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Genomic characterization of *Burkholderia glumae* K6 and *B. gladioli* UPMBG7: causal agents of bacterial panicle blight in Malaysia

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Abstract Bacterial panicle blight (BPB), caused by *Burkholderia glumae* and *B. gladioli*, is a major threat to rice in Malaysia, with potential yield losses up to 75%. In June 2021, BPB symptoms were detected in Kedah rice fields. Isolates were identified through phenotypic traits, pathogenicity tests, and molecular analysis (16S rRNA and *gyrB* sequencing). Whole-genome sequencing of *B. glumae* K6 and *B. gladioli* UPMBG7 was performed using the Illumina NovaSeq 6000 platform. The assembled draft genome of *B. glumae* K6 contained 210 contigs, with a total genome size of 6.57 Mbp, 68.33% G+C content, and 5,641 coding sequences (CDS). It harbored toxoflavin biosynthesis genes (*toxABCDE, toxJ*) and a Type III secretion system (T3SS), contributing to its pathogenicity. *B. gladioli* UPMBG7 contained 124 contigs, with a total genome size of 8.22 Mbp, G+C content of 67.99, and 7,022 coding proteins. *B. gladioli* lacked the *toxI* gene for toxoflavin production but compensated with pyoverdine siderophore genes (*pvdA*), which facilitate iron acquisition. These findings highlight divergent infection mechanisms, with

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B. glumae relying on toxoflavin-mediated virulence and *B. gladioli* on resource competition. The genomic insights support the development of resistant rice cultivars, biological controls, and molecular diagnostics to improve BPB management in Malaysia.

Keywords Bacterial panicle blight disease · *Burkholderia glumae* · *Burkholderia gladioli* · Whole-genome sequencing · Rice · Illumina NovaSeq 6000

Introduction

Burkholderia glumae, the causal agent of BPB disease, was initially identified in Japan in the early 1950s as a rice pathogen that caused seedling blight on rice (Nandakumar et al., 2009). The disease is primarily spread to many rice-growing regions through contaminated seeds, wind-driven rain, and irrigation water. In Malaysia, BPB outbreaks have become a serious challenge, with yield losses reaching 75% in severely affected fields. The first confirmed outbreak in Malaysia occurred in two distinct states, Penang and Kelantan. This outbreak spanned from December 2017 to March 2018 (Ramachandran et al., 2021). BPB symptoms include seedling blight, sheath rot, and panicle sterility. Distinctive signs are upright, straw-coloured panicles with florets showing dark borders, while the rachis remains green. These symptoms contribute to significant grain loss and poor crop quality (Nandakumar et al., 2009).

Previous genomic studies on *B. glumae* have identified key virulence determinants, including toxoflavin biosynthesis genes and quorum-sensing regulators crucial for pathogenicity (Kang et al., 2008; Kim et al., 2004). Research on *B. gladioli* highlighted its genomic diversity and adaptability, enabling it as a plant pathogen and an opportunistic human pathogen (Pérez-Mendoza et al., 2014; Pritchard et al., 2020). However, comparative genomic analyses between these two pathogens, particularly in the context of BPB, remain scarce. The lack of in-depth comparative studies limits our understanding of their distinct pathogenic strategies and their implications for disease management or further research to develop targeted control measures.

This study provides a comparative genomic analysis of *B. glumae* K6 and *B. gladioli* UPMBG7, two major BPB pathogens in Malaysian rice fields. By identifying key virulence factors, including toxoflavin biosynthesis (*toxABCDE, toxJ*) and Type III secretion system (T3SS) in *B. glumae* and pyoverdine siderophores (*pvdA*) in *B. gladioli*, this research enhances our understanding of their infection strategies and potential control targets.

Materials and methods

Samples collection and isolation of bacterial pathogen

Symptomatic rice panicles were collected from BPB outbreak sites in Kedah, Perak, and Selangor. Sampling was conducted from June 2021 to January 2022 based on BPB outbreak reports from the Department of Agriculture (DOA) Malaysia. Collected rice panicles and sheaths were excised, surface sterilized, and inoculated onto King's B agar for 24–48 h at 41 °C. Colonies showing typical *Burkholderia* morphology were purified and stored in 20% (v/v) glycerol stock at −70 °C.

Molecular characterization, sequence and phylogenetic analysis

Genomic DNA extraction from bacterial cultures was carried out using the Presto™ Mini gDNA Bacteria Kit according to the manufacturer's instructions (Geneaid Biotech Ltd., Taiwan). Subsequently, PCR amplification was conducted to identify *Burkholderia* using universal and specific genes: 16 s Ribosomal RNA (16 s rRNA), DNA gyrase subunit B of *B. glumae* (*gyrB*), and *gyrB* gene of *B. gladioli* (Mulaw et al., 2018; Sayler et al., 2006). For the amplification of the 16 s RNA gene, the expected size of the resulting amplicons was ~ 1500 base pairs. The expected size of the amplicon for *B. glumae gyrB* gene was ~ 530 base pair. Lastly, the expected size of the amplicon for *B. gladioli gyrB* gene was ~ 479 bp (Mulaw et al., 2018).

Amplicons were sequenced using the Sanger method, assembled using Bioedit 7.2.5 software, aligned with NCBI BLASTn, and used to construct phylogenetic trees (MEGA v11, Maximum Likelihood, 1,000 bootstraps). All sequences were submitted to the GenBank database.

Pathogenicity tests

Rice seedlings of MR219 were planted in the glasshouse with regulated temperatures (day: 35 °C, night: 25 °C) and humidity (70–100%) conditions. Rice was then inoculated with 1 mL of 10^8 CFU ml^{-1} bacterial suspension from ten bacterial suspensions of *B. glumae* and two bacterial strains of *B. gladioli* isolates into the panicles and crowns of 75-day-old rice seedlings using a sterile syringe on ten biological replicates per treatment. Control rice seedlings inoculated with sterilized water. Disease progression was monitored for 28 days after inoculation (DAI), with severity recorded every 24 h. (Nandakumar et al., 2009; Lalithiya et al., 2017). Statistical analysis was performed using one-way analysis of variance (ANOVA) to determine significant differences among 12 *Burkholderia* isolates inoculated on rice cultivar MR219. A randomized complete block design (RCBD) was used with three blocks. Post-hoc multiple comparisons were performed using Tukey's Honestly Significant Difference (HSD) test at a significance level of $p < 0.05$. The model included both treatment and block effects. Disease severity percentages were used to construct the Area Under the Disease Progress Curve (AUDPC) to quantify disease progression over time, proposed by Shanner and Finney (1977). All statistical analysis data were analyzed using Statistical Analysis Software (SAS) (version 9.4).

Library preparation, quality control and whole-genome sequencing

Genomic DNA was extracted from *B. glumae* K6 and *B. gladioli* UPMBG7. A DNA library was prepared using the Illumina TruSeq® kit, following the method of Pasquali et al. (2019). Pooled libraries with different indices underwent high-throughput sequencing on the Illumina NovaSeq 6000 platform, which uses sequencing-by-synthesis technology, generating paired-end reads (2 × 150 bp). FastQC software V0.20.0 was used for quality control.

Genome assembly and gene function analysis

Genome assembly was modified through three programs (Abbas et al., 2014). Initial assembly with SOAPdenovo v2.04, SPAdes v3.15.4, and AbySS v2.1.5, along with CISA software, was employed to integrate the assembly results. Genome annotations were performed using the NCBI Prokaryotic Genome Annotation Pipeline (PGAP), including transfer and ribosomal RNAs. Quorum Sensing (QS) and Flagellar pathways of the annotated gene via Kyoto Encyclopedia of Genes and Genomes (KEGG) databases. (Ashburner et al., 2000; Galperin et al., 2015; Kanehisa et al., 2004, 2006; Li et al., 2002). KEGG annotation was used to map genes to metabolic pathways and virulence-related systems, providing insights into the functional roles of key genes. NCBI non-redundant protein database was annotated based on the protein database for species classification.

Results and discussion

Samples collection and isolation of bacterial pathogen

BPB symptoms included grain rotting, seedling blight, and florets with dark lesions at the base. Infected panicles often remained upright, exhibiting sheath lesions with necrotic centers and reddish-brown borders, as well as small tan leaf or spikelet spots (1–5 mm) with brown margins (Fig. 1). A total of 22 *B. glumae* and 5 *B. gladioli* isolates were recovered. Colonies were cream with yellow pigmentation. *B. glumae* and *B. gladioli* both produce yellow pigmentation on King's B agar, but the underlying mechanisms and implications of this pigmentation differ between the two species. *B. glumae* produces yellow pigmentation due to synthesizing the phytotoxin toxoflavin, which contributes to plant cell damage and is associated with the bacterium's virulence, allowing it to effectively establish infections in its host (Lehman et al., 2015; Tsukuda et al., 2018). In contrast, yellow pigmentation in *B. gladioli* is the production of pyomelanin, a secondary metabolite that copes with environmental stresses by protecting against oxidative damage and aiding in iron acquisition (Ma et al., 2016; Pritchard et al., 2020).

Molecular characterization, sequence and phylogenetic analysis

PCR amplification of 27 isolates using 16S rRNA, *B. glumae gyrB* and *B. gladioli gyrB* primer pair showed around 1500 bp, 530 bp, and 479 bp

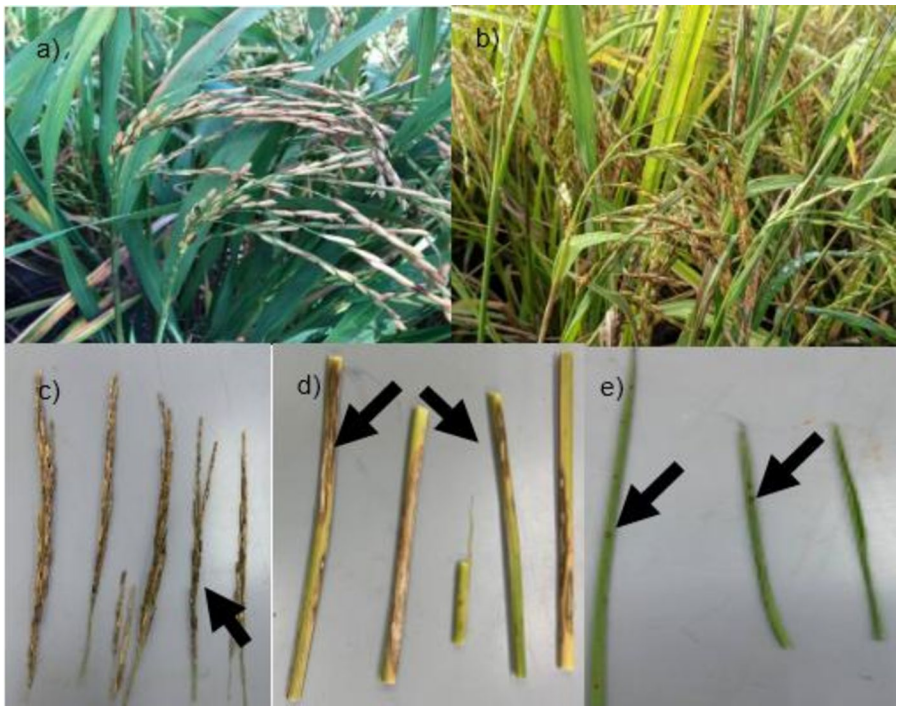

Fig. 1 Symptoms of Panicle Blight on Rice Field. (**a**) and (**b**) Rice crop in Sekinchan and Yan showed BPB disease outbreak; (**c**) Panicles containing floret with dark gray and a reddish-brown lesion across the floret; (**d**) Leaf sheath has a lesion several centimeter long with gray necrosis; (**e**) Leaves contain small circular tan lesion with brown border

amplicon, respectively (Fig. 2). BLASTn search for all sequenced primers has similarity ranging from 96–100%. Phylogenetic analysis of 16S and *gyrB* gene clustered isolates with reference strains, confirming species-level identification. The tree with the greatest log-likelihood is displayed, with the bootstrap support percentages marked at the nodes. The scale bar represents the expected number of changes per site. Based on the results, identifying *Burkholderia* species using the *gyrB* gene is significantly more efficient than using the 16S rRNA gene sequence, as the phylogenetic analysis revealed a higher genetic variation in *gyrB*, with a nucleotide base substitution of 0.1, compared to 16S rRNA, with a nucleotide base substitution of 0.02. The acquired 16 s rRNA and *gyrb* gene sequences have been submitted to NCBI GenBank (Table 1). The phylogenetic analysis of *B. glumae* and *B. gladioli* provides key insights into their evolutionary adaptations to rice as a host. The maximum-likelihood tree constructed using 16S rRNA and *gyrB* sequences revealed that *B. glumae* forms a distinct monophyletic cluster closely related to other rice-pathogenic *Burkholderia* species, while *B. gladioli* remain more phylogenetically diverse, clustering with both plant-associated and opportunistic strains (Sayler et al., 2006).

Pathogenicity tests

After 4–7 days of inoculation, MR219 cultivars began to develop bacterial panicle blight, whereas the control rice remained healthy. Up to 28 days after inoculation, the symptoms were evaluated and documented. The symptomatic plants first appeared when florets became brown across the floret base and gradually developed into classic panicle blight, characterized by florets with a completely dark brown base and sterile florets. After 28DAI, all plants developed BPB disease, with floret blight expanding consistently from the bottom to the top of the florets. The disease incidence ranges from 27 to 77% due to severe panicle loss and severity progress.

A one-way ANOVA revealed statistically significant differences in disease severity among the 12 *Burkholderia* isolates tested on rice cultivar MR219 ($F_{11,22} = 15.00$, $p < 0.0001$; $R^2 = 0.888$). A significant block effect was also observed ($F_{2,22} = 4.64$, $p = 0.0209$), indicating variability across replicates. Post-hoc Tukey's HSD test grouped the isolates into four significance categories (A–D). *B. gladioli* isolate UPMBG7 exhibited the highest disease severity (77.43%, $p < 0.05$) and the highest AUDPC value (1123.58), followed by *B. glumae* isolate BGS.AS1

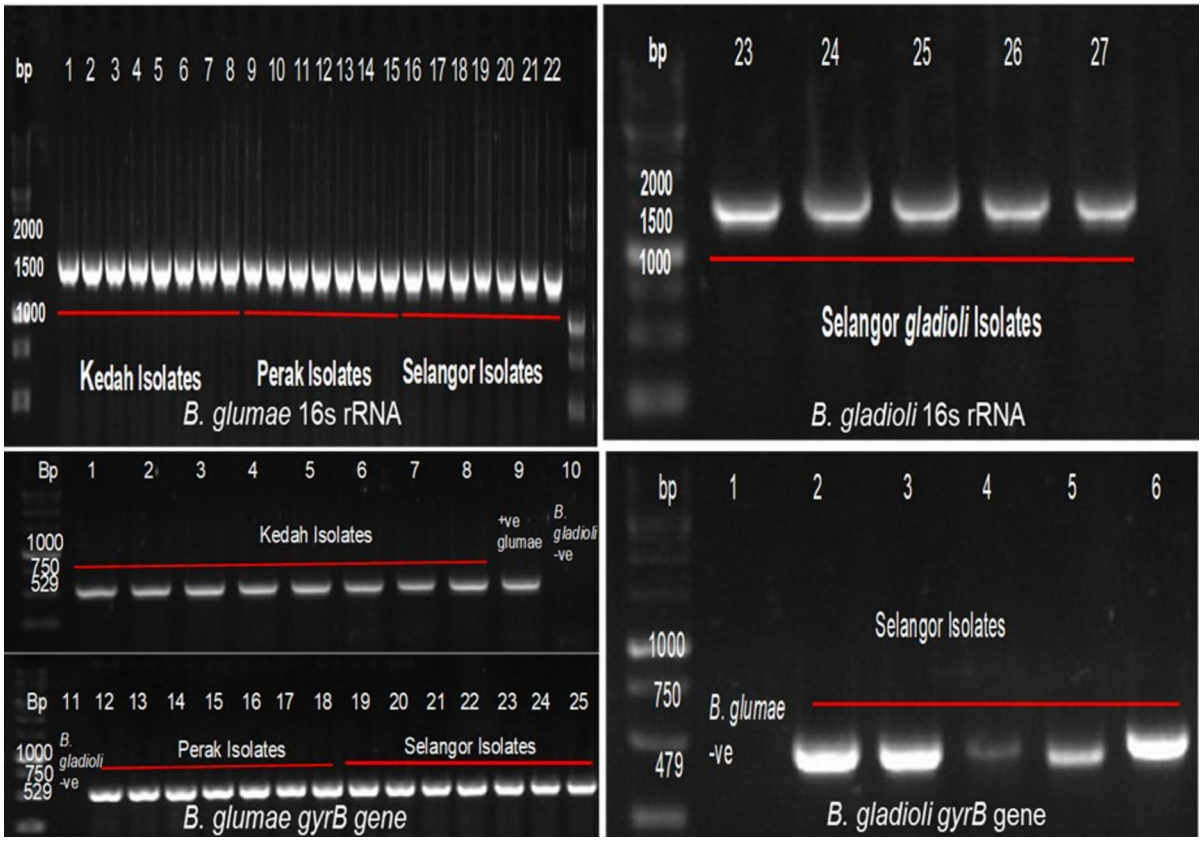

Fig. 2 PCR amplification of 16 s rRNA and *gyrB* regions from *B. glumae* and *B. gladioli*

(68.15%, AUDPC = 948.28). In contrast, *B. glumae* isolate BGK.AZ1 showed the lowest disease severity (27.60%) and the lowest AUDPC value (410.76). These results confirm substantial pathogenic variation among the isolates, with *B. gladioli* strains demonstrating significantly higher virulence than most *B. glumae* strains (Table 2).

Fig. 3 and Fig. 4 show that disease severity increased steadily at 7DAI until 28DAI for all isolates. Within the context of our research, the interpretation of the Area Under the Disease Progress Curve (AUDPC) values presents a nuanced understanding of disease progression and the responses of different cultivars to the pathogen. Our findings reveal a spectrum of disease severity, as reflected by the range of AUDPC values from 410.762 to 1123.575. The AUDPC values were compared across different *Burkholderia* strains to evaluate their virulence levels in rice cultivar MR219. The strain with the lowest AUDPC value of 410.762 demonstrates the least disease progression, suggesting a reduced susceptibility to the host. Conversely, the strain with the highest AUDPC value of 1123.575 exhibits the most substantial disease progression, indicating greater susceptibility. The range of AUDPC values from the studies suggests varying degrees of resistance or susceptibility to the disease, with the cultivars having an AUDPC of 410.762, indicating partial resistance toward *B. glumae*, while those with higher values are generally less resistant. From the disease severity index analysis, our studies find that *B. gladioli* have more severe virulence than *B. glumae* strain. However, in our study, *B. glumae* is more prevalent than *B. gladioli* in the BPB disease outbreak. The results suggest that the interactions between these pathogens and their host plants, as well as the environmental conditions, may influence their virulence outcomes.

Table 1 List of Genbank accession numbers of *Burkholderia* in this study

Strains	State	Country	Host	Species	16 s rRNA Accession	*gyrB* Accession
BGK.AZ1	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OK632514	OL461777
BGK.AZ2	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OK632515	OL461778
BGK.AZ3	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OK632516	OL461779
BGK.AZ4	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OK631741	OL461780
BGK.AZ5	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OL347392	OL461781
BGK.AZ6	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OL347393	OL461782
BGK.AZ7	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OL347394	OL461783
BGK.AZ8	Kedah	Malaysia	*Oryza sativa*	*Burkholderia glumae*	OL347395	OL461784
BGS.AS1	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062124	ON086704
BGS.AS2	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062125	ON086705
BGS.AS3	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062126	ON086706
BGS.AS4	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062127	ON086707
BGS.AS5	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062128	ON086708
BGS.AS6	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062129	ON086709
BGS.AS7	Selangor	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062130	ON086710
BGP.A1	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062131	ON086711
BGP.A2	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062132	ON086712
BGP.A3	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062133	ON086713
BGP.A4	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062134	ON086714
BGP.A5	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062135	ON086715
BGP.A6	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062136	ON086716
BGP.A7	Perak	Malaysia	*Oryza sativa*	*Burkholderia glumae*	ON062137	ON086717
UPMBG7	Selangor	Malaysia	*Oryza sativa*	*Burkholderia gladioli*	OM869953	OM824438
UPMBG8	Selangor	Malaysia	*Oryza sativa*	*Burkholderia gladioli*	OM869954	OM824439
UPMBG9	Selangor	Malaysia	*Oryza sativa*	*Burkholderia gladioli*	OM869955	OM824440
UPMBG15	Selangor	Malaysia	*Oryza sativa*	*Burkholderia gladioli*	OM869956	OM824441
UPMBG17	Selangor	Malaysia	*Oryza sativa*	*Burkholderia gladioli*	OM869957	OM824442

Table 2 Percentage of disease severity and Area Under Disease Progress Curve (AUDPC) on MR219 cultivars for 12 selected isolates *Burkholderia* species on panicles

Variety	Strain	AUDPC value	Percent Disease Severity (%) after 28 days inoculation (28DAI ± SD)	Disease score at 28DAI	Isolate Virulence level
MR219	BGK.AZ1	410.76	27.60 ± 3.98^{D}	2	Low
	BGS.AS3	658.15	47.35 ± 6.25^{C}	3	Intermediate
	BGK.AZ4	666.48	49.19 ± 7.56^{C}	3	Intermediate
	BGP.A2	790.44	49.82 ± 3.06^{BC}	3	Intermediate
	BGS.AS5	779.77	61.01 ± 3.27^{ABC}	4	High
	BGP.A4	927.79	61.28 ± 3.45^{ABC}	4	High
	BGS.AS4	804.10	63.81 ± 5.70^{ABC}	4	High
	BGP.A6	1053.75	63.94 ± 3.23^{ABC}	4	High
	BGK.AZ7	761.79	67.81 ± 4.86^{AB}	4	High
	BGS.AS1	948.28	68.15 ± 7.61^{A}	4	High
	UPMBG8	1081.73	72.36 ± 3.42^{A}	4	High
	UPMBG7	1123.58	77.43 ± 4.87^{A}	4	High

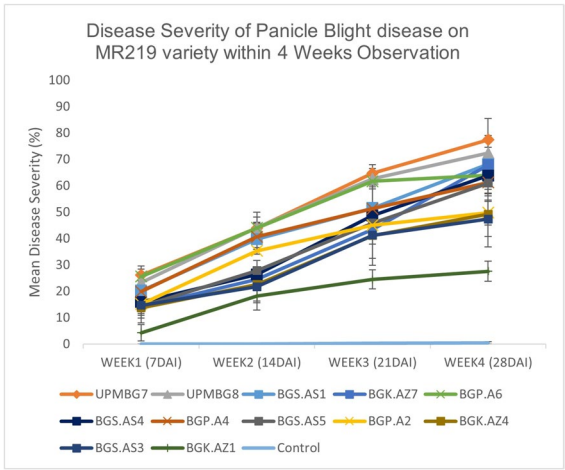

Fig. 3 The percentage of disease severity on MR219 cultivar after 28DAI

Genome assembly

Whole-genome sequencing of *B. glumae* K6 and *B. gladioli* UPMBG7 was performed using the Illumina NovaSeq 6000 platform, generating paired-end reads (2 × 150 bp). The draft genome sequences of K6 had a total length of 6.57 Mbp, with 210 contigs, an N50 value of 77,057 bp, and 68.33% G + C contents. The average read depth of the genome is 205.0X. The completeness of the *B. glumae* K6 genome assembly was assessed using BUSCO v5.8.3. The results indicate a highly complete genome, with 99.5% of the expected single-copy orthologs identified. The genome contained 6,016 genes with 5,947 coding sequences, eight rRNA gene operons, and 57 tRNA genes. *B. gladioli* UPMBG7 had a total length of 8.22 Mbp, with 124 contigs, with an N50 value of 207,968 bp and 67.99% G + C contents. The average read depth of the genome is 85.0X. UPMBG7 genome contained 7,237 genes with 7,164 coding sequences, 8 rRNA operons, and 60 tRNA genes. The completeness of the *B. gladioli* UPMBG7 genome assembly was evaluated, and the results indicate a 100.0% completeness score. Our analysis reveals a close match between our *B. glumae* strain and the reference sequences in terms of both genome size and GC content.

Gene annotation and functional analysis

Species identification was carried out using gene sequences through protein sequence analysis with BLAST v2.15.0. The Non-Redundant Protein Database (NR) annotation of species and genes demonstrated that 4,988 genes (92.03%) annotated from the K6 strain exhibit similarity to *B. glumae* species. Meanwhile, the NR gene annotated for the UPMBG7

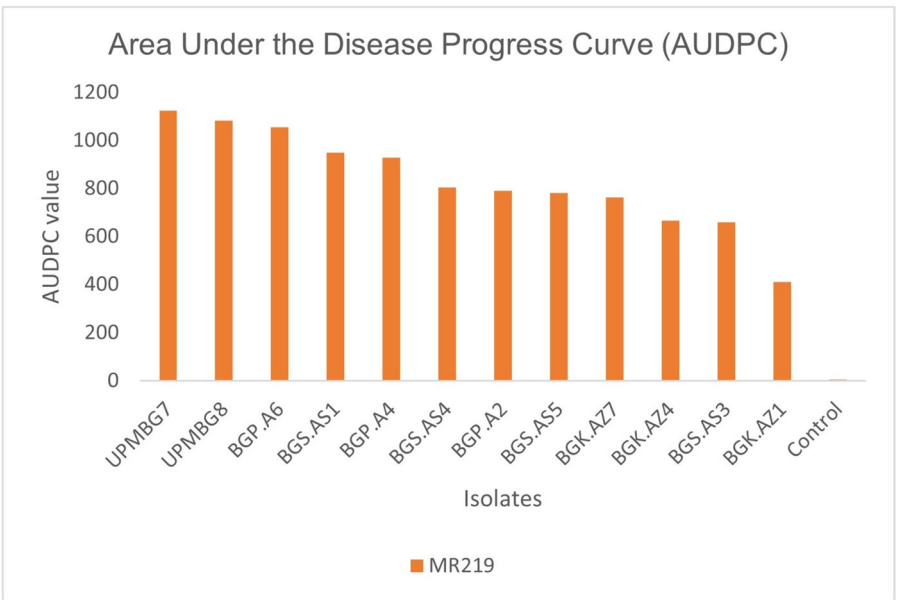

Fig. 4 Area Under the Disease Progress Curve (AUDPC) of 12 *Burkholderia* isolates inoculated onto MR219 cultivar

strain showed 5,700 genes (84.36%) annotated from the UPMBG7 strain, which are comparable to those of the *B. gladioli* species.

Ecological significance of genomic differences

The genomic differences observed between *B. glumae* and *B. gladioli* have significant ecological implications. The larger genome size of *B. gladioli*, coupled with the presence of additional genes related to stress tolerance, suggests that this strain may have enhanced environmental resilience compared to *B. glumae*. This could explain why *B. gladioli* exhibits more severe virulence when isolated in rice in a controlled environment. However, despite its severe virulence, *B. glumae* appears more prevalent in field outbreaks, potentially due to its specific virulence factors, such as toxoflavin production, which facilitate infection in natural field conditions. These findings highlight the importance of understanding pathogen-host-environment interactions to develop effective disease management strategies.

KEGG pathway analysis using a hypergeometric distribution with a q-value threshold of < 0.05 showed that *B. gladioli* and *B. glumae* possess genes required for flagellar assembly, which is essential for their motility and ability to colonize host plants (Fig. 5). *B. gladioli* lacks the *toxI* gene, essential for producing the virulence factor toxoflavin present in *B. glumae* (Lehman et al., 2015). Consequently, while *B. gladioli* can effectively move and colonize plants; however, it does not produce toxoflavin, which limits its ability to cause host tissue damage compared to *B. glumae*. This

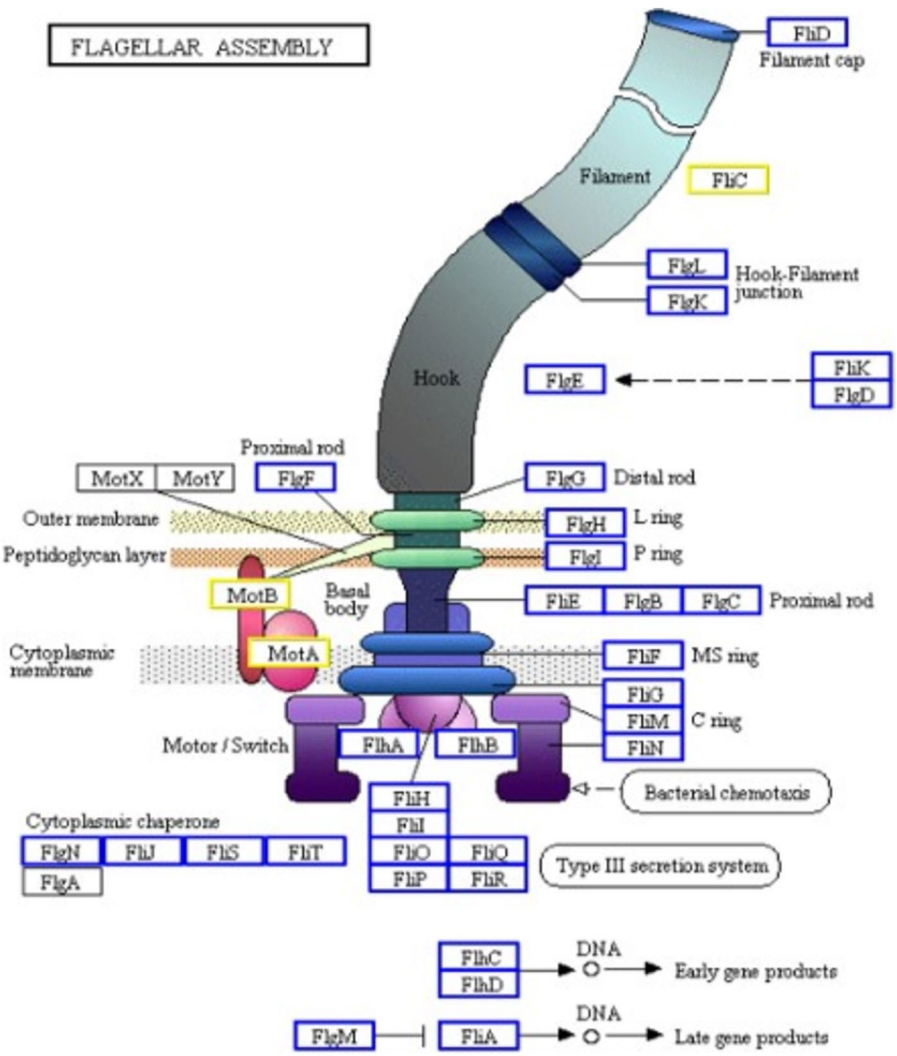

Fig. 5 KEGG pathway for flagellar assembly

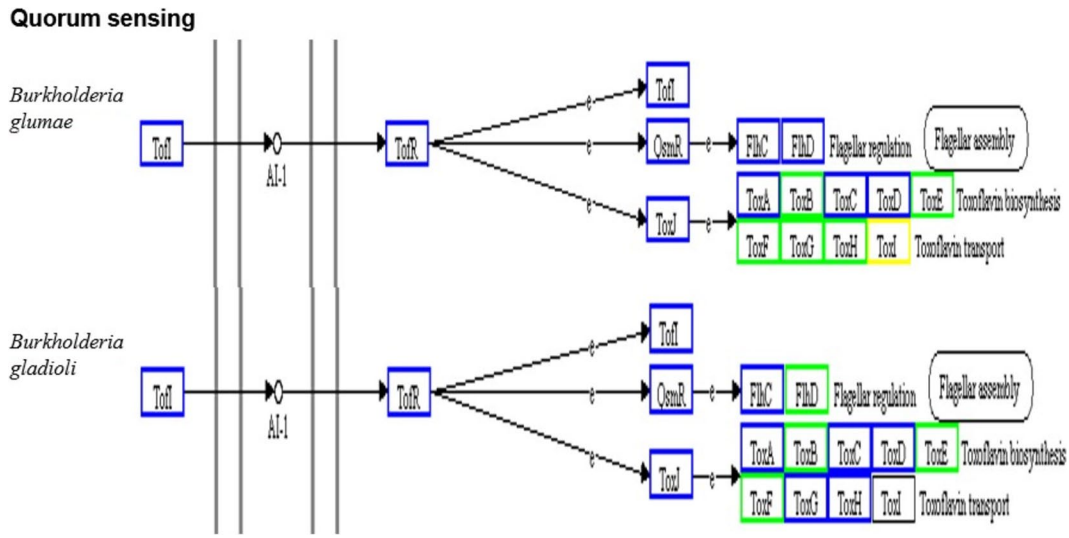

Fig. 6 The proposed KEGG pathway for the quorum sensing system of *B. glumae* K6 and *B. gladioli* UPMBG7. Highlighted boxes indicate the gene produced by the *B. glumae* K6 and *B. gladioli* UPMBG7 strains involved in the quorum sensing system

implies that *B. gladioli* relies on alternative pathogenic mechanisms for infection (Fig. 6). The flagellar assembly process in *Burkholderia* species involves several genes and proteins that contribute to flagella synthesis. Early gene products, such as *FlhA, FlhB, FliO*, *FliP*, *FliQ*, and *FliR*, play crucial roles in initiating flagella assembly. Proteins such as *MotA* and *MotB* anchor the motor complex to the cell membrane, which is essential for bacterial motility. Genes that have not been experimentally verified are indicated by white boxes, meaning their role in the flagellar pathway is uncertain (Jang et al., 2014; Wang et al., 2022).

Quorum sensing (QS) regulates gene expression in response to population density, affecting behaviors like biofilm formation and virulence factor production. In *B. glumae*, QS is mediated by N-acyl homoserine lactones (AHLs), specifically C6-HSL and C8-HSL. These molecules trigger the expression *of toxJ* and *toxR* genes, which contribute to toxoflavin biosynthesis. In contrast, *B. gladioli* produces pyoverdine, a high-affinity siderophore that helps the bacterium acquire iron from host tissues, enhancing its survival and pathogenicity (Pérez-Mendoza et al., 2014; Wang et al., 2021).

Under the quorum sensing pathway, the *toxI* genes were indicated with a white box for *B. gladioli* UPMBG7 strains, indicating that this gene was absent. The *toxI* gene is often linked to certain strains of *B. glumae*, which is known to produce the phytotoxin toxoflavin, contributing to its pathogenicity in plants, particularly rice. Instead of relying on toxin production, *B. gladioli* employs colonizing plant tissues, promoting systemic infection, and utilizing plant resources for growth. Furthermore, *B. gladioli* can produce other bioactive compounds and enzymes contributing to its pathogenicity in rice and other hosts (Köhl and Schmitzet, 2018; Pritchard et al., 2020). The gene *pvdA* in *B. gladioli* is essential for its pathogenicity, particularly toward crops such as rice. These genes are critical in synthesizing pyoverdine, a high-affinity iron-chelating compound known as a siderophore. The *pvdA* gene initiates pyoverdine biosynthesis, creating a molecule specifically designed to capture ferric iron (Fe^{3+}) with high affinity. This process allows *B. gladioli* to acquire iron more effectively than other organisms in the host environment, hijacking this essential resource from the plant and giving the bacterium a survival advantage. By utilizing this strategy, *B. gladioli* can effectively thrive in the hostile environments of its plant hosts, leading to significant agricultural impacts, particularly in crops like rice (Pérez-Mendoza et al., 2014; Wang et al., 2021).

Comparative annotation and functional insights

We compared our *Burkholderia* strain's annotation and genomic features with the reference genome

and other published *Burkholderia* genomes of the same species through the GenBank NCBI database. Despite the inherent limitations of second-generation sequencing, resulting in contigs and occasional gaps in the assembly, our annotation efforts were remarkably successful in capturing key genetic components. These genes play a pivotal role in the pathogenicity and virulence of the bacterium. Our annotation revealed that the structures of these virulent genes closely resemble those found in the reference genome. This robust annotation confirms the presence of these virulent genes in our strain and highlights their integrity and potential functionality (Table 3).

In addition, 13 genes associated with the virulence factors were selected, as identified in a previous study. Furthermore, 22 open reading frames (ORFs) within the genetic region were unveiled by annotating the genome sequence using NCBI PGAP. A comparative analysis of our virulence genes' nucleotide sequences with other genome data in NCBI revealed no significant differences. The predicted sequences of virulence genes exhibited a remarkable similarity, with a size and nucleotide matching over 98% when compared to those archived in the NCBI database. The presence of the QsmR transcriptional regulator, a LuxR-type quorum-sensing regulator, further highlights the complexity of their infection strategies. This regulator modulates multiple virulence pathways, including quorum sensing, motility (*flhC, flhD*), and secretion systems, underscoring the importance of bacterial communication in host infection (Chun et al., 2009; Vander Broek & Stevens, 2017). The Type III secretion system (T3SS), plays a fundamental role in host invasion and immune suppression. The *HrpB* protein, a key component of T3SS, facilitates bacterial attachment and injection of effector proteins into rice cells, effectively disrupting host immune responses and enhancing bacterial colonization. However, while both pathogens share this core virulence mechanism, they diverge in their infection strategies, particularly in their reliance on toxin production and nutrient acquisition (Green & Mecsas, 2016; Lipscomb & Schell, 2011; Mannaa et al., 2018). A major distinction between *B. glumae* and *B. gladioli* lies in their ability to manipulate host physiology through different virulence pathways. *B. glumae* K6 relies

Table 3 Genes involved in the virulence factor of *Burkholderia* spp

No	System	Gene	Description	Reference
1	Toxoflavin Production (*B. glumae*)	*TofI*	*N-Acyl* homoserine lactone (AHL) synthase for *N-hexanoyl* homoserine lactone (C6-HSL) and C8-HSL	Kim et al., (2004)
2		*TofR*	Cognate receptor for C8-HSL	Kim et al., (2004)
3		*ToxR*	LysR-type transcriptional activator	Ham et al., (2010)
4		*ToxJ*	LuxR C-terminal-related transcriptional regulator	Ham et al., (2010)
5		*toxF, toxG, toxH, toxI*	Toxoflavin transport	Peng et al., (2015)
6		*ToxA, ToxB, ToxC, ToxD, ToxE*	Toxoflavion biosynthesis	Peng et al., (2015)
7	Flagella Formation	*QsmR*	Ic1R-type transcriptional regulator	Kim et al., (2007)
8		*FlhC*	Flagellar assembly, biosynthesis, chemotaxis	Kim et al., (2007)
9		*FlhD*	Flagellar assembly	Kim et al., (2007)
10	Type III Secretion System	*HrpB*	Regulatory factor for expression of T3SS	Kang et al., (2008)
11	Lipase Formation	*LipA*	Cell-wall degrading enzyme	Zhou-qi et al., (2016)
12		*LipB*	Biosynthesis and activation of *LipA*	Zhou-qi et al., (2016)
13	Siderophore Production (*B. gladioli*)	*PvdA*	Encodes enzyme for pyoverdine synthesis	Pérez-Mendoza et al., (2014)

on toxoflavin biosynthesis, regulated by the *toxABCDE* gene cluster. This toxin is tightly controlled by quorum sensing via the ToxJ-ToxR regulatory system, ensuring synchronized production during infection to maximize host tissue damage. In contrast, *B. gladioli* employs pyoverdine siderophores (*pvdA*) as an alternative virulence strategy. Beyond these core virulence traits, both pathogens encode lipopolysaccharide (LPS) biosynthesis genes (*lipA, lipB*), which contribute to bacterial adhesion, immune evasion, and outer membrane stability by binding to host cell receptors, facilitating infection (Bertani & Ruiz, 2018; Ham et al., 2010; Lee et al., 2019; Tribble & Lamont, 2010).

In our study, comparative genomic analysis was conducted using KEGG pathway annotation to categorize genes based on their functions. While descriptive data effectively highlighted the presence and absence of key virulence genes, we acknowledge the absence of inferential statistical tests to determine the significance of gene content variations in our strains. However, our findings were strongly supported by BLAST analysis against the NCBI GenBank database, which validated the absence of *PvdA* in *B. glumae K6* and *ToxI* in *B. gladioli UPMBG7* (Tables 4 and 5). The significant BLAST hits (E-value > 1.0) for these genes in their respective genomes confirm their absence, while genes detected in both strains showed high sequence identity (> 95%) with known homologs from *Burkholderia* spp. Instead of using inferential statistics, our approach relies on sequence-based validation.

However, while the study effectively establishes these novel insights, it could further emphasize how these findings fill gaps in existing research. For instance, past studies on *B. glumae* focused on its quorum-sensing mechanisms and toxoflavin-mediated virulence (Kim et al., 2004; Peng, 2015), but

Table 4 The genetic regions of *B. glumae* K6 that encode important genes involved as virulence factors

ORF No	Start	End	Putative Protein	Gene Name	Bases	Contigs	Accession no
ORF 1	7035	7646	GNAT family N-acetyltransferase/AHL synthase	*TofI*	612	84	JAMYCS010000087.1
ORF 2	5211	8882	AHL receptor	*TofR*	3672	84	JAMYCS010000087.1
ORF 3	41863	42862	LysR family transcriptional regulator	*ToxR*	1000	6	JAMYCS010000006.1
ORF 4	50199	51029	LuxR C-terminal-related transcriptional regulator	*ToxJ*	831	6	JAMYCS010000006.1
ORF 5	40634	41371	class I SAM-dependent methyltransferase	*ToxA*	738	6	JAMYCS010000006.1
ORF 6	39934	40512	GTP cyclohydrolase II	*ToxB*	579	6	JAMYCS010000006.1
ORF 7	38186	39877	WD40 repeat domain-containing protein	*ToxC*	1692	6	JAMYCS010000006.1
ORF 8	37098	38078	Formylglycine-generating enzyme family protein/TRP2	*ToxD*	981	6	JAMYCS010000006.1
ORF 9	35944	37029	Bifunctional diaminohydroxyphosphoribosylaminopyrimidine deaminase/5-amino-6-(5-phosphoribosylamino) uracilreductase *RibD*/deaminase	*ToxE*	1084	6	JAMYCS010000006.1
ORF10	42958	43533	DMT family transporter	*ToxF*	576	6	JAMYCS010000006.1
ORF11	43573	44712	Efflux RND transporter periplasmic adaptor subunit	*ToxG*	1140	6	JAMYCS010000006.1
ORF12	44709	47801	Efflux RND transporter permease subunit	*ToxH*	3093	6	JAMYCS010000006.1
ORF13	47873	49410	Efflux transporter outer membrane subunit	*ToxI*	1538	6	JAMYCS010000006.1
ORF14	95842	96669	IclR family transcriptional regulator	*QsmR*	828	10	JAMYCS010000010.1
ORF15	8203 5893	9357 7047	Flagellin	*FlhC*	1155	57 69	JAMYCS010000059.1 JAMYCS010000071.1
ORF16	51921	52240	Flagellar transcriptional regulator FlhD	*FlhD*	320	39	JAMYCS010000041.1
ORF17	27443	28846	Helix-turn-helix transcription regulator of pathogenicity genes	*HrpB*	1404	63	JAMYCS010000065.1
ORF18	2292	3278	Lipoyl synthase	*LipA*	987	83	JAMYCS010000086.1
ORF19	3271	4023	Lipoyl(octanoyl) transferase *LipB*	*LipB*	753	83	JAMYCS010000086.1

Table 5 The genetic regions of *B. gladioli* UPMBG7 that encode important genes involved as virulence factors

ORF No	Start	End	Putative Protein	Gene Name	Bases	Contigs	Accession no
ORF 1	9949	10527	GNAT family N-acetyltransferase/AHL synthase	*TofI*	579	43	JANIEE010000046.1
ORF 2	8646	10697	AHL receptor	*TofR*	2052	43	JANIEE010000046.1
ORF 3	29000	29997	LysR family transcriptional regulator	*ToxR*	998	52	JANIEE010000056.1
ORF 4	113847	114661	LuxR C-terminal-related transcriptional regulator	*ToxJ*	815	2	JANIEE010000002.1
ORF 5	27769	28506	class I SAM-dependent methyltransferase	*ToxA*	738	52	JANIEE010000056.1
ORF 6	27070	27648	GTP cyclohydrolase II	*ToxB*	579	52	JANIEE010000056.1
ORF 7	25322	27013	WD40 repeat domain-containing protein	*ToxC*	1692	52	JANIEE010000056.1
ORF 8	24226	25202	Formylglycine-generating enzyme family protein/ TRP2	*ToxD*	977	52	JANIEE010000056.1
ORF 9	23078	24153	Bifunctional diaminohydroxyphosphoribo-sylaminopyrimidine deaminase/5-amino-6-(5-phosphoribosylamino) uracilreductase *RibD*/ deaminase	*ToxE*	1076	52	JANIEE010000056.1
ORF10	30093	30668	DMT family transporter	*ToxF*	576	52	JANIEE010000056.1
ORF11	30708	31847	Efflux RND transporter periplasmic adaptor subunit	*ToxG*	1140	52	JANIEE010000056.1
ORF12	31844	34936	Efflux RND transporter permease subunit	*ToxH*	3093	52	JANIEE010000056.1
ORF13	65230	66031	IclR family transcriptional regulator	*QsmR*	802	32	JANIEE010000035.1
ORF14	197016	198170	Flagellin	*FlhC*	1155	15	JANIEE010000015.1
ORF15	204467	204786	Flagellar transcriptional regulator FlhD	*FlhD*	320	15	JANIEE010000015.1
ORF16	185840	187239	Helix-turn-helix transcriptional regulator	*HrpB*	1400	8	JANIEE010000008.1
ORF17	88686	89678	Lipoyl synthase	*LipA*	993	23	JANIEE010000023.1
ORF18	89671	90359	Lipoyl(octanoyl) transferase *LipB*	*LipB*	689	23	JANIEE010000023.1
ORF 19	115282	116622	NAD (P)/FAD-dependent oxidoreductase	*PvdA*	1341	1	JANIEE010000001.1

this research reveals additional genomic elements that contribute to its adaptability in different environments. Similarly, the ecological versatility of *B. gladioli* has been recognized as both a plant pathogen and an opportunistic human pathogen (Pérez-Mendoza et al., 2014; Pritchard et al., 2020). This comparative approach helps delineate their distinct pathogenic strategies and provides a foundation for improved disease management.

Furthermore, given *B. glumae* dependence on toxoflavin, quorum sensing inhibitors that disrupt toxoflavin biosynthesis could serve as a novel strategy to mitigate disease severity. Similarly, iron-chelating compounds may limit *B. gladioli* infections by interfering with pyoverdine-mediated iron acquisition. The identification of virulence regulators such as QsmR and T3SS effectors suggests additional molecular targets that could be explored for RNA interference-based strategies or the development of small-molecule inhibitors aimed at blocking bacterial signalling pathways. Additionally, these findings can contribute to molecular breeding programs, particularly in the selection of rice cultivars resistant to bacterial attachment and immune suppression. Genome editing approaches, such as CRISPR-Cas9, could be explored to introduce resistant alleles that disrupt bacterial colonization.

While this study provides a comprehensive genomic perspective on *B. glumae* and *B. gladioli* virulence, several limitations must be acknowledged. One of the primary limitations is the absence of in planta validation of the identified virulence genes. Although comparative genomic analysis provides strong evidence of their involvement in pathogenicity, functional validation using mutant strains, gene knockout studies, and complementation assays would further substantiate these findings. For instance, targeted deletion of genes such as qsmR, toxA, or T3SS effectors, followed by inoculation on susceptible rice cultivars, could help determine their contribution to disease severity. In addition to gene-level validation, future research should include transcriptomic (RNA-seq) and proteomic profiling of infected rice

tissues during different stages of infection. This would allow researchers to identify virulence genes that are actively expressed in planta and uncover host responses that correlate with pathogen attack.

This study highlights the distinct but complementary pathogenic strategies of *B. glumae* and *B. gladioli*, offering new insights into their genomic adaptations and virulence mechanisms. By linking these findings to future gene-function studies, transcriptomics, and phenotypic assays, this work lays the groundwork for practical applications in quorum-sensing inhibition, genome editing, biocontrol, and resistance breeding, contributing to sustainable BPB management in rice production systems.

Genome sequence accessibility

The complete genome sequences of *B. glumae* K6 and *B. gladioli* UPMBG7 are available in the NCBI GenBank for further research and reference. *B. glumae* K6 is linked to assembly accession number JAMYCS000000000, while *B. gladioli* UPMBG7 has the assembly accession number JANIEE000000000.

Conclusion

Comparative genomic analysis revealed distinct adaptations between Malaysian *B. glumae* K6 and *B. gladioli* UPMBG7. *B. glumae* K6 carried a complete tox operon (*toxABCDE, toxJ*), regulated by quorum-sensing genes (*tofI, tofR, toxR*), enabling toxoflavin biosynthesis and secretion, a major virulence factor that induces host necrosis. While similar systems are found in other regions, gene variations suggest possible regional adaptations affecting disease severity. In contrast, *B. gladioli* UPMBG7 lacked the *toxI* regulator but encoded pyoverdine siderophore genes (*pvdA*), enhancing iron uptake under nutrient limitation. This indicates differing strategies: *B. glumae* relies on toxoflavin cytotoxicity, whereas *B. gladioli* adopts resource competition via iron acquisition.

These genomic insights offer opportunities for disease management. Potential strategies include toxoflavin-degrading bacteria (*Pseudomonas putida*), quorum-sensing inhibitors (e.g., furanones), or targeting *pvd* siderophore genes with iron-chelating compounds. T3SS effectors (*hrcC, hrpB*) also represent useful markers for breeding resistant cultivars.

The study highlights that region-specific genomic variants (e.g., *qsmR, toxR*) can aid molecular diagnostics and surveillance. Integrating these with metagenomic sequencing provides early detection tools, enabling precision agriculture approaches to control BPB. Ultimately, whole-genome data support breeding resistant rice varieties, designing biological controls, and tailoring management to pathogen diversity.

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Declarations

Conflicts of interest The authors declare no conflict of interest.

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