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Epidemiology, Diversity and Classifications of Parvoviruses

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Abstract

The development and application of more sensitive and new molecular approaches in the epidemiological research of viral infections of economic significance to the swine industry, detection of infectious viral agents has increased globally. The observation holds true for the member of the Parvovirinae family's subfamily Parvovirinae that infects pigs, since the use of cutting-edge molecular techniques like metagenomics has led to the discovery of numerous other new members of the family.

Keywords: Parvovirinae; Metagenomics; Zoonotic; Brevidensovirus

Introduction

Surprisingly, the list keeps growing, and some of the items on it may spread zoonotic diseases. At least ten novel swine-infecting viruses have been introduced to the subfamily in the past ten years, and ongoing research is being done to identify their occurrence and prevalence of the old and new swine parvoviruses in the herds of countries that raise pigs. The topic, however, is actually about the African continent, where there is now a lack of information regarding surveillance programmes for the viruses among swine herds in the region's pig-producing nations. For the implementation of efficient control and prevention of its spread, timely detection and identification of the viral pathogens are absolutely essential. So, in addition to providing current highlights on the reported instances of the viral agents in the African sub-region, this review gives a succinct summary of the epidemiology of novel swine parvoviruses worldwide.

Diversity

The Latin word "parvum," which is translated as "little," is the source of the prefix "parvo" in parvoviruses. Therefore, parvoviruses are a class of relatively small viruses that are housed in non-enveloped, icosahedral capsids and have linear, single-stranded DNA (ssDNA) genomes ranging in size from 4 to 6.3 kilobases (kb). They are common infectious agents of many hosts, including vertebrates, higher mammals, including humans, as well as non-vertebrate arachnids. According to Kailasan et al., their ancestors appear to have first appeared millions of years ago and have subsequently spread around the world [1]. Despite sharing comparable genomic characteristics and appearing to have arisen from the same ancestor, parvoviruses typically show very low relatedness at the nucleotide or protein level, illustrating their vastly divergent evolutionary history. Their diversity appears to have an impact on the clinical outcomes they have on their hosts, which can range from non-pathogenic infections to the symptoms of extremely fatal diseases. Densovirinae and Parvovirinae are the two subfamilies of the family Parvoviridae, which includes parvoviruses. Depending on the types of hosts they infect, members of the Parvoviridae family were divided into the two subfamilies. In contrast to those that infect vertebrate hosts, the groups of parvoviruses that infect invertebrate hosts (arthropods and crustaceans) belong to the subfamily Densovirinae. The subfamily Densovirinae, which includes the genera Densovirus, Brevidensovirus, Iteravirus, and Pefudensovirus, is part of the ninth edition of the taxonomical grouping created by the International Committee on Taxonomy of Viruses (ICTV). The subfamily Parvovirinae, which includes the genera: Bocavirus, Dependovirus, Erythrovirus, Amdovirus, and Parvovirus.

Classifications of Parvoviruses

To achieve enhanced taxonomic clarity for parvoviruses, the newest ICTV report used a modified standard for classification that needs a whole or nearly complete genome of the viruses. This led to the introduction of new species and genera into the two subfamilies of the family Parvoviridae. Based on their need for reproduction, members of the subfamily Parvovirinae are now divided into depend parvoviruses and autonomous parvoviruses. The autonomous parvoviruses do not need a helper virus to replicate well inside of cells, unlike the dependent varieties. Amdoparvovirus, Aveparvovirus, Dependoparvovirus (Dependovirus), Erythroparvovirus (Erythrovirus), Copiparvovirus, Bocaparvovirus (Bocavirus), Protoparvovirus (Parvovirus), and Tetraparvovirus are the eight genera that make up the subfamily Parvovirinae [2]. The classical forms appear in the final four genera. The use of more recent, novel molecular tools like high-throughput sequencing and the sporadic advances in molecular technology that led to the development of nucleic acid amplification techniques for pathogen detection have enabled the discovery of several novel swine parvoviruses. The novel viruses have been classified in a number of study proposals, with the majority of the classifications being taken into account in the most recent ICTV report that is still pending approval. Eight ungulate porcine parvovirus species, each with roughly twelve pig-infecting viruses and their variations, are now classified under four genera. The subfamily has yet to classify more recent ones into species and genera. They consist of PPVs 5, 6, and 7. However, the genus' sole pig-infecting virus to far has been porcine parvovirus 1 [3]. Due to their comparatively similar genetic homologies, human parvovirus 4 (PARV4), porcine parvovirus 2 (PPV2), and porcine parvovirus 3 (PPV3) were placed in the same genus as Tetraparvovirus. Porcine parvovirus 4 (PPV4) and bovine parvovirus 2 are both members of the genus Copiparvovirus, and it has been suggested that this group also includes the novel PPVs 5 and 6, as they have consistently clustered together in phylogenetic analyses. Additionally, according to the published classification, four separate genera of swine parvoviruses

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are currently made up of eight pig viruses and virus variations that are grouped together under the genus Bocaparvovirus [4].

There is still a lack of knowledge on the significance and pathogenicity potential of the viral pathogens to the world swine population. Many of the recently found pig parvoviruses have not yet been thoroughly examined. Because of this, an overview of the detection and prevalence of other viruses will be appropriately stressed while the classical porcine parvovirus (PPV), also known as porcine parvovirus type 1 (PPV1), will be briefly reviewed for the purposes of this review as a representative of other porcine parvoviruses [5]. The epidemiology of porcine parvoviruses in swine herds of African nations will also be highlighted in the later section of this review, taking into account the past, present, and future. This is quite important given that the area is seen to be moving relatively quickly. low rate of adoption of new technology for infectious agent identification; this aberrant trend may jeopardise worldwide efforts to prevent and control the numerous new and reemerging animal and human diseases that are wreaking havoc on our planet.

History of Porcine Parvovirus

Early in the 1960s, occurrences of reproductive failure on commercial swine farms were common due to unidentified causes that specialists believed could be related to a variety of factors, including nutrition and the environment [6]. Following its isolation in Germany as a byproduct of the production of the classical swine fever virus in pig cell cultures in the middle of the 1960s, PPV1 was subsequently confirmed to be linked to swine reproductive losses. The clinical signs of the PPV1 reproductive disease, which are widely known as SMEDI, were later identified as recurrent oestrus in sows, abortion, and farrowing of mummified or stillborn foetuses [7]. The virus's pathologic impact on a pregnant pig and its foetuses is caused by its preference for actively proliferating cells like depending on the stage of the sow's gestation, foetal infections frequently result in death due to the presence of cells such foetal myocardiocytes. Numerous findings from earlier research on the main etiologic factors of swine reproductive failure have identified PPV as a significant cause of porcine foetal death. Approximately 35% (105 of 302) of the dead foetuses collected and analysed in the USA were found to have PPV1 in their system, according to identified PPV1 as the sole cause of an acute outbreak of abortions in a domesticated herd of 500 wild boar females in Heilongjiang province, China; meanwhile, Tummaruk and Tantilertcharoen (2012) found that 86% (143/166) of gilts killed due to reproductive failure in Thai swine herds were PPV1-positive. PPV1 seropositive [8].

Result

The virus, which is endemic in many regions of the world and may infect pig herds of all types, is thought to be particularly stable in the environment. A non-enveloped viral capsid encases PPV1's tiny, single-stranded, negative-sense DNA genome, which is about 5 kb in size [9]. Unique to the genome are different palindromic hairpin termini and the presence of two significant open reading frames (ORFs). Non-structural proteins 1 (NS1) are encoded by the ORF1 located at the 5′ end of the viral genome, and two more non-structural proteins can be produced via alternative splicing (NS2 and NS3). The non-structural proteins have some key enzymatic roles in the replication and packaging of viruses. At the 3′ end of the viral genome is the ORF2 that codes for the capsid proteins. While the VP3 protein is created by the proteolytic cutting of the VP2 protein, the VP1 and 2 proteins are created from variously spliced mRNAs. As previously discussed, the icosahedral capsid of the virus is built using about sixty copies of the capsid proteins. The virus's unusual environmental stability has an impact on how contagious and widespread it is. It is extremely thermostable and can withstand dry heat up to a temperature of 90 °C [10].

Discussion and Conclusion

Additionally, the virus is resistant to disinfectants such sodium hypochlorite at low concentrations of 2500 ppm and up to 70% ethanol. As a result, the viral diseases can continue to spread from one farm to another for months in contaminated pens, farm equipment, and clothing [11]. To stop the transfer of diseases and other related ones from farm to farm, careful adherence to biosecurity measures is required in piggier operations [12]. It is also possible to improve farm-to-farm transmission by replacing asymptomatic gilts. This is due to the fact that sick pigs that have had a good vaccination may not always have any clinical indication Foerster. When a susceptible farm uses an infected boar or sperm for breeding, the virus can also spread there; a seronegative boar can also contract the disease while mating with an infected sow's vaginal secretions. Additionally, the viral disease can spread within a herd through infected pigs' faeces, nasal secretions, and oral secretions [13].

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Conflict of Interest

None

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