Association Between Virulence Genes and Serovars, Sequence Types of *Glaesserella (Haemophilus) parasuis* Isolates from the Nasal Cavity of Live Piglets

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**Abstract:** This study analyzed the 19 virulence genes (VGs) of 117 *Glaesserella (Haemophilus) parasuis* isolates from the nasal cavities of live piglets from the south of China and assessed the associations between VGs and serovars, sequence types (STs) of these isolates. The detection rate of 19 VGs ranged from 1.7% to 95.2%, with *vacJ* and *clpP* (95.7%) as the most prevalent. Of the 117 *G. parasuis* isolates, 105 were assigned to ten distinct serovars (1, 2, 4-10 and 15), and twelve of the isolates tested were non-typable (NT). The serovar 10 (17.9%) was the most prevalent. The *G. parasuis* isolates belonging to the same ST and serovar harbored different VGs, and all isolates exhibited considerable genetic heterogeneity. Significant correlations were found between VGs and serovars, different pathogenic serovar groups, and members of clade 2 (based on ST). The results complement epidemiological data of *G. parasuis* and will help the scientific community understand the extreme genetic diversity and pathogenesis of *G. parasuis*, which will aid in the development of *G. parasuis* vaccines.

**Keywords:** *Glaesserella (Haemophilus) parasuis*, Virulence gene, Serovar, Sequence type, Live piglet

**Introduction**

*Glaesserella (Haemophilus) parasuis* (*G. parasuis*), the pathogen that causes Glässer's disease, has brought huge economic losses to the global swine industry [1,2]. *G. parasuis* is a commensal bacterium in the swine upper respiratory tract that contains strains ranging from non-virulent to highly virulent. Virulent strains can invade and cause systemic disease under certain conditions [3-5]. To date, 15 serovars have been identified, in addition to some non-typable (NT) strains [6,7]. Serovar identification of the isolates is the basis for designing vaccination programs [9]. Some earlier studies suggested that *G. parasuis* serovars were virulence markers and could be divided into three pathogenic groups [2]. However, later studies found that isolates allocated into non-pathogenic serovars can also cause disease, and virulence of the isolates allocated to the same serovar can vary greatly [9-11]. Thus, it remains

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**Canlı Domuz Yavrularının Burun Boşluğundan İzole Edilen *Glaesserella (Haemophilus) parasuis*’in Virülans Genleri İle Serovar ve Sekans Tipleri Arasındaki İlişki**

**Öz:** Bu çalışmada, Çin’in güneyinde canlı domuz yavrularının burun boşluklarından elde edilen 117 *Glaesserella (Haemophilus) parasuis* (*G. parasuis*) izolatının 19 virülans geni (VG’ler) analizi edildi ve VG’ler ile serovarlar ve sekans tipleri (ST’ler) arasındaki ilişki değerlendirildi. 19 VG’nin pozitiflik oranları %1,7 ile %95,2 arasında değişmekle birlikte, en yaygın (%95,7) *vacJ* ve *clpP* genleri saptandı. 117 *G. parasuis* izolatının 105’si on farklı serovar (1, 2, 4-10 ve 15) içerisinde yer alırken, test edilen izolatlardan 12’si serotiplendirilemedi (NT). Serovar 10 (%17,9) en yaygın olanıydı. Aynı sekans tipi ve serovara ait olan *G. parasuis* izolatları farklı VG’ler barındırırken, tüm izolatlar önemli ölçüde genetik heterojenite sergiledi. VG’ler ile serovarlar, farklı patojenik serovar grupları ve ST tabanlı monofiletik grup 2 (klad 2) üyeleri arasında önemli korelasyonlara sahipti. Bulgular, *G. parasuis*’in epidemiyojik özelliklerini tamamlamaktadır. *G. parasuis* etkisine karşı karşı aşı geliştirilmesi için birlikte poop olan genetik çeşitliliğinin ve patogenezisinin ayrıntılarına dikkat çekicidir.

**Anahtar sözcükler:** *Glaesserella (Haemophilus) parasuis*, Virülans gen, Serovar, Sekans tipi, Canlı domuz yavrusu

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**How to cite this article?**


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unclear whether sero var can be used as a marker of virulence in \textit{G. parasuis}.

It is generally believed that a single virulence gene (VG) may not be a decisive factor in triggering the pathogenesis of multifactorial diseases such as Glässer’s disease, and the pathogenesis of bacteria often depends on the interaction and expression regulation of many VGs. Thus, a comprehensive analysis of VGs in clinical isolates may be helpful to predict the pathogenicity of novel \textit{G. parasuis} isolates as they are identified. Although the characteristics of \textit{G. parasuis} isolates from clinical cases have been extensively studied, an in-depth analysis of \textit{G. parasuis} isolates from the swine upper respiratory tract has not been performed. In this study, we analyzed the characteristics, including serovars and VGs, of \textit{G. parasuis} isolates from the nasal cavities of live piglets in the south of China. Our results provide more information on the epidemiology and pathogenesis of \textit{G. parasuis}.

\section*{Material and Methods}

\subsection*{Identification and Serotyping}

Nasal swabs were collected from the nasal cavities of live piglets without obvious clinical symptoms of Glässer’s disease between 2007 and 2016 in three provinces (Guangdong, Jiangxi, and Shanghai) in the south of China. Nasal swabs were inoculated on blood agar medium with 0.0025\% of NAD immediately after sampling. Suspect \textit{G. parasuis} colonies were identified by NAD-dependency and 16S rRNA PCR \cite{12}. The isolates underwent molecular serotyping via a multiplex PCR assay described in Howell et al.\cite{13}.

\begin{table} 
\centering 
\begin{tabular}{|l|l|l|l|} 
\hline 
VGs & Primers & Sequence (5’→3’) & Product Size \\
\hline 
hhdA & hhdAF & GGTTTCATGTCACAAACACGCCAACATC & 964 \\
& hhdAR & GATAATTACCCGCTGCTCTCTATGTATC & 557 \\
\hline 
hhdB & hhdBF & ATCTTGCCCTGATTAGAGATGGAG & 563 \\
& hhdBR & GTGAATATAGCCCTTATCCAAATAGGC & 563 \\
\hline 
fluA & fluAF & ATGTTTTGTTGATATGAGATGATA & 406 \\
& fluAR & AACACGGAGGTAGTTGCTTGACT & 406 \\
\hline 
vtA & vta1F & TTTAGGTAAAAGATAAGCAAGGAAATCCC & 380 \\
& vta1R & CCAACACAAAAACCTACCCTCCTCC & 380 \\
\hline 
wbgY & wbgyF & TTGGGCCCTGTCGCCCTATTTTC & 386 \\
& wbgyR & GAAGCCTATCTGTGAATACCGGC & 386 \\
\hline 
\hline 
\hline 
H0254 & H0254F & CAGTTGAAAGTCGTGATGTGGAACC & 415 \\
& H0254R & GGAGGCAGCTTTCAGGTGAG & 415 \\
\hline 
capD & capDF & CGAAGGGAGGTGGTCTCAATCCA & 524 \\
& capDR & GAGGCTCAGTTTGAGCTTTGGC & 524 \\
\hline 
rfaE & rfaEF & GCCAGGGCGAGGTGGTGGATAA & 969 \\
& rfaER & TGGTCGCGTAAATGGAATG & 969 \\
\hline 
legB & legBF & ATGAAATTGATTTATTATTATAGACTCATTCTT & 540 \\
& legBR & CTATAGCACCATGTGAGTACATCTCTC & 540 \\
\hline 
HPM1370 & HPM1370F & ATGCTAAAAAGAGATTTGATGATGATATATT & 520 \\
& HPM1370R & TATATATGTTAATACTACATAC & 520 \\
\hline 
HPM1371 & HPM1371F & ATGAACTCTTACATCTGCGCCCTTCCC & 720 \\
& HPM1371R & ATCCATATAGTACAGTGTGAT & 720 \\
\hline 
HPM1372 & HPM1372F & ATGAAATGTCGCTGTAAATGCGCTG & 462 \\
& HPM1372R & TCGCCCCCATGTGATATGATGAC & 462 \\
\hline 
HPM1373 & HPM1373F & ATGAAATGTCGCTGTAAATGCGCTG & 331 \\
& HPM1373R & TCTCATACACCATCACCACACAC & 331 \\
\hline 
clpP & clpPF & AGAGTGAGGGAGGTGAG & 443 \\
& clpPR & TTGTTGCTCGCAGTGTGTT & 443 \\
\hline 
cheY & cheYF & CCTTATAGGGCGAGTGTCTCC & 377 \\
& cheYR & TCAGGAGCGGCTCAGTATC & 377 \\
\hline 
vacJ & vacJF & ACCGGCCGATGTTGAGAAATG & 377 \\
& vacJR & TAAATGTGACGAGGGGTTG & 377 \\
\hline 
\end{tabular} 
\caption{Primers used to amplify VGs} 
\end{table}
VG Analysis

Nineteen VGs were analyzed using PCR as previously described \cite{14-23}. Details of all primers used are listed in Table 1.

Sequence Types (STs) Analysis

A STs analysis was carried out using the Multi-locus Sequence Typing (MLST) method as previously described \cite{24,25}. A neighbor-joining tree was built using the MEGA version 5.0 software based on the MLST target sequences.

Statistical Analyses

Chi-square and Fisher’s exact tests were used to assess the associations between serovars, ST, and VGs using SPSS version 18.0, and p values lower than 0.05 were considered statistically significant associations.

RESULTS

Identification and Serotyping

A total of 117 *G. parasuis* isolates were obtained from 710 nasal swab samples. Of the 117 *G. parasuis* isolates, 105 were assigned to ten distinct serovars, and twelve of the isolates tested were NT. Serovar 10 (17.9%) was the most prevalent, followed by serovars 15 (14.5%), 6 (12.0%), 8 (11.1%), 4 (8.5%), 9 (7.7%), 1 (7.7%), 7 (6.0%), 5/12 (4.3%), and 2 (0.9%) (Fig. 1-A). Serovars 3, 11, 13, and 14 were not identified. Serovars 4, 6, 15, and NT were observed in all three provinces. However, serovar 2 was observed only in Shanghai and serovar 7 was observed only in Jiangxi (Fig. 1-B).

VG Analysis

The VGs *vacJ* and *clpP* (95.7%) were the most prevalent, followed by *cheY* (93.2%), *rfaE* (92.3%), *hsdR* (91.5%), *capD* (88.9%), *fluA* (40.2%), *vta1* (35.9%), *hhdA* (33.3%), *hhdB* (26.5%), *HPM1372* (22.2%), *nhaC* (21.4%), *isgB* (19.7%), *H0254* (10.3%), *fimB* (10.3%), *wbgY* (7.7%), *HPM1373* (6.8%), *HPM1371* (5.3%), *HPM1370* (1.7%) (Fig. 2). All *G. parasuis* isolates were clustered according to the presence of VGs. Four clusters were obtained (clusters A, B, C, and D) (Fig. 3). Cluster A includes serovars 1, 2, 4, 6, 7, 8, 9, 10, 15, and NT isolates, harboring 4 to 11 VGs; Cluster B includes serovars 4, 5/12, 6, and NT isolates, harboring 9 to 17 VGs; Cluster C includes serovars 1, 7, and 10, harboring 5 to 8 VGs; and Cluster D includes only NT isolates, harboring 0 to 4 VGs. Interestingly, some serovars were distributed in 2 or 3 clusters. For example, serovars 4 and 6 were found in clusters A and B, serovars 1, 7, and 10 were found in clusters A and C, and NT isolates were found in clusters A, B, and D (Fig. 3).

Association Between Serovars and VGs

The distribution of VGs in the isolates allocated to different serovars varied greatly, and a significant correlation was found between serovars and some VGs. A significant
positive correlation was found between the following: serovar 1 and \( vta1 \); serovar 4 and \( hhdb, H0254, nhaC, \) and \( vta1 \); serovar 5/12 and \( fhua, wbg1, hhda, hhdb, lsgb, H0254, nhaC, vta1, \) and \( HPM1373 \); serovar 6 and both \( HPM1371, \) and \( HPM1372 \); serovar 7 and both \( hhda \) and \( vta1 \); serovar 8 and \( hhda, hhdb, \) and \( HPM1371 \); serovar 10 and \( fimB, HPM1371, \) and \( HPM1372 \); serovar 15 and \( hsdR \). However, a significant negative correlation was found between serovar 1 and \( capD \), serovar 4 and \( HPM1371, \) serovar 6 and \( vta1, serovar 8 and vta1, serovar 9 and both \( fhua \) and \( HPM1371, \) and the following: serovar 10 and \( fhua, hhda, hhdb, nhaC, \) and \( vta1 \), serovar 15 and \( hhda, lsgb, nhaC, \) and \( HPM1371, \) and NT and \( tfae, vacj, cheY, clpP, \) and \( hsdR (P<0.05, Table 2). \)
Table 2. Association between serovars and VGs of G. parasuis isolates

<table>
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<th>Serovar</th>
<th>VGs</th>
<th>VG+</th>
<th>VG-</th>
<th>−VG+</th>
<th>−VG−</th>
<th>OR</th>
<th>95% CI</th>
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<td>/</td>
<td>0.000003</td>
</tr>
<tr>
<td></td>
<td>cheY</td>
<td>7</td>
<td>4</td>
<td>102</td>
<td>4</td>
<td>0.07</td>
<td>0.01-0.34</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>clpP</td>
<td>7</td>
<td>4</td>
<td>105</td>
<td>1</td>
<td>0.02</td>
<td>0-0.20</td>
<td>0.000212</td>
</tr>
<tr>
<td></td>
<td>hsdR</td>
<td>7</td>
<td>4</td>
<td>100</td>
<td>6</td>
<td>0.11</td>
<td>0.03-0.48</td>
<td>0.007</td>
</tr>
</tbody>
</table>

VG+: Number of isolates in the corresponding serovar but carrying the VG; VG−: Number of isolates in the corresponding serovar but no carrying the VG; −VG+: Number of isolates no in the corresponding serovar but carrying VG; −VG−: Number of isolates no in the corresponding serovar but no carrying VG.
Oliveira and Pijoan [2] reported that *G. parasuis* was divided into three groups based on different serovars: highly pathogenic serovars (1, 5, 10, 12, 13, and 14), moderately pathogenic serovars (2, 4, and 15), and non-pathogenic serovars (3, 6, 7, 8, 9, and 11). The current study identified a significant correlation between different pathogenic serovar groups and several VGs. The highly pathogenic serovars had a significant positive association with *wbgY*, *fimB*, and 1371, and a significant negative association with *hhdB*. The moderately pathogenic serovars had a significant positive association with *hsdR* and *vta1*, and a significant negative association with *HPM1371*. The non-pathogenic serovars had a significant negative association with *H0254*, *fimB*, and *vta1* (P<0.05, Table 3).

### Table 3. Association between pathogenic serovar group and VGs of *G. parasuis* isolates

<table>
<thead>
<tr>
<th>Pathogenic Serovar Group</th>
<th>VGs</th>
<th>VG +</th>
<th>VG−</th>
<th>−VG +</th>
<th>−VG−</th>
<th>OR 95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highly pathogenic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>wbgY</em></td>
<td>7</td>
<td>28</td>
<td>1</td>
<td>70</td>
<td>17.5</td>
<td>2.06-148.84</td>
<td>0.002</td>
</tr>
<tr>
<td><em>hhdB</em></td>
<td>5</td>
<td>30</td>
<td>25</td>
<td>46</td>
<td>0.31</td>
<td>0.11-0.90</td>
<td>0.038</td>
</tr>
<tr>
<td><em>fimB</em></td>
<td>9</td>
<td>26</td>
<td>3</td>
<td>68</td>
<td>7.85</td>
<td>1.97-31.28</td>
<td>0.002</td>
</tr>
<tr>
<td><em>HPM 1371</em></td>
<td>27</td>
<td>8</td>
<td>32</td>
<td>39</td>
<td>4.11</td>
<td>1.64-10.28</td>
<td>0.002</td>
</tr>
<tr>
<td><em>HPM 1373</em></td>
<td>6</td>
<td>29</td>
<td>1</td>
<td>70</td>
<td>14.48</td>
<td>1.67-125.66</td>
<td>0.005</td>
</tr>
<tr>
<td>Moderately pathogenic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>hsdR</em></td>
<td>22</td>
<td>6</td>
<td>78</td>
<td>0</td>
<td>0</td>
<td>/</td>
<td>0.0002</td>
</tr>
<tr>
<td><em>vta1</em></td>
<td>15</td>
<td>13</td>
<td>22</td>
<td>56</td>
<td>2.94</td>
<td>1.21-7.17</td>
<td>0.021</td>
</tr>
<tr>
<td><em>HPM 1371</em></td>
<td>4</td>
<td>24</td>
<td>55</td>
<td>23</td>
<td>0.07</td>
<td>0.02-0.22</td>
<td>0.000</td>
</tr>
<tr>
<td>Non-pathogenic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>H0254</em></td>
<td>1</td>
<td>42</td>
<td>10</td>
<td>53</td>
<td>0.13</td>
<td>0.02-1.06</td>
<td>0.026</td>
</tr>
<tr>
<td><em>fimB</em></td>
<td>0</td>
<td>43</td>
<td>12</td>
<td>51</td>
<td>0</td>
<td>/</td>
<td>0.001</td>
</tr>
<tr>
<td><em>vta1</em></td>
<td>9</td>
<td>34</td>
<td>28</td>
<td>35</td>
<td>0.33</td>
<td>0.14-0.80</td>
<td>0.014</td>
</tr>
</tbody>
</table>

*VG +*: Number of isolates in the corresponding serovar but carrying the VG. *VG−*: Number of isolates in the corresponding serovar but no carrying the VG. −VG +: Number of isolates no in the corresponding serovar but carrying VG. −VG −: Number of isolates no in the corresponding serovar but no carrying VG.

The ST analysis revealed two major clades (clade 1 and clade 2) based on the MLST target sequences of 43 *G. parasuis* isolates. Clade 1 includes 37 isolates of serovars 1, 2, 4, 6, 7, 8, 9, 10, 15, and NT, harboring 1 to 11 VGs each.

**Fig 4.** Neighbour-joining tree based on the MLST target sequences of 43 *G. parasuis* isolates.
During the current study, we did not isolate any isolates representing serovars 11 and 14, and we only isolated a single strain from serovar 2. This suggests that serovars of *G. parasuis* from the swine nasal cavity exhibit a complex regional distribution across provinces in China. In both the current study and the study conducted by Zhang et al., the detection frequency of serovars 4 and 5 was relatively low. Strains in serovars 4 and 5 are widely regarded as pathogenic strains, and they are most often identified from pigs with Glasser’s disease. Although the detection frequency of serovars 4 and 5 was not high in live piglets, these isolates may nonetheless cause disease when an animal is under stress. Of note, the dominant serovars identified in this study, serovar 10 and serovar 15, were previously considered to be highly and moderately pathogenic, respectively. These two serovars have rarely been isolated in diseased pigs in China. Further attention and research are required to determine whether the presence of strains from serovars 10 and 15 in the respiratory tract of live piglets would cause localized disease, or even a potential disease epidemic.

In this study, all *G. parasuis* isolates were divided into four clusters according to the presence of VGs. Though serovars 2, 5, 8, 9, 10, and 15 were only distributed in one cluster, isolates belonging to the same serovar harbored different VGs. These differences were also present among strains that belonged to the same ST and serovar. For example, strains SG25 and N1-24, isolated from different farms, were both allocated to ST185 and serovar 8, and possessed seven identical VGs. However, strain SG25 had five more VGs than N1-24. Similarly, strains OY2 and QY6-1, isolated from the same farm, were allocated to ST255 and serovar 15, but strain QY6-1 has one more VG (*rfαE*) than OY2. Interestingly, strain QY6, isolated from the nasal cavity of the same piglet as strain QY6-1, also harbored *rfαE*. These results suggest that *G. parasuis* isolates may undergo multiple gene exchanges while coexisting in the respiratory tract. The VGs of isolates allocated to the same ST and serovar varied greatly, which may lead to differences in the pathogenicity and immunogenicity of strains belonging to the same ST and serovar. Once these strains invade the host tissues and organs, they may cause localized disease and eventually become epidemics. At that point, even if the serovars of commercially available vaccines and pathogenic strains were the same, the differences in VGs may lead to immune failures. That scenario would pose a substantial challenge to the development of a new vaccine.

Van et al. reported that the detection frequency of the VGs *vta1*, *hsdR*, *fimB*, *nhaC*, *fhuA*, *capD*, *wbgY*, and *H0254* was 92.5%, 47.9%, 37.2%, 38.3%, and 13.7%, respectively. Boerlin et al. reported that the detection frequency of the VGs *vta1*, *hsdR*, *fimB*, *nhaC*, *fimB*, *capD*, *wbgY*, and *H0254* was 62.5%, 35.7%, 30.3%, 12.5%, 8.9%, 8.9%, and 0%, respectively. Boerlin et al. reported that the detection frequency of the VGs *vta1*, *hsdR*, *fimB*, *nhaC*, *fhuA*, *capD*, *wbgY*, and *H0254* was 92.5%, 47.9%, 37.2%, 38.3%,

---

### Table 4. Association between MLST clade and VGs of G. parasuis isolates

<table>
<thead>
<tr>
<th>VG</th>
<th>Clade1+</th>
<th>Clade1−</th>
<th>Clade2+</th>
<th>Clade2−</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>vta1</em></td>
<td>13</td>
<td>24</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>/</td>
<td>0.004</td>
</tr>
<tr>
<td><em>nhaC</em></td>
<td>8</td>
<td>29</td>
<td>5</td>
<td>1</td>
<td>0.06</td>
<td>0.01-0.59</td>
<td>0.007</td>
</tr>
<tr>
<td><em>hhdA</em></td>
<td>8</td>
<td>29</td>
<td>5</td>
<td>1</td>
<td>0.06</td>
<td>0.01-0.59</td>
<td>0.007</td>
</tr>
<tr>
<td><em>hhdB</em></td>
<td>7</td>
<td>30</td>
<td>5</td>
<td>1</td>
<td>0.05</td>
<td>0.01-0.50</td>
<td>0.004</td>
</tr>
<tr>
<td><em>lsgB</em></td>
<td>5</td>
<td>32</td>
<td>4</td>
<td>2</td>
<td>0.08</td>
<td>0.01-0.56</td>
<td>0.01</td>
</tr>
<tr>
<td><em>H0254</em></td>
<td>0</td>
<td>37</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>/</td>
<td>0.000006</td>
</tr>
<tr>
<td><em>wbgY</em></td>
<td>1</td>
<td>36</td>
<td>4</td>
<td>2</td>
<td>0.01</td>
<td>0-0.14</td>
<td>0.001</td>
</tr>
<tr>
<td><em>fimB</em></td>
<td>1</td>
<td>36</td>
<td>3</td>
<td>3</td>
<td>0.03</td>
<td>0-0.38</td>
<td>0.006</td>
</tr>
<tr>
<td><em>HPM 1373</em></td>
<td>1</td>
<td>36</td>
<td>3</td>
<td>3</td>
<td>0.03</td>
<td>0-0.38</td>
<td>0.006</td>
</tr>
</tbody>
</table>

+: Number of isolates in the corresponding clade but carrying the VG; −: Number of isolates in the corresponding clade but no carrying the VG.
Olvera et al.\(^{[16]}\) reported that isolates without vta\(^{A1}\) are generally avirulent. In this study, the presence of vta\(^{A1}\) was associated with a significantly decreased probability of membership in the non-pathogenic serovar group. This indicates that isolates allocated to the non-pathogenic serovar group were positive for lsgB, 16 were positive for fhuA, and 1 was positive for HPM-1372, indicating that isolates allocated to the non-pathogenic serovar group were positive for lsgB, 16 were positive for fhuA, and 1 was positive for HPM-1372. Our results indicate that the distribution of VGs in G. parasuis is diverse and complex.

38.3%, 23.4%, 22.3%, and 17%, respectively; Turni et al.\(^{[32]}\) reported that the detection frequency of hhdA and hhdB was 36% and 13.3%, respectively, which differs from our results for most of the above VGs. Although previous studies\(^{[31]}\) have shown that the VGs lsgB, fhuA, capD, HPM-1372, and HPM-1373 were not observed in any isolates from non-pathogenic serovar group, our results showed that 8 of 43 isolates from the non-pathogenic serovar group were positive for lsgB, 16 were positive for fhuA, and 39 were positive for capD. These results are consistent with previous studies\(^{[2]}\) on the detection frequency of these VGs. The development of a SYBR green I multiple real-time fluorescence PCR assay for detection of Glaesserella (Haemophilus) parasuis and Pasteurella multocida. Kafkas Univ Vet Fak Derg, 28 (1): 11-17, 2002. DOI: 10.9775/kvd.2001.26302

The authors declare that data supporting the findings of this study are available upon request.

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Conflict of Interest

The authors declare no conflict of interest.

Authors’ Contributions

LP and YXY conceived the experiments and wrote the paper. All authors performed the experiments. All authors have interpreted the data, revised the manuscript, and approved the final version.

References


Research Article


