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Research article

Molecular characterization of porcine parvovirus 1 based on partial VP2 gene in the ovaries of Thai pigs

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Abstract

Porcine parvovirus 1 (PPV1) is the causative agent of swine reproductive disease, particularly in naive gilts and sows. This study aimed to investigate the prevalence and genetic diversity of the partial nucleotide sequence of the VP2 gene and to compare the substitution of amino acid residues that affect relevant biological properties. The prevalence of PPV1 was found to be 12% (12/100) when the viral genome was detected using polymerase chain reaction (PCR). Determination of the genetic diversity of a partial nucleotide sequence of the VP2 gene through phylogenetic analysis indicated that a single cluster of Thai PPV1s was allocated on the phylogenetic tree. According to a comparison of the substitution of amino acid residues that affected the biological properties at 378, 383, 365, and 436 of the VP2 capsid protein between the 12 Thai PPV1s, the Kresse strain (a surrogate pathogenic strain), and the NADL-2 strain (a surrogate nonpathogenic strain). It was determined that the substitution of amino acid residues at 378, 383, and 436 of 12 Thai PPV2s was identical to those of the Kresse strains. The substitution of amino acid residues at 436 of the 12 Thai PPV1s was similar to that of a proven virulent strain in vivo. Additionally, substituting amino acid residue at 320 of the VP2 capsid protein revealed that seven Thai PPV1s were associated with isoleucine PPV1s and identical to that of both surrogate strains, whereas five Thai PPV1s were associated with threonine. This outcome was similar to what had been deposited in GenBank. Our data suggest that Thai PPV1s isolated from the ovaries of pigs raised in Chiang Mai may have originated from the Kresse strains. Based on a change of VP2 capsid protein that occurred amongst the substitution amino acid residue at 320 of the VP2 capsid protein, viruses found in this region were determined to be similar to those found in other areas. This was likely because the viruses had adapted to evade the immune systems of animals.

Keywords: Genetic diversity, Kresse strain, Porcine parvovirus

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INTRODUCTION

Incidences of the porcine parvovirus have been reported in pig populations throughout the world, including in Thailand (Streck et al., 2013; Saekhow and Ikeda, 2015; Garcia-Camacho et al., 2020; Deka et al., 2021; Li et al., 2021; Mai et al., 2021; Kim et al., 2022). At present, porcine parvovirus 1 (PPV1) and PPV2-PPV7 have been detected in several regions around the world as a consequence of molecular characterization. Notably, a degree of success has been achieved in detecting the whole genome or the nearly complete genome of PPV2-PPV7 using certain molecular tools. This has been attributed to the fact that the PPV1-PPV7 parvoviruses share comparable biological characteristics of genome organization and the phospholipase A2 (PLA2) motif within their genomes (Streck et al., 2013; Cotmore et al., 2019). However, phylogenesis analysis of the nearly complete or entire genome has revealed discoveries of porcine parvovirus in the form of PPV2 to PP7. PPV1 is classified as a protoparvovirus belonging to the subfamily *Parvovirinae*, family *Parvoviridae* (Cotmore et al., 2019; Walker et al., 2020). The viral genome consists of two main open frames (ORFs), ORFs1 and ORFs2. ORFs 1 is encoded as nonstructural protein (NSP) 1 and NSP 2, while ORFs 2 is encoded as viral proteins (VP) 1, VP 2, and VP 3 capsid proteins (Mészáros et al., 2017).

PPV1 has been linked to reproductive disease in seronegative pig herds. The pathogenesis of PPV1 infection in pregnant sows involves abortions, mummified fetuses, stillbirths, and infertility. These are known to occur as consequences of infections in pregnant pigs during different stages of pregnancy (Joo et al., 1976; Too and Love, 1986). PPV1 can be detected by the distribution of a viral genome in the cells of various organs, including the heart, lungs, kidneys, spleen, endometrial of the uterus, small intestines, and follicular fluid in the ovaries (Kresse et al., 1985; Oraveerakul et al., 1990; Oraveerakul et al., 1993; Bachanek-Bankowska et al., 2018).

According to the entire capsid protein sequences that are encoded for VP1/VP2, PPV1 can be divided into two groups: a classical group of viruses and a new group (Zimmermann et al., 2006). Notably, viruses of the classical PPV1 virus group have more commonly been reported (Ellis et al., 2000; Kim et al., 2003; Mészáros et al., 2017). The classical group of the PPV1 virus comprises two strains that have been examined in terms of their pathogenicity. Accordingly, the Kresse strain serves as a surrogate pathogenic strain for the virus, while the NALD-2 strain serves as a surrogate nonpathogenic strain for the virus (Joo et al., 1976; Too and Love, 1986; Zeeuw et al., 2007). It has been hypothesized that the VP2 capsid protein of the two surrogate strains could modify certain biological properties e.g. viral tissue tropism while stimulating the host immune system to produce a tentative neutralizing antibody (Vasudevacharya and Compans, 1992; Sun et al., 2015; Liu et al., 2020). This would likely be achieved by stimulating the host immune system to produce a tentative neutralizing antibody and through viral tissue tropism. Epitopes that generate neutralizing antibodies are found in the VP2 capsid protein have been identified according to the antigenic structure of PPV using synthetic peptides, monoclonal antibodies, and viral-like particles (Zhou et al., 2010; Guo et al., 2014; Sun et al., 2015; Hua et al., 2020; Liu et al., 2020; Streck et al., 2022). The VP2 capsid protein plays a role in cell-virus interaction before viral replication

because a change in residue on the VP2 capsid protein has been known to contribute to viral replication. Because the Kresse strains exhibit the ability to replicate in more cell types or organs than the NADL-2 strains, it has been suggested that the VP2 capsid protein of the two surrogate strains could effectively modify viral tissue tropism. (Vasudevacharya and Compans, 1992; Fernandes et al., 2011) When comparing the substitution amino acid residues of the VP2 capsid protein of the two surrogate strains, the viral VP2 capsid proteins were found to be very similar. This determination indicated that there was a six-residue discrepancy in the VP2 amino acid that included 45, 215, 378, 383, 436, and 565 (Bergeron et al., 1996). When three of the residues, namely 378, 383, and 436, on the VP2 capsid protein of the Kresse strain changed, the Kresse strains were observed to reduce their ability to replicate in the originating porcine cells. (Fernandes et al., 2011) Moreover, a study conducted on field strains has determined that residue 436 in the virulent strains might affect the pathogenicity of PPV1 in vivo. (Zeeuw et al., 2007)

In Thailand, two studies on the prevalence of PPV1 in pigs have been conducted. The prevalence of infection by serological examination in pigs was determined to be 97%, whereas infection by genome detection was 53% (Tummaruk and Tantilertcharoen, 2012; Saekhow et al., 2015). Despite previous investigations on its prevalence, there has been a deficiency in the number of studies on the genetic diversity of the ORF2 gene that encoded the VP2 capsid protein; on the other hand, there have been no studies on PPV1 involving pig ovarian tissue samples. Thus, the current study will determine the prevalence and analyze the partial expression of the VP2 gene in pig ovarian tissue samples obtained from pigs raised in Chiang Mai and Lamphun Provinces of Thailand.

MATERIALS AND METHODS

Sample collection

Ovary samples from 100 domestic pigs were obtained in 2021 from slaughterhouses located in Chiang Mai and Lamphun Provinces. All the clinically healthy pigs were around six months of age and had been raised in Chiang Mai and Lamphun Provinces. The pig ovary collection protocol complied with relevant animal welfare rules (reference number R20/2563) and was approved by the Animal Ethics Committee of the Faculty of Veterinary Medicine, Chiang Mai University.

Viral DNA extraction

Initially, about 0.1 g of each ovary sample was suspended in 0.9 mL of normal saline solution and homogenized in 1.5 mL tubes using a tissue homogenization device set (Bioneer, Daejon, Korea). The tissue homogenates were centrifuged at 3,000 g for ten minutes. The supernatant was then collected and maintained at −20°C. Subsequently, the viral DNA was isolated from the supernatant of the 10% ovary homogenate using a Neucleospin tissue kit (Machery-Nagel, Duren, Germany) according to the manufacturer's instructions.

Detection of PPV1 genomes by PCR

To investigate the prevalence of PPV1, PCR was conducted using a pair of primers as screening primers, namely mPPVF and mPPVR (Table 1). The PCR reaction was conducted in the presence of Taq polymerase (Toyobo, Osaka, Japan). The PCR profile consisted of an initiation step at 94 °C for five minutes followed by 35 cycles of denaturation at 94 °C for 30 sec, an annealing step at 55 °C for 30 sec, an extension step at 72 °C for 1.5 min, and a final step at 72°C for seven minutes. The expected size of the PCR products was determined by electrophoresis in 1.5% agarose gel containing 1:20,000 a RedSafe™, nucleic acid staining solution (iNtRON Biotechnology, Soul, Korea) in Tris-acetate-EDTA (TAE) buffer at 100 V for 30 min. The presence of the viral genome was assigned based on the expected size of the PCR product after pictures were recorded under a UV light source (Analytica, CA, USA).

Table 1 A list of the PCR primers

Primers	Sequence (5'-3')	Polarity	Position *	Purpose	Reference
mPPVF	CACAGAAGCAACAGCAATTAGG	+	3781–3802	Screening a prevalence	(Ogawa et al., 2009)
mPPVR	CTAGCTCTTGTGAAGATGTGG	-	3963–3983		
P1F	AACTCACTCATGGCAAACAAACAGA	+	3637–3661	Amplification and nucleotide sequencing of the partial VP2 gene	(Soares et al., 2003)
P8R	CAATGATAGTAGTACATGATTAACCAA	-	4580–4606		
P4F	CACTTTTACCTTCAGATCCAATAGG	+	4083–4107	Nucleotide sequencing of the partial VP2 gene	
P5R	TGCTGTTAATGGTCCATATGTATTGA	-	4140–4165		

*The Kresse strain (accession number U44978.1) was used as a reference strain for primer binding sites in this study

Phylogenetic analysis of VP2 capsid protein gene

In terms of all ovary tissue samples for which the PPV1 genome could be detected, 816 bp of VP2 were analyzed in terms of their genetic diversity. The positions located through the ORF2 of the Kresesse strain were used in this study. These were generated with pairs of the primers P1F and P8R. The amplification cycle consisted of an initiation step at 94 °C for 5 minutes followed by 35 cycles of denaturation at 94 °C for 30 sec, an annealing step at 58 °C for 1 min, an extension step at 72 °C for 1.5 min, and a final step at 72°C for 7 minutes. The PCR products were then purified using PCR Clean-up (Bioneer, Soul, Korea) according to the protocol suggested by the manufacturer. The purified PCR products and sequencing primers were sent to Marcogen, Korea as the nucleotide sequencing provider company. Two pairs of primers were used to determine nucleotide sequencing including the P1F and P4F forward primers and the P5R and P8R reward primers (Table 1). Chromatogram form direct sequencing involved the two PCR primers, while the internal overlapped sequence was subjected to phylogenetic analysis. The phylogenetic tree was constructed by using the Maximum- Likelihood method with 1,000 bootstraps using 816 bp of ORF2 that had been encoded with the VP2 capsid protein and Tamura-Nei model implemented in MEGA7 and 11. (Kumar et al., 2016; Tamura et al., 2021) The nucleotide sequences used in the phylogenetic analysis contained 12 Thai PPV1s and 36 nucleotide sequences

that had been deposited in GenBank. These included both surrogate strains (NADL-2 and Kresse strains). The accession numbers that belonged to Thai PPV1s were LC705159 to LC705170.

Analysis of the substitution of amino acid residues in the VP2 capsid protein

The substitution of amino acid residues in the VP2 capsid protein used in this study was obtained by the translation of the nucleotide sequence. The substitution of amino acid residues in the VP2 capsid protein of the surrogate strains (NADL-2 and Kresse strains) were identified as 378, 383, 565, and 436. Nucleotide sequences were translated to amino acid residues using MEGA7 and 11. Amino acid residues of the VP2 capsid protein were then aligned with two surrogate strains using ClustralW2 and any similarities between the Thai PPV1 viruses were determined using BLASTP alignment. (Thompson et al., 1994; Boratyn et al., 2019)

RESULTS

Prevalence of PPV1 in pig ovary samples

PCR was used to investigate the PPV1 genome in the ovary samples of about six months old pigs. Pairs of the screening primers used in this study revealed that 12 of 100 sows were positive (12%). Our results suggest that a low prevalence of PPV1 was detected in the ovaries of pigs raised on farms in our study location.

Genetic diversity of Thai PPV1 by phylogenic analysis

To investigate the genetic diversity of Thai PPv1s, partial sequences of the VP2 gene of Thai PPV1s were amplified using a pair of primers (P1 and P7). Phylogenetic analysis was constructed using a partial nucleotide sequence of ORF2 that had been encoded for the VP2 capsid protein in Thai PPV1s, and 36 referenced strains of nucleotide sequences have been deposited in GenBank, including surrogate strains (NADL-2 and Kresse strains). By examining the phylogenetic tree, the overall PPV1 VP2 nucleotide sequences indicated that they could be divided into two clades, specifically clade I and clade II (Figure 1). Twelve VP2 proteins of the Thai PPV1 nucleotide sequences were allocated to clade I as a single cluster with a nucleotide sequence difference being observed among the 12 Thai PPV1s with a maximum of 0.3% difference among them. Additionally, the phylogenetic tree showed that the VP2 nucleotide sequences of PPV1 were related geographically. This result suggested that the viral clusters that belonged to the clade I on the tree were often a single cluster originating from the same geographic region (Figure 1), e.g., Brazil, China, South Korea, and Germany. Our results indicate that a single cluster of the PPV1 strain would likely be correlated with a specific geographic region.

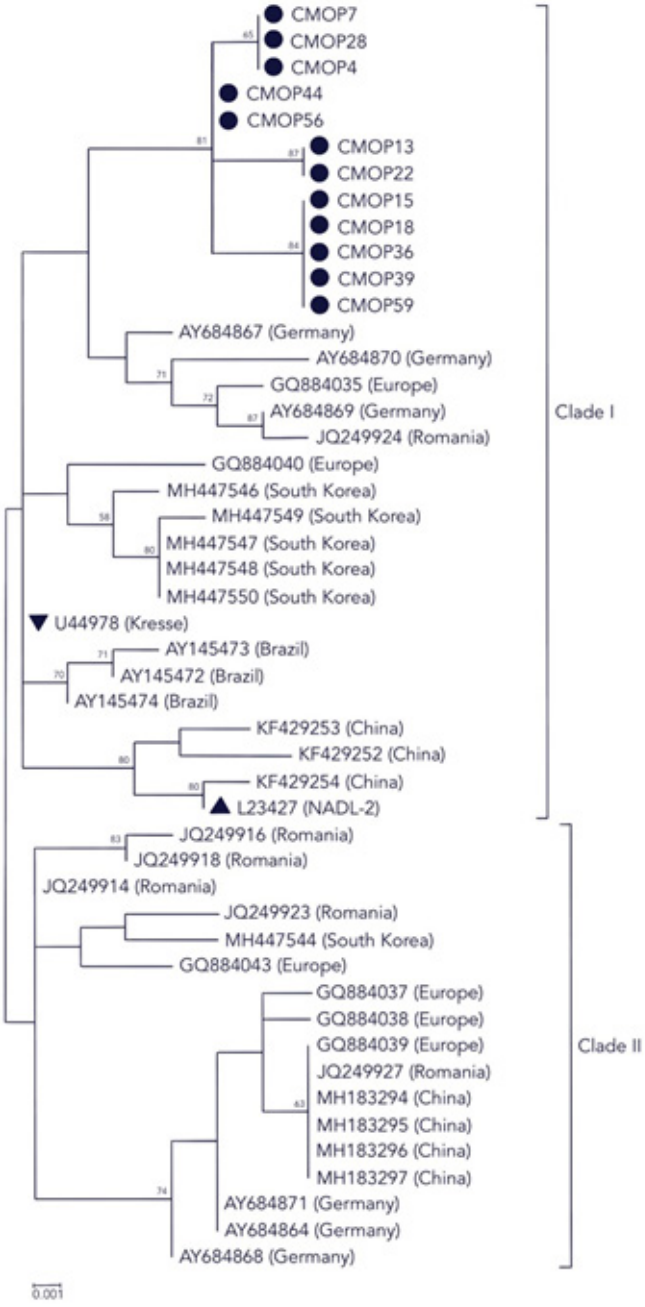

Figure 1 Genetic diversity of PPV1. It was constructed using a fragment of the ORF2 nucleotide sequence encoding VP2 capsid protein of 12 Thai PPV1s and 36 referenes nucleotide sequences of PPV1s that were deposited in the GenBank. The 12 Thai PPVs nucleotide sequences are denoted by black circles. For the both surrogate strains, black triangles represents the Kreese strain whereas the black inverted triangle represents the NALD2 strain. Initially, this research comprised 100 nucleotide sequences of PPV1, with 12 Thai nucleotide sequences PPV1 and 88 sequences from other geographical regions being included. However, within each study, the nucleotide sequence similar to the previous one was eliminated.

VP2 analysis by substitution of amino acid residues

A comparison substitution was conducted involving the amino acid residues in the VP2 capsid protein of the 12 Thai PPV1s that were found to affect relevant biological properties. In this experiment, the Kresse and NADL-2 strains were compared. The substitutions of the amino acid residues were 378, 383, 436, and 565. Consequently, the substitution amino acid residues mentioned above, together with other substitution amino acid residues in the VP2 capsid protein, were sorted into relevant reference strains in GenBank. In this study, the results of comparisons made involving the affected biological properties of the amino acid residues comprised 378, 383, and 565 of the 12 Thai PPV1s with two reference strains indicated that all 12 Thai PPV1s were comprised of glycine, glutamine, and lysine. This outcome was similar to the results of an analysis of the Kresse strain. In this position, the remaining affected biological properties of the amino acid residue 436 that belonged to all 12 Thai PPV1s involved threonine, serine for NADL-2, and proline for the Kresse strain (Figure 2 and Table 2). An observation of the amino acid residue 436 revealed that the 12 Thai PPV1s were similar to those of the proven virulent strains. The remaining amino acid residue 320 among the Thai PPV1s indicated that the similar residue of both reference strains was isoleucine, which was found in five Thai PPV1additiononally, seven of the Thai PPV1s that showed threonine were sorted in GenBank for the identical amino acid residue 320. The countries where threonine substitution has been found include Brazil, China, and Germany. Our data suggest that the Kresse strain has been spreading in Chiang Mai and Lamphun Provinces and that the virus has adapted as a variant that will likely affect the viral population.

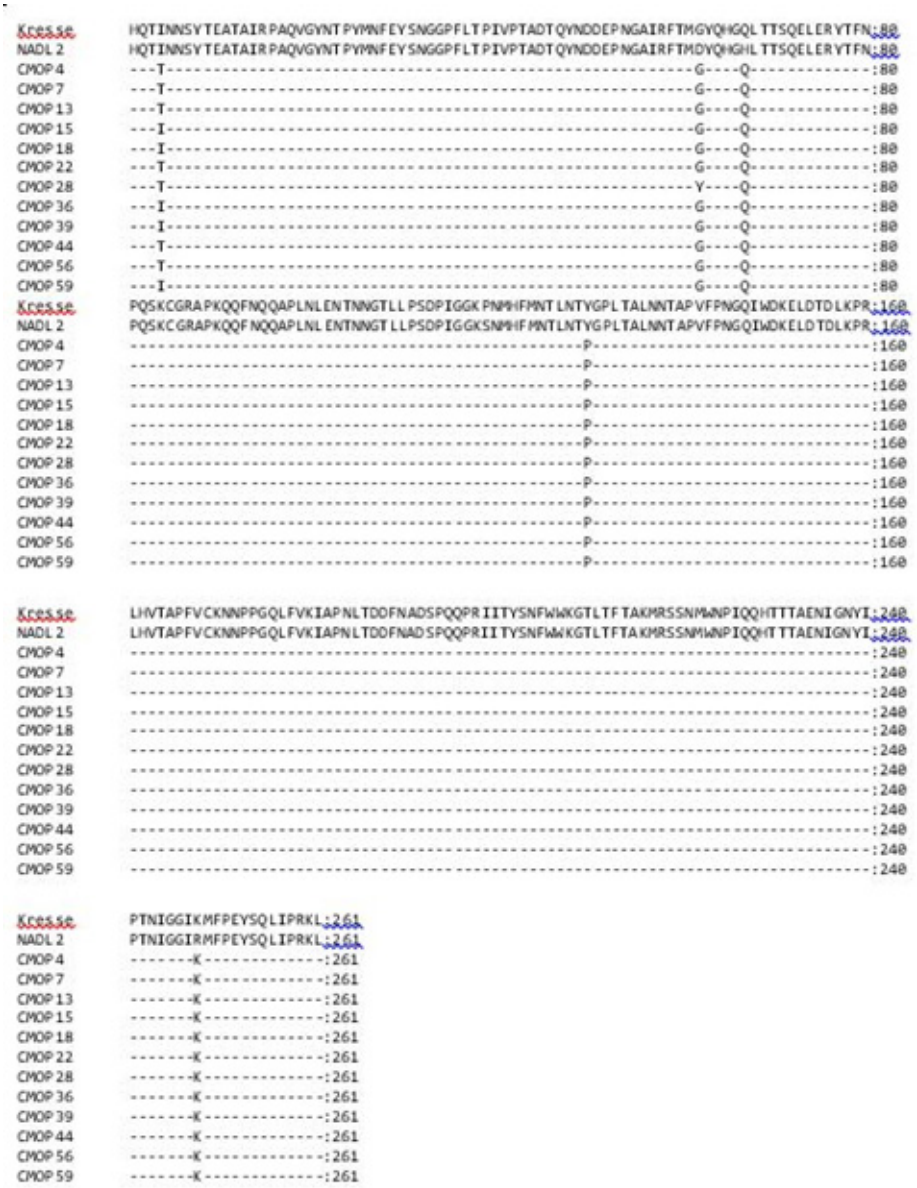

Figure 2 Alignment of the substitution of VP2 capsid protein. The alignment contained Kresse and NADL-2 strains as reference strains and 12 Thai PPV1s. The comparison of amino acid residue began at number 371 of the VP2 capsid protein of the Kresse strain and had a total amino acid residue of 261.The residues compared with two reference strains were 378, 383, 436, and 565 and were placed above the filter square. A residue at 320 of the Thai PPV1s indicate a number on the red filter square. Dashes in the residues indicate that they were identical to both reference strains. The number above the head of the row indicates the number for the Kresse residues, whereas the number at the end of the row is indicative of the length of each residue row.

Table 2 The comparison of substitution amino acid residues in the VP2 capsid protein

Strains	Substitution of amino acid residues of VP2 capsid protein				
	320[a]	378[b]	383[b]	565[b]	436 [b,c]
NADL-2	I	D	H	R	S
Kresse	I	G	Q	K	P
Thai PPV1s	T (*n* = 7)/I (*n* = 5)	G (*n* = 12)	Q (*n* = 12)	K (*n* = 12)	T (*n* = 12)

a = VP2 capsid protein residues 320 of Thai PPV2s. The properties belonging to residues containing (i) five Thai PPV1s included isoleucine, which was identical to both surrogate strains, while (ii) seven Thai PPV1s were found to contain threonine. This was indicative of differences to both surrogate strains along with a degree of similarity to the reference strains deposited in GenBank.
b = substitutions of the amino acid residues in the VP2 capsid protein used to compare the 12 Thai PPV1s that were observed in their biological characteristics. The outcomes were then compared with those of the Kresse and NADL-2 strains.
c = VP2 capsid protein residues 436 of Thai PPV2s. The amino acid residues in the VP2 capsid protein of Thai PPV2 are identical to those of a known virulent strain of German PPV1.

DISCUSSION

Presently, porcine parvoviruses belonging to the family *Parvoviridae* were found to include PPV1 through PPV7 (Cotmore et al., 2019). Accordingly, PPV1 is known to cause a reproductive system disease, while the pathogenesis of PPV2-PPV7 is unknown. Due to certain biological properties that include the virus's durability in the environment and the relative degree of persistence of infection (Gradil et al., 1990; Eterpi et al., 2009). Hence, reports of the prevalence of PPV1 may be found in numerous geographic areas. This study aimed to examine the prevalence, the genetic diversity of the *VP2* gene, and the residues of the VP2 capsid protein of PPV1 in Thai pig ovaries. In Thailand, there have been two reports of PPV1 via serology and viral genome detection; however, no investigations have been conducted on the genetic diversity of PPV1 in the country. (Tummaruk and Tantilertcharoen, 2012; Saekhow et al., 2015) The findings of our study provide evidence of an organ that was capable of detecting PPV1 that would be applicable to diagnosing the reproductive disease. Moreover, we chose a part of the *VP2* gene because this fragment is an appropriate fragment to demonstrate the distribution of genetic diversity and the substitution of the affected amino acid residues of the biological property residues.

In this study, the prevalence of PPV1 was detected by PCR in 12% of the ovaries of 6 months old pigs (12/100). In a previous study conducted in Chiang Mai, the prevalence of PPV1 in the tonsils of pigs was 53% (42/80), which contrasts with the prevalence reported in this study (Saekhow and Ikeda, 2015). Based on the results of both studies conducted in this area, the sources of organs for virus detection generated a difference in this degree of prevalence. Notably, in the studies using tonsils or blood, high degrees of prevalence were found as follows; 67% (80/120) in Japan (Saekhow et al., 2015), 61% (61/100) in Germany (Streck et al., 2013), and 53% (42/80) in Thailand (Saekhow et al., 2015). However, using of ovarian was found to be low, which included only 12% (6/49) in the study conducted in the USA (Pogranichniy et al., 2008) and in this study as well. There has been a certain amount of controversy over whether the distribution in some organs may have resulted from white blood cell travel or the production of the virus in the organs (McKillen et al., 2007; Miao et al., 2009). It is known that a form of tropism of the parvovirus family

members is likely to infect dividing cells (Joo et al., 1977; Paul et al., 1979; Oraveerakul et al., 1993). We know that there are dividing cells in the ovaries, but the virus has rarely been found in these cells. Contrastively, tonsils are abundant with activated lymphocytes, which are the cells that have been studied as receptors of viruses. However, this likely would have occurred because a viral receptor could not be found. The detection of the PPV1 genome in the follicular fluid of slaughtered pigs occurred because there were no cells in the fluid, which indicated that the virus had been produced in the ovaries (Pogranichniy et al., 2008). It was also found that PPV1 could infect an embryo by attaching itself to the zona pellucida (Wrathall and Mengeling, 1979). Moreover, a viral infection in ovary cells may impair the function of the ovaries in terms of the production of the hormones necessary for ovulation and pregnancy maintenance. Some details, such as those associated with the receptor and cell type, should be further studied to better understand the biological properties of PPV1.

Phylogenetic analysis of the VP2 gene was conducted and the results were divided into two large clades, namely clade I and clade II. By dissecting a member of the PPV1 nucleotide sequences, the members in clade 1 were found to have the same members as the classic group, while the members in clade 2 were determined to be the same as the new group. The results of using the nucleotide sequence fragment that encodes for partial VP2 gene were consistent with studies that used the entire length of the capsid protein sequences that are encoded for VP1 capsid protein (Zimmermann et al., 2006). As mentioned above, in this study, nucleotide sequences of 12 Thai PPV1s were isolated from the pig ovaries. However, it is unclear whether the virus is generated by ovarian cells. Hence, to reconstruct a phylogenetic analysis, 36 references nucleotide sequences from the various source of pigs and contries that were deposited in GenBank were chosen. Based on our results of the phylogenetic analysis, it was revealed that 12 Thai PPV1s belonged to a single cluster, and this cluster was allocated within the classic group. The outcomes of our phylogenetic analysis correlated with the geographic region because at least four countries have reported such a correlation. These countries include Germany, South Korea, China, and Brazil. We found that there was a single cluster of PPV1 evasion in pigs that originated from a few distinct sources, and that there was a relationship between the dominant viral nucleotide sequences and the geographic location in our analysis. The possible explanation for this occurrence is indicative of immunity host selection and viral adaptation. Because, it was determined that the dominant viral strains correlated with the geographical region that had been investigated for the foot and mouth disease virus (FMDV). The selection of FMDV strains has been influenced by several factors such as viral immunity, viral genetic change, and viral adaptation (Haydon et al., 2001; Grenfell et al., 2004). The process that happens due to immune evasion occurs as a result of certain genetic changes. Due to this realization, the virus emerged as the dominant strain in the geographical region (Haydon et al., 2001; Cooke and Westover, 2008; Bachanek-Bankowska et al., 2018). It is conceivable that the detection of a single virus group resulted from a limited number of viral origins contributing to the detection of that virus group. It is also the virus itself that has been altered since the virus tends to evolve in an attempt to avoid detection by the host's immune system.

We also analyzed VP2 by comparing the substitution of VP2 protein residues in 12 Thai PPV1s with two reference strains, namely the Kresse and NADL-2 strains. It has been hypothesized that the VP2 capsid protein of the two surrogate strains could modify certain biological properties or pathogenicity. This determination indicated that there was a six-residue discrepancy in the VP2 amino acid that included 45, 215, 378, 383, 436, and 565 (Bergeron et al., 1996). When three of the residues, namely 378, 383, and 436, on the VP2 capsid protein of the Kresse strain changed, the Kresse strains were observed to reduce their ability to replicate in the originating porcine cells (Fernandes et al., 2011). Moreover, a study conducted on field strains has determined that residue 436 in the virulent strains might affect the pathogenicity of PPV1 in vivo (Zeeuw et al., 2007). This study involved 12 Thai PPV1s, and the Kresse and NADL-2 strains at four residues containing 378, 383, 436, and 565, respectively. A comparative investigation revealed that 378, 383, and 565 residues of twelve Thai PPV2s were identical to those of the Kresse strain. The last residue on PPV1 that affected its biological features, site 436, was determined to be threonine, as has been observed in this study. (Figure 2 and Table 2) Notably, threonine was discovered in 436 residues of the VP2 capsid protein of a proven virulent strain of German PPV1 (Zeeuw et al., 2007). According to the findings of this investigation, the Kresse strain has most certainly become endemic. The degree of occurrence would need to be closely monitored in Thailand, regardless of whether or not the virus produces a form of pathogenicity. Additionally, at residue 320, the five Thai PPV1s were attributed to isoleucine which was comparable to both reference strains, and the seven Thai PPV1s were found to be threonine (Figure 2, Table 2). Not only, our results but also threonine have been reported in other areas such as Brazil, China, and Germany. The resultant 320 residues occurred as a consequence of its epitope location; thus, upon closer examination, the sites identified in this research study were found to be congruent with the previously identified B cell epitopes (Sun et al., 2015; Liu et al., 2020). Thus, amino acid changes of VP2 capsid protein may be the result of immune evasion. An intriguing aspect of amino acid residues is that they impact the biological properties of a virus. Consequently, this should be studied further to determine whether it is related to cell entry or the pathogenicity of PPV1.

CONCLUSION

The outcomes of our study revealed a degree of genetic diversity in the VP2 gene and affected amino acid residues in the VP2 capsid protein of Thai PPV1s in the ovaries of pigs at around six months of age in Chiang Mai and Lamphun Provinces. Accordingly, the characterization of the nucleotide sequence and the substitution of amino acid residues were determined to affect the biological properties of the VP2 capsid protein. Through phylogenetic analysis and a study of certain VP2 capsid proteins, an adaptation of the virus has resulted in strains that have been detected in the Chiang Mai and Lamphun Provinces of Thailand. The residues of amino acid in Thai PPV1s obtained in this investigation were determined to likely have been from the Kresse strain and a proven virulent strain.

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