through to severe acute respiratory illness, usually with low mortality. Chronic respiratory disease is also possible.	Irvine and Brown (2009)
Current geographical distribution	Endemic in many parts of the world, often with co-circulating subtypes. Within-subtype variability is frequently geographically associated. In particular, subtypes of H1N1 have historically had distinct North American and Eurasian lineages.	Irvine and Brown (2009)

Table 24: *The novel H1N1 influenza A virus found in the 2009 human pandemic*

Property	Information	References
The virus	The novel H1N1 subtype is made up of genetic elements of human, avian and swine flu H1N1 viruses. The swine flu elements are from those viruses typically found in Asia and Europe. The place of origin of the virus is unknown.	New Scientist, 2009; WHO, 2009a
Transmission routes in humans	Person-to-person in aerosols from coughing or sneezing, and contaminated hands or surfaces. As this is a new virus most people have little immunity thus it could cause more infections than with seasonal flu. There are no known instances of people being infected by exposure to pigs or other animals (although the reverse has occurred).	WHO, 2009a; Irvine and Brown (2009)
The disease in humans	Variable, including fever, cough, headache, muscle and joint pain, sore throat and runny nose and sometimes vomiting and diarrhoea. Severity ranges from very mild symptoms in the majority of people, to severe illnesses and death. More than half of hospitalised people had underlying health conditions.	WHO, 2009a
Current geographical distribution	On 11 June 2009, the World Health Organisation (WHO) designated this virus as a pandemic. As at 26 June 2009, there were 59,814 laboratory confirmed cases and 263 deaths, worldwide.	WHO, 2009b



### *The safety of pork and pork products*

On 30 April 2009, a Joint FAO/WHO/OIE Statement on influenza A (H1N1) was issued on the safety of pork. It states (FAO/WHO/OIE, 2009):

'In the ongoing spread of influenza A (H1N1), concerns about the possibility of this virus being found in pigs and the safety of pork and pork products have been raised. Influenza viruses are not known to be transmissible to people through eating processed pork or other food products derived from pigs.

Heat treatments commonly used in cooking meat (e.g. 70°C/160°F core temperature) will readily inactivate any viruses potentially present in raw meat products. Pork and pork products, handled in accordance with good hygienic practices recommended by the WHO, Codex Alimentarius Commission and the OIE, will not be a source of infection. Authorities and consumers should ensure that meat from sick pigs or pigs found dead are not processed or used for human consumption under any circumstances.'

This statement has more recently been reissued, and at 26 June 2009 remains the situation and advice.

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## 7. GLOSSARY

Ab	Antibody
Ag	Antigen
AGID	Agar gel immunodiffusion test
ASF	African swine fever
ASFV	African swine fever virus
BT	Bluetongue
BTV	Bluetongue virus
CFT	Complement fixation test
CSF	Classical swine fever
CSFV	Classical swine fever virus
ELISA	Enzyme-linked immunosorbant assay
FMD	Foot and mouth disease
FMDV	Foot and mouth disease virus
FVO	Food and Veterinary Office
HE	Hepatitis E
HEV	Hepatitis E virus
HPAI	Highly pathogenic avian influenza
HPAIV	Highly pathogenic avian influenza virus
LPAI	Low pathogenic avian influenza
LPAIV	Low pathogenic avian influenza virus
LSD	Lumpy skin disease
LSDV	Lumpy skin disease virus
ND	Newcastle disease
NDV	Newcastle disease virus
OIE	World Organisation for Animal Health
PCR	Polymerase chain reaction
PPR	Peste des petits ruminants
PPRV	Peste des petits ruminants virus
RVF	Rift Valley fever
RVFV	Rift Valley fever virus
S&GP	Sheep pox and goat pox
S&GPV	Sheep pox and goat pox virus
SVD	Swine vesicular disease
SVDV	Swine vesicular disease virus
VI	Virus isolation
VN	Virus neutralisation
VS	Vesicular stomatitis
VSV	Vesicular stomatitis virus

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