Identification of Reassortant Pandemic H1N1 Influenza Virus in Korean Pigs

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Since the 2009 pandemic human H1N1 influenza A virus emerged in April 2009, novel reassortant strains have been identified throughout the world. This paper describes the detection and isolation of reassortant strains associated with human pandemic influenza H1N1 and swine influenza H1N2 (SIV) viruses in swine populations in South Korea. Two influenza H1N2 reassortants were detected, and subtyped by PCR. The strains were isolated using Madin–Darby canine kidney (MDCK) cells, and genetically characterized by phylogenetic analysis for genetic diversity. They consisted of human, avian, and swine virus genes that were originated from the 2009 pandemic H1N1 virus and a neuraminidase (NA) gene from H1N2 SIV previously isolated in North America. This identification of reassortment events in swine farms raises concern that reassortant strains may continuously circulate within swine populations, calling for the further study and surveillance of pandemic H1N1 among swine.

Keywords: H1N2 influenza A virus, pandemic (H1N1) 2009 influenza A virus, reassortment, South Korea, swine

Swine influenza viruses (SIVs) are enveloped, segmented, single-stranded, negative-sense RNA viruses belonging to the family Orthomyxoviridae [22]. The viral envelope is studded with two glycoproteins [hemagglutinin (HA) and neuraminidase (NA)], which are essential for the biological function of the virus. The 16 known HA proteins and 9 known NA proteins combine in various permutations to create many different subtypes [1, 8, 27].

Influenza viruses undergo major evolutionary change by acquiring a new genomic segment from another influenza virus, effectively becoming a new subtype, by a process known as antigenic shift [26]. Animals such as pigs and birds facilitate viral evolution by serving as reservoirs of influenza viruses. Swine have become known as “mixing vessels” for influenza viruses because of their dual susceptibility to both human and animal strains of influenza, which allows them to serve as containers for the genetic reassortment of these strains [3, 4, 6, 7].

Since 1999, several newly developed viruses created by the reassortment of human, swine, and avian genes have been isolated in the United States [21, 41]. Such genetic reassortment can occur when swine are simultaneously infected with different influenza A viruses, such as human, swine, and avian strains. This process has created new strains of influenza A, including those with pandemic potential. Indeed, the reassortment of influenza A viruses among human, European and Asian swine, and avian strains appears to have created a new influenza A virus that led to the first pandemic in this century: the 2009 (H1N1) swine influenza pandemic. As it contains two genes from flu viruses that normally circulate among swine in Europe and Asia as well as avian and human genes, the H1N1 strain is referred to as a quadruple reassortant virus [5, 9, 28, 38].

This study detected and isolated swine H1N2 reassortant strains of pandemic H1N1 virus in swine populations in South Korea. Based on the phylogenetic analysis and molecular characterization of virus isolates, this study aimed to determine whether these isolates have undergone various forms of genetic evolution, such as mutations, antigenic drift as point mutations, and/or antigenic shift as genetic reassortment.

Materials and Methods

Samples for Virus Isolation
A total of 313 lung, tonsil, and nasal swab samples were obtained between January 2010 and September 2010, from various swine farms in nine provinces of South Korea. All specimens were submitted to the virology laboratory of the College of Veterinary Medicine, Seoul
National University. Ninety-one samples showed fever at the sampling, and others appeared clinically normal. Upon arrival of samples, the nasal swab samples were collected into an antibiotic-containing transport medium, and pooled organs were homogenized in phosphate-buffered saline (PBS) after chopping. Ten-percent homogenate suspensions were prepared in PBS (pH 7.4) and centrifuged at 3,000 × g for 10 min. Supernatants were used for RNA extraction and virus isolation.

RNA Extraction and Reverse Transcription
Viral RNA was extracted from each suspension using TRIzol LS (Invitrogen Corp., Carlsbad, CA, USA) according to the manufacturer’s instructions. Reverse transcription (RT) was performed using random hexamer primers (TaKaRa, Otsu, Japan). A volume of 10 µl of extracted RNA and 1 µl of 100 pmol random primer were mixed and heated to 95°C for 5 min and then chilled at 4°C for 5 min. After adding the remaining reagents, 5× First-Strand Buffer (250 mM Tris-HCl, 375 mM KCl, 15 Mm MgCl2), 2 µl of 0.1 M DTT, 1 µl of dNTPs, and 200 µl of M-MLV reverse transcriptase, the resulting mixture was incubated at 37°C for 60 min. The reaction was then inactivated by heating at 95°C for 5 min. The cDNA was either stored at −20°C or immediately amplified by RT-PCR.

Virus Detection by PCR
SIVs were detected by RT-PCR targeting the M gene. The PCR was performed using iStarMaster mix PCR Kits (iNtRON Biotechnology, Sungnam, South Korea) according to the manufacturer’s instructions. An SIV-positive control was kindly provided by the Research Unit (National University) according to the manufacturer’s instructions. The nucleotide sequences were aligned using the Basic Local Alignment Search Tool (BLAST, http://blast.ncbi.nlm.nih.gov/blast.cgi). The nucleotide sequences were aligned using the ClustalX ver. 2.0.12 with reference viruses using BioEdit software ver. 7.0.5.3 (http://www.ncbi.nlm.nih.gov/Blast.cgi). The amino acid sequence of isolates was also generated and aligned using the same programs (ClustalX ver. 2.0.12, BioEdit software ver. 7.0.5.3).

RESULTS
Detection and Subtyping of SIV Isolates
After performing RT-PCR targeting the M gene, 25 samples from 21 farms in seven provinces of South Korea, except Jeonbuk and Gwangwon, showed positive results for SIV (Table 1). SIV positive pigs were collected from the provinces of Chungnam and Kyonggi (n = 5), Gyeongbuk and Gyeongnam (n = 4), Chungbuk and Cheonnam (n = 3), and Jeju (n = 1). SIV was detected from nasal swab samples in 2 pigs and the other positive samples were detected from pooling of solid organs and lymph nodes.

Two influenza A viruses (Sw/Korea/4940/2010 and Sw/Korea/4941/2010) were isolated using MDCK cells. These two isolates were collected in April 2010, and their symptoms included respiratory abnormalities like respiratory distress

Sequencing and Phylogenetic Analysis
Amplification of the full gene of the viral HA, NA, M, and NS segments and partial gene of polymerase PBI (202–843), PB2 (1291–2103), PA (754–1197), and NP (496–1036) gene segments was performed with primers as previously described [16,42]. PCR for sequencing was performed using iStarMaster mix PCR according to the manufacturer’s instructions. SIV sequences were confirmed and highly similar sequences were identified using the Basic Local Alignment Search Tool (BLAST, http://blast.ncbi.nlm.nih.gov/blast.cgi). The nucleotide sequences were aligned using the ClustalX ver. 2.0.12 with reference viruses using BioEdit software ver. 7.0.5.3 (http://www.ncbi.nlm.nih.gov/Blast.cgi). The nucleotide sequences were aligned using the ClustalX ver. 2.0.12 multiple sequence alignment program (http://www.clustal.org) [33] and compared with reference viruses using BioEdit software ver. 7.0.5.3 (http://www.ncbi.nlm.nih.gov/Blast.cgi). The nucleotide sequences were aligned using the ClustalX ver. 2.0.12, BioEdit software ver. 7.0.5.3).

Virus Isolation and Subtyping of SIV
Among the samples, pooled organs from 25 samples determined influenza positive by RT-PCR were prepared in PBS as described in

the section above. The supernatant was filtered at 0.45 µm and immediately used for inoculation. Viruses were isolated in Mad–Darby canine kidney (MDCK) cells. A cell monolayer of MDCK was prepared by 24 h of incubation in Dulbecco’s modified Eagle’s medium (DMEM) with 7% fetal bovine serum in 12-well culture plates. The medium was discarded and the cell monolayer rinsed twice with PBS. The cells were incubated with 3 ml of prepared inoculums for 1 h at 37°C, after which the inoculums were removed and the cell monolayer was rinsed once with PBS. After 4 ml of DMEM containing 0.3 µg/ml of TPCK-trypsin was added per well, the cells were further incubated at 37°C for 48–76 h and observed daily for CPE [19,28,29]. After observation of CPE, the supernatant of isolates was used for further passage, identified by HA testing, and tested for influenza type A by RT-PCR assay. The isolate subtype was determined using one-step multiplex RT-PCR with primers to amplify H1, H3, N1, and N2 SIVs [17].
### Table 1. Details of positive samples for SIV.

<table>
<thead>
<tr>
<th>Sample number</th>
<th>Collected tissues</th>
<th>Age</th>
<th>Geographic origin</th>
<th>Sampling date</th>
<th>Clinical signs</th>
<th>Other viral or bacterial infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>4632</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Suckling</td>
<td>Gyeongbuk</td>
<td>Jan 20, 2010</td>
<td>Unknown</td>
<td>PRRSV+</td>
</tr>
<tr>
<td>4679</td>
<td>Lung, Lymph node</td>
<td>Weaning</td>
<td>Gyeongbuk</td>
<td>Feb 5, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+, E. coli (intestine)</td>
</tr>
<tr>
<td>4730</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Kyonggi</td>
<td>Feb 25, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, E. coli (intestine)</td>
</tr>
<tr>
<td>4731</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Kyonggi</td>
<td>Mar 9, 2010</td>
<td>Unknown</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4779</td>
<td>Lung, Lymph node</td>
<td>Suckling</td>
<td>Gyeongbuk</td>
<td>Feb 25, 2010</td>
<td>Respiratory sign</td>
<td>PCV2+, E. coli (intestine), Strepto spp. (lung)</td>
</tr>
<tr>
<td>4803</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Kyonggi</td>
<td>Mar 15, 2010</td>
<td>Enteric sign</td>
<td>PRRSV+, E. coli (intestine)</td>
</tr>
<tr>
<td>4851</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Gyeongbuk</td>
<td>Mar 24, 2010</td>
<td>Respiratory sign</td>
<td>PCV2+, E. coli (intestine), Strepto spp. (lung)</td>
</tr>
<tr>
<td>4876</td>
<td>Lung</td>
<td>Weaning</td>
<td>Gyeongnam</td>
<td>Mar 30, 2010</td>
<td>Unknown</td>
<td>PRRSV+, PCV2+, APP (Lung), E. coli (intestine)</td>
</tr>
<tr>
<td>4901</td>
<td>Lung, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Chungnam</td>
<td>Apr 6, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4902</td>
<td>Lung, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Chungnam</td>
<td>Apr 6, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4927</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Suckling</td>
<td>Kyonggi</td>
<td>Apr 7, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4929</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Gyeongnam</td>
<td>Apr 8, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4936</td>
<td>Lung, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Chungnam</td>
<td>Apr 8, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+, E. coli (intestine)</td>
</tr>
<tr>
<td>4940</td>
<td>Lung, Lymph node</td>
<td>Suckling</td>
<td>Chungnam</td>
<td>Apr 9, 2010</td>
<td>Respiratory sign</td>
<td>E. coli (intestine)</td>
</tr>
<tr>
<td>4941</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Suckling</td>
<td>Gyeongnam</td>
<td>Apr 9, 2010</td>
<td>Neurological sign</td>
<td>E. coli (intestine)</td>
</tr>
<tr>
<td>4975</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Gyeongbuk</td>
<td>Apr 16, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, E. coli (intestine)</td>
</tr>
<tr>
<td>4979</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Suckling</td>
<td>Chungnam</td>
<td>Apr 20, 2010</td>
<td>Enteric sign</td>
<td>E. coli (intestine)</td>
</tr>
<tr>
<td>4984</td>
<td>Lung, Lymph node</td>
<td>Suckling</td>
<td>Jeonnam</td>
<td>Apr 21, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>4985</td>
<td>Nasal swab</td>
<td>Suckling</td>
<td>Jeonnam</td>
<td>Apr 21, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, PCV2</td>
</tr>
<tr>
<td>4986</td>
<td>Nasal swab</td>
<td>Suckling</td>
<td>Jeonnam</td>
<td>Apr 21, 2010</td>
<td>Respiratory sign</td>
<td>PCV2+</td>
</tr>
<tr>
<td>4999</td>
<td>Lung, Lymph node</td>
<td>Weaning</td>
<td>Gyeongnam</td>
<td>May 3, 2010</td>
<td>Unknown</td>
<td></td>
</tr>
<tr>
<td>5008</td>
<td>Lung</td>
<td>Weaning</td>
<td>Chungnam</td>
<td>May 7, 2010</td>
<td>Unknown</td>
<td>PRRSV+, E. coli (intestine), Strepto spp. (lung)</td>
</tr>
<tr>
<td>5015</td>
<td>Lung, Liver, Kidney, Spleen, Lymph node</td>
<td>Weaning</td>
<td>Chungnam</td>
<td>May 13, 2010</td>
<td>Unknown</td>
<td>PRRSV+, PCV2+</td>
</tr>
<tr>
<td>5171</td>
<td>Lung, Lymph node</td>
<td>Suckling</td>
<td>Jeju</td>
<td>Jul 8, 2010</td>
<td>Respiratory sign</td>
<td>PRRSV+, Strepto spp. (lung)</td>
</tr>
<tr>
<td>5185</td>
<td>Lung, Lymph node</td>
<td>Suckling</td>
<td>Chungnam</td>
<td>Jul 13, 2010</td>
<td>Respiratory sign</td>
<td>PCV2+</td>
</tr>
</tbody>
</table>

### Table 2. Subtype and brief history of SIV isolates in MDCK cells used in this study.

<table>
<thead>
<tr>
<th>Virus name</th>
<th>Subtype</th>
<th>Tissues collected from</th>
<th>Age</th>
<th>Sampling date</th>
<th>Geographic origin</th>
<th>Clinical signs</th>
<th>Other viral and bacterial infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>A/Sw/Korea/4940/10</td>
<td>H1N2</td>
<td>Lung, Lymph node, Spleen</td>
<td>Suckling</td>
<td>Apr 9, 2010</td>
<td>Chungnam</td>
<td>Respiratory sign</td>
<td></td>
</tr>
<tr>
<td>A/Sw/Korea/4941/10</td>
<td>H1N2</td>
<td>Lung, Liver, Kidney, Spleen, Tonsil, Lymph node</td>
<td>Suckling</td>
<td>Apr 9, 2010</td>
<td>Gyeongnam</td>
<td>Neurological sign</td>
<td>E. coli + (intestine)</td>
</tr>
</tbody>
</table>
and abdominal breathing, and neurological abnormalities (Table 2). At necropsy, some blood-tinged fibrinous exudate in the airways, enlarged and hemorrhagic bronchial lymph nodes, and stomach that contained milk curd were evident. Lungs were red, wet, and heavy, with distinct areas of purple, firm, collapsed lungs.

The one-step multiplex RT-PCR using a dual priming oligonucleotide (DPO) system was performed to identify the H1N2 subtypes without SIV co-infection. DPO contains two separate priming regions joined by a polydeoxyinosine linker for a high level of PCR specificity, even under suboptimal PCR conditions. The H1 and N2 SIV subtypes of 2 isolates were identified.

**Plaque Formation by Plaque Assay**

The formation of plaques with influenza viruses could be seen 72 h after infection, by visual examination. After 4 days, the plaques stand out as white and small in 6-well culture plates. They were best seen against a clear, black background; they appeared as round, uncolored areas contrasting with the red color of the surrounding living cells, stained with neutral red.

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**Fig. 1.** Phylogenetic analysis of the HA (A) and NA (B) of swine H1N2 isolates. Trees were constructed using the neighbor-joining method and applied the maximum composite likelihood model with 1,000 bootstrap replications using the MEGA (4.0) program. Similarities were determined by comparison with sequences of reference strains available in GenBank.
The suspension of pure plaque was inoculated to MDCK cells, and induced CPE through tissue culture passage. HA titers of 1/16 to 1/256 were obtained from second passage to fourth tissue culture passages. By the 5th passages, infectious virus was demonstrated by plaque assay ranging from 8 × 10⁶ to 1.3 × 10⁷ plaque-forming units (pfu/ml).

Phylogenetic Analysis
The results of phylogenetic analysis indicated that all the genes of Sw/Korea/4940/2010 and Sw/Korea/4941/2010, with the exception of NA, fell closely into the cluster of H1N1 pandemic viruses. H1 of these isolates showed 98.84% nucleotide identity with local human pandemic H1N1 isolates in South Korea (Korea/CJ1,9,15,24/09) [30] and 98.46% nucleotide identity with swine pandemic H1N1 (Sw/Korea/SCJ02, 07,10,13/2009; Fig. 1A). The NA genes of all H1N2 isolates showed 96–99.7% nucleotide identity with both Northern American swine H1N2 and pre-existing Korean H1N2 SIVs (Sw/Minnesota/00194/2003; Sw/Korea/CY02/2007; Sw/Korea/PZ4/2006; Sw/Korea/JL01/2005; Sw/Korea/S14-15/2006; Fig. 1B) [12–14, 16, 21, 23, 39]. The internal genes of swine isolates (M, NP, NS, PA, PB1, and PB2) had the greatest similarity to pandemic (H1N1) 2009 viruses, with 94–99.98% nucleotide identity. In the M gene, Sw/Korea/4940/2010 and Sw/Korea/4941/2010 grouped with the European avian-like

Fig. 1. Continued.
strain because they shared 98% of their nucleotide identity with A/swine/England/WVL7/1992 and A/swine/Spain/50047/2003, and 94% with a wholly avian-like H1N1 SIV (A/swine/Zhejiang/1/2007). The lineage of gene segments of the isolates is shown in Table 3.

**DISCUSSION**

The success of interspecies transmission of the influenza virus is dependent on viral gene constellation and binding to the host cells by α-2,3- and α-2,6-galactose sialic acid receptors on the host cell surface.

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**Table 3.** Genotypes of influenza virus H1N2 isolates from pigs, South Korea.

<table>
<thead>
<tr>
<th>Virus</th>
<th>Subtype</th>
<th>Lineage of gene segments of isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>HA (HA)</td>
</tr>
<tr>
<td>Sw/Korea/4940/2010</td>
<td>H1N2</td>
<td>Classical swine</td>
</tr>
<tr>
<td>Sw/Korea/4941/2010</td>
<td>H1N2</td>
<td>Classical swine</td>
</tr>
</tbody>
</table>

HA, hemagglutinin; NA, neuraminidase; PB2, basic polymerase; PB1, polymerase; PA, acid polymerase; NP, nucleoprotein; M, matrix; NS, nonstructural; Sw, swine (GenBank Accession No. JF346676–JF346747).

Nucleotide position of partial viral gene: PB2, 1291–2103; PB1, 202–843; PA, 754–1197; NP, 496–1036. Amplification of viral genes was performed as previously described [16]. Amplification of HA, NA, M, and NS genes was performed as previously described [15, 17].
The isolation of reassortant strains from swine populations of different countries also raises concerns about the possible establishment of an H1N1 pandemic among swine populations [1, 20]. Owing to continued transmission of the 2009 pandemic influenza virus H1N1 within and between human and swine populations [9, 25, 30], swine reassortant viruses with pandemic virus genes have been detected in many countries [15, 24, 30, 31, 37]. Recently, a reassortant virus containing HA and internal genes from the pandemic H1N1 virus, and NA genes from swine subtype H1N2 virus has been characterized in Italy [20]. A reassortant strain containing 7 RNA segments derived from different virus circulating in China and the NA gene from the pandemic H1N1 virus has been identified in Hong Kong [35]. Unlike the reassortant H1N1 virus in Hong Kong, a H1N2 reassortant strain in the United Kingdom containing HA and NA genes from the swine subtype H1N2 virus and internal genes from the pandemic H1N1 virus has been isolated from swine [10]. In addition, a reassortant H1N1 virus from a swine was also discovered in Germany and Thailand [15, 32]. The reassortant virus isolated in Germany consists of all gene segments from pandemic H1N1 except for the NA derived from Eurasian swine lineage [32]. A novel swine reassortant H1N1 virus reported in Thailand consists of 7 RNA gene segments from the 2009 pandemic virus H1N1 and a NA gene from a Thai swine H1N1 isolate [15]. Such reassortment events involving pandemic H1N1 influenza viruses have increased recently [10, 35].

In this study, among the 313 tissue samples, including lung, tonsil, and nasal swabs, obtained from swine farms in South Korea, influenza A viruses were detected in 25 samples by RT-PCR targeting the M gene, which is highly conserved in influenza A viruses. Among these 25 samples, 2 viruses (Sw/Korea/4940/2010 and Sw/Korea/4941/2010) were isolated using MDCK cells and determined as H1N2 subtype by multiplex RT-PCR using a dual priming oligonucleotide (DPO) system [17]. The 2 isolates identified in this study, both confirmed to have undergone antigenic changes by comparison of amino acid sequences at HA antigenic sites, have been confirmed to be the result of reassortment of genes among the classical swine, human, avian, and Eurasian swine strains. Furthermore, there are porcine trachea epithelial cells that express both the α-2,3-galactose (NeuAcc2,6Gal) linkage on sialyloligosaccharides for avian influenza and the NeuAcc2,6Gal linkage for human influenza. The identification of these reassortant events can, therefore, provide further evidence of the role of swine as mixing vessels and the possibility of the emergence of new influenza strains [2, 36].

The widespread human-to-swine transmission of the pandemic (H1N1) 2009 influenza virus in South Korea was reported [30]. However, no case of reassortment of the pandemic virus from swine in this country has been reported prior to this study. The identification of a swine reassortant virus H1N2 associated with the H1N1 pandemic influenza virus in this study shows the possibility of the development of a novel swine virus following transmission of the pandemic influenza virus from human to swine populations in South Korea.

The reassortant virus characterized in this study, as that between the 2009 pandemic virus H1N1 and the SIV subtype H1N2, is the first of its kind in Korea and the second in Asia after Hong Kong. As described previously, the pandemic H1N1 virus and H1N2 SIVs may continuously co-circulate within swine populations in South Korea, providing opportunities for reassortment. The emergence of novel reassortant H1N2 strains derived from 2009 pandemic virus H1N1 within swine populations in this country raises the concern that these viruses will become established within swine populations. Thus, it is very important to monitor the emergence of novel swine reassortant strains. This newly identified reassortant virus may represent not only a pandemic zoonotic threat but also an agricultural and economic threat for the South Korean agricultural and economic threat for the South Korean economy.
swine industry. As such, it is necessary for researchers to identify and monitor the emergence of virulent variations and novel viruses within swine populations in South Korea.

REFERENCES


