



SHORT COMMUNICATION

Bordetella hinzii Genomic Characteristics and Vaccine Target Discovery

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Abstract

Objective: This study was aimed at analyzing the pathogenic characteristics of *Bordetella hinzii* (*B. hinzii*), and elucidating its antibiotic resistance mechanisms, virulence gene distribution, and vaccine development potential.

Methods: Genomes of 38 global strains were downloaded from the NCBI database. After quality control with CheckM, pan-genome analysis was performed with Roary, SNP-based phylogenetic trees were constructed with snippy, and antibiotic resistance genes (CARD database) and virulence genes (VFDB database) were predicted with abricate. Bioinformatics tools were integrated to screen candidate vaccine proteins.

Results: All strains carried intrinsic resistance genes, including efflux pumps (*MexAB-OprM*) and the β -lactamase *HBL-1*, which conferred resistance to ten antibiotic classes. Core virulence genes (*bplA-L*, *bvgAS*, and flagella-associated genes) were highly conserved (detection rate >92.1%). SNP analysis classified the strains into six clades, and human-derived isolates showed host-specific clustering. The pan-genome was open (7,530 genes; Heaps' law parameter $B = 0.35$). Two outer membrane proteins, LptD and BamD, were identified as vaccine candidates (antigenicity scores >0.4; no host homology).

Conclusion: *B. hinzii* exhibits cross-host transmission risks, driven by conserved virulence genes and an open genome enabling adaptive evolution. LptD and BamD represent potential vaccine targets requiring further validation. This study provides a theoretical basis for clinical management and prevention strategies.

Key words: *Bordetella hinzii*, Multidrug resistance, Virulence genes, Pan-genome, Vaccine development

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INTRODUCTION

Bordetella hinzii (*B. hinzii*) is a Gram-negative, rod-shaped, obligately aerobic bacterium classified within the genus *Bordetella*, which includes notable human pathogens such as *B. pertussis* (whooping cough) and *B. bronchiseptica*. *B. hinzii* was initially recognized primarily as an avian commensal inhabiting the respiratory tracts of poultry, particularly turkeys

and chickens, and its pathogenic potential for humans remained obscure until relatively recently. Perceptions markedly shifted in 1994, when a report from Japan documented the first human infection in a patient with HIV/AIDS. This critical discovery revealed that *B. hinzii* is an opportunistic pathogen capable of causing substantial disease in immunocompromised individuals. Subsequent

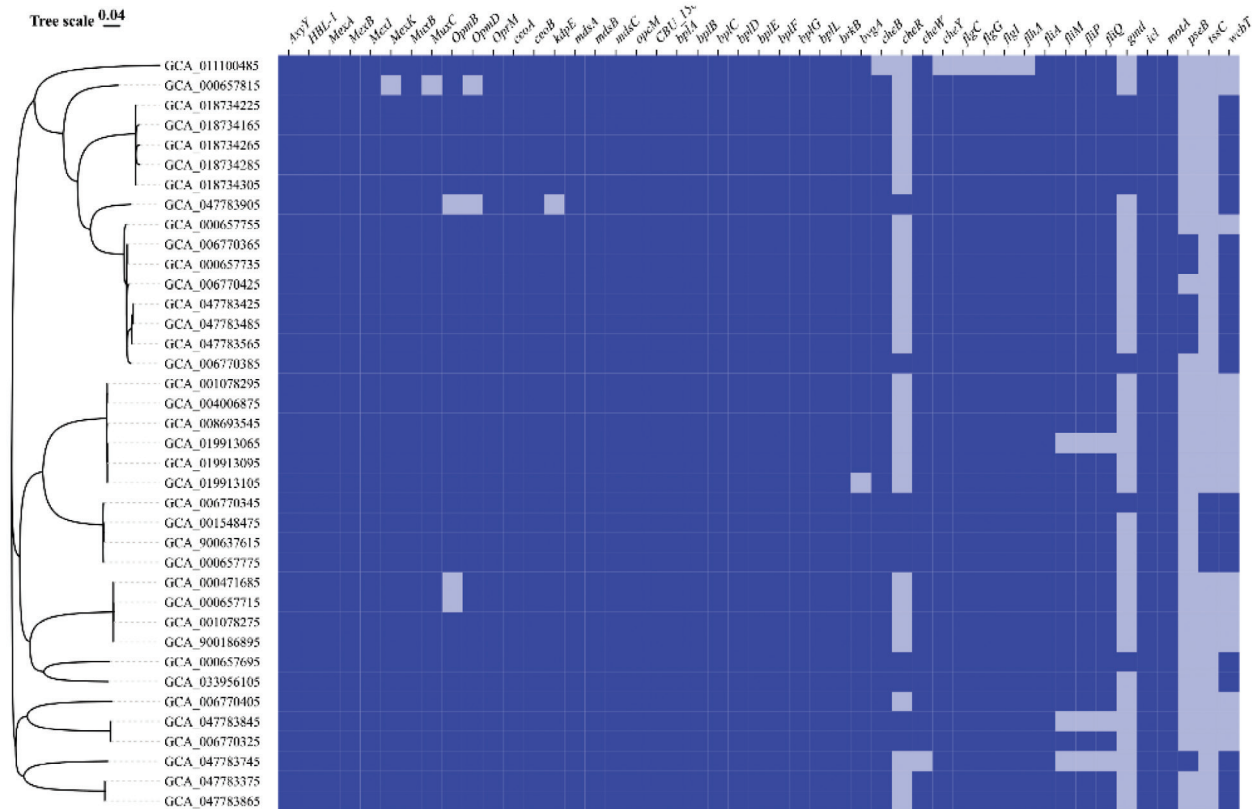


FIGURE 1 | Distribution of resistance and virulence genes.

clinical investigations and case reports progressively revealed the diverse spectrum of infections associated with *B. hinzii* in humans. Beyond its initial association with respiratory illness, this bacterium has been implicated in a range of serious conditions, including meningitis (inflammation of the brain and spinal cord membranes), bacteremia (bacteria in the bloodstream, which can lead to sepsis), pulmonary infections (pneumonia), endocarditis (infection of the heart valves), chronic cholangitis (inflammation of the bile ducts), and soft tissue abscesses. This expanding clinical profile underscores *B. hinzii*'s adaptability as a human pathogen. The zoonotic origin of *B. hinzii* is a critical aspect of its epidemiology. Whereas this bacterium can reside harmlessly in birds, transmission to humans can occur, probably through occupational exposure (e.g., in poultry workers) or environmental contamination. More alarmingly, evidence suggests that, after being introduced into human populations, *B. hinzii* can achieve sustained human-to-human transmission, particularly in healthcare settings. This potential for person-to-person spread markedly elevates *B. hinzii*'s public health threat, particularly as an opportunistic pathogen exploiting vulnerabilities in immunocompromised hosts, such as people with HIV/AIDS, and people undergoing chemotherapy or receiving immunosuppressive therapy [1,2].

A major leap in the understanding of this emerging pathogen occurred in 2013, with the publication of the first complete genome sequence of *B. hinzii*. This genomic milestone provided invaluable insights into genetic makeup, virulence factors, and evolutionary relationships. Crucially, the findings highlighted *B. hinzii*'s emergence as a novel human pathogen warranting focused attention. Given the ability of its close genetic relatives (e.g., *B. pertussis* and *B. paraptussis*) to cause substantial human epidemics, and *B. hinzii*'s zoonotic transmission ability and potential for persistent interhuman transmission, comprehensive investigations of its biology, evolution, and pathogenicity are essential [1,2].

Toward this goal, our research group has identified a clinical case of pulmonary infection caused by *B. hinzii* (strain GCA_033956105), thus highlighting its clinical relevance. *B. hinzii* shows a concerning convergence of the following key characteristics: zoonotic adaptation (jumping from animals to humans), genomic plasticity (allowing for evolution and adaptation), and nosocomial resilience (ability to persist and spread in healthcare environments). Its evolutionary trajectory from an avian commensal to a multidrug-resistant opportunistic pathogen capable of causing severe human disease, notably underscores the critical urgency for enhanced genomic surveillance at the dynamic animal-human interface [3,4].

To address these pressing concerns, we used a strategically curated collection of *B. hinzii* isolates sourced globally from diverse hosts (human, avian, and environmental) and geographical locations. Our primary aim was to decipher the intricate evolutionary mechanisms, such as horizontal gene transfer, recombination, and mutation, underlying this pathogen's remarkable ability to persist across disparate ecological niches (e.g., birds, humans, and healthcare settings). Understanding these fundamental drivers of adaptation and transmission is essential. The insights gained might ultimately contribute to safeguarding global public health by directly informing the development of improved diagnostic tools for rapid and accurate detection; guiding the design of more effective therapeutic strategies (particularly in light of multidrug resistance); and critically enhancing pandemic preparedness for emerging *Bordetella* species such as *B. hinzii*.

MATERIALS AND METHODS

Dataset retrieval and quality control

A total of 39 *B. hinzii* genome sequences were retrieved from the NCBI Assembly database with the keyword “*Bordetella hinzii*” (retrieval date: February 26, 2025). Quality control was performed on all genomes with CheckM v1.0.13 (<https://github.com/Ecogenomics/CheckM>). Genomes with >95% completeness and <5% contamination were retained, thus yielding 38 qualified genomes for downstream analyses. Average nucleotide identity values between all genomes and the *B. hinzii* reference strain (GCA_900637615) were calculated with FastANI. An average nucleotide identity >95% confirmed species identity.

Pan-genome analysis

Gene prediction and annotation for the 38 genomes were conducted with Prokka v1.14.5 (<https://github.com/tseemann/prokka>). The resulting GFF files were analyzed with Roary v3.12.0 (<https://github.io/Roary>) to construct the pan-genome. The pan-genome growth curve was modeled with Heaps' law ($y = Ax^B + C$), where B indicates openness ($B > 0.35$) or closure. The core genome dynamics was fitted to the exponential decay model $y = Ae^{Bx} + C$.

Phylogenetic analysis

Core genome SNPs were identified by mapping sequences to the reference genome (GCA_900637615) with snippy v4.6.0 (<https://anaconda.org/bioconda/snippy>). Recombination events were removed with Gubbins, and a maximum-likelihood phylogenetic tree was constructed with FastTree.

Comparative genomics

ISEScan (<https://github.com/xiezhq/ISEScan>) was used to detect insertion sequences (IS). Prophages were predicted via PHASTEST (<https://phastest.ca/>), and categorized as intact (score >90), questionable (70–90), or incomplete (<70). Only regions with scores ≥70 were retained. Resistance genes were identified with Abricate

against the CARD database, and virulence genes were annotated against the VFDB database.

Vaccine candidate screening

Core gene protein sequences were extracted. Essential proteins were filtered with GepTop v2.0 (<http://gepa.org.cn/geptop/predict.html>; cutoff: 0.24). Subcellular localization was predicted via PSORTb v3.0 (<https://www.psort.org/psortb/>), by selecting outer membrane proteins. Further screening included transmembrane helices (<2) via TMHMM-2.0 (<https://services.healthtech.dtu.dk/services/TMHMM-2.0/>), antigenicity (>0.4) via VaxiJen v2.0 (<https://www.ddg-pharmfac.net/vaxijen/VaxiJen/VaxiJen.html>), and non-allergenicity via AllerTOP v2.1 (https://www.ddg-pharmfac.net/allertop_test/). Physicochemical properties were evaluated with ProtParam (<https://web.expasy.org/protparam/>), by selecting proteins with molecular weight 10–110 kDa, an instability index <40, and an aliphatic index >50. Non-homology to human proteins was confirmed via BLASTp (<https://blast.ncbi.nlm.nih.gov>).

RESULTS

Strain characteristics

The dataset included 38 strains: 17 from human infections, 19 from birds/poultry, and 2 of unknown origin. The geographical distribution was as follows: USA (22), China (2), Russia (8), Australia (2), France (1), Hungary (1), and unknown (2).

Virulence and resistance genes

Abricate identified 18 resistance genes across all strains, including efflux pumps (including *MexAB-OprM*, *MdsABC*, and *CeoAB-OpcM*), the *KdpD/KdpE* two-component system, and the β -lactamase gene *HBL-1*. The universal presence of genes encoding efflux pumps such as *MexAB-OprM*, *MdsABC*, *CeoAB-OpcM*, and *HBL-1* suggested genetic potential for intrinsic resistance to multiple antibiotic classes including β -lactams, aminoglycosides, and fluoroquinolones. Importantly, the presence of these genes alone does not confirm resistance, because phenotypic resistance depends on actual expression and activity levels. Functional validation (e.g., measuring expression via RT-qPCR or proteomics) is required to assess the genes' contributions to resistance phenotypes. This prediction is supported by numerous studies indicating that overexpression of these efflux systems (or their homologs) is a well-established mechanism of multidrug resistance in *Bordetella*.

Twenty-eight virulence genes associated with motility, immune evasion, and regulation were detected. All strains (38/38, 100%) carried *brkB* and *bplA-L* (except *bplL*), which are critical for host cell invasion. The *BvgAS* system (*bvgA*: 37/38, 97.4%) suggested dynamic virulence regulation. Chemotaxis genes (*cheB*, *cheW*, and *cheY*) and flagellar genes (*flgC*, *flgG*, *flgI*, *fliA*, and *fliM*) indicated motility and tissue colonization potential (Fig 1).

A total of 55 prophage regions were detected across the 38 strains with the online tool PHASTER, with an average length of 41 kb (range: 6.9–59.9 kb) and GC content ranging from 6.19% to 69.11%. Erwini_phiEt88 (NC_015295) was detected in 20 strains (52.63%), and the longest fragment (59.9 kb, GCA_900186895) encoded 61 proteins potentially involved in host metabolic regulation. The specific prophages Entero_HK022 (NC_002166),

SNP analysis of 38 *B. hinzii* genomes was performed via snippy with GCA_900637615 as the reference genome. The number of SNPs identified ranged from 1 to 3,838. The *B. hinzii* strains were divided into six clades (clades 1–6) and two monophyletic groups (Fig 3). Within each clade, the SNP differences did not exceed 250, thus demonstrating host-associated clustering. Human-derived strains clustered in clades 1, 3, and 5. Notably,



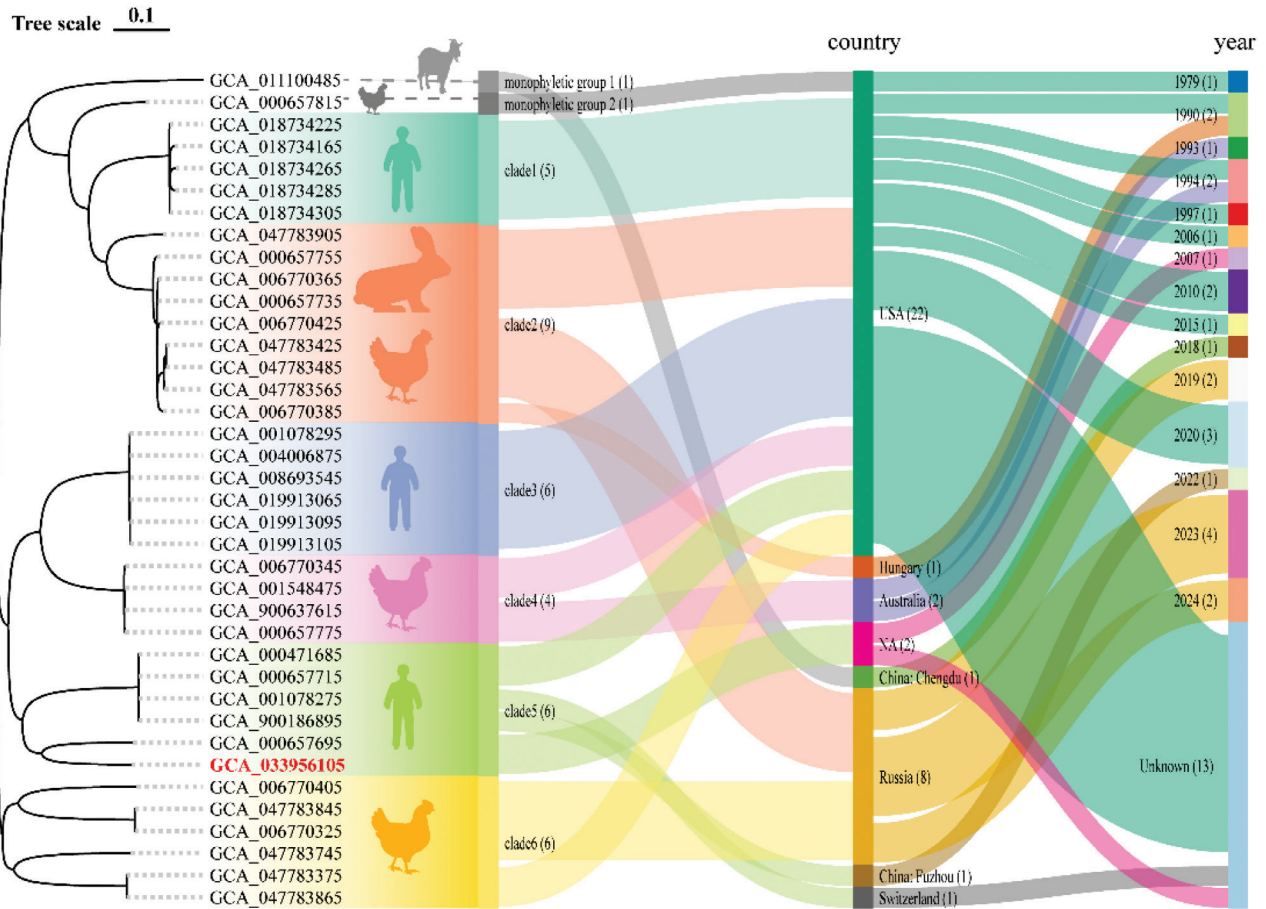


FIGURE 3 | SNP phylogenetic tree (Strains from Fujian Province are labeled in red; host origin information for GCA_004006875, GCA_000471685, and GCA_900186895 is not available in NCBI).

the strain isolated in Fuzhou, China (GCA_033956105), in this study exhibited close phylogenetic relatedness and high genomic similarity to the Switzerland-derived strain GCA_000657695.

Pan-genome analysis

After annotation of genomic sequences from 38 *B. hinzii* strains, we performed pan-genome analysis with Roary. The results revealed a total pan-genome size of 7,530 genes, comprising 3,579 core genes (47.53%), 410 soft-core genes (5.44%), 1,048 shell genes (13.92%), and 2,493 cloud genes (33.11%). To investigate the expansion dynamics and openness of the *B. hinzii* pan-genome, we applied Heaps' law ($y = Ax^B + C$) to model the pan-genome growth curve. As shown in the figure, the pan-genome size increased progressively with the addition of more genomes, and the growth trend was best fitted by the equation $y = 1089.17x^{0.35} + 3503.86$. An exponent B (0.35) greater than 0 indicated an open pan-genome structure (Fig 4).

Vaccine candidate protein screening

Screening of 3,579 core gene proteins identified through Roary-based pan-genome analysis was performed to

select essential proteins and predict subcellular localization. The results revealed three outer membrane proteins as potential candidates. Subsequent analyses of transmembrane helix count, antigenicity, allergenicity, and non-host homology led to the exclusion of bamA_2, because of its host homology. The remaining two proteins, lptD and bamD, were subjected to evaluation of the following physicochemical properties aligning with criteria for viable vaccine candidates: molecular weight <110 kDa for both proteins, instability index <40 (indicating high stability), and aliphatic index >40 (suggesting thermostability) (Table 1). Phylogenetic analysis with the maximum likelihood method was conducted with the 50 most closely related sequences from NCBI to assess evolutionary conservation and divergence. bamD and lptD were most closely associated with MCJ9708576.1:1-247 *B. hinzii* and WP 029581559.1:3-800 *B. hinzii*, respectively (Fig 5).

DISCUSSION

B. hinzii has garnered attention because of its association with respiratory infections in animals. Notably, this pathogen has been identified in various avian species (including

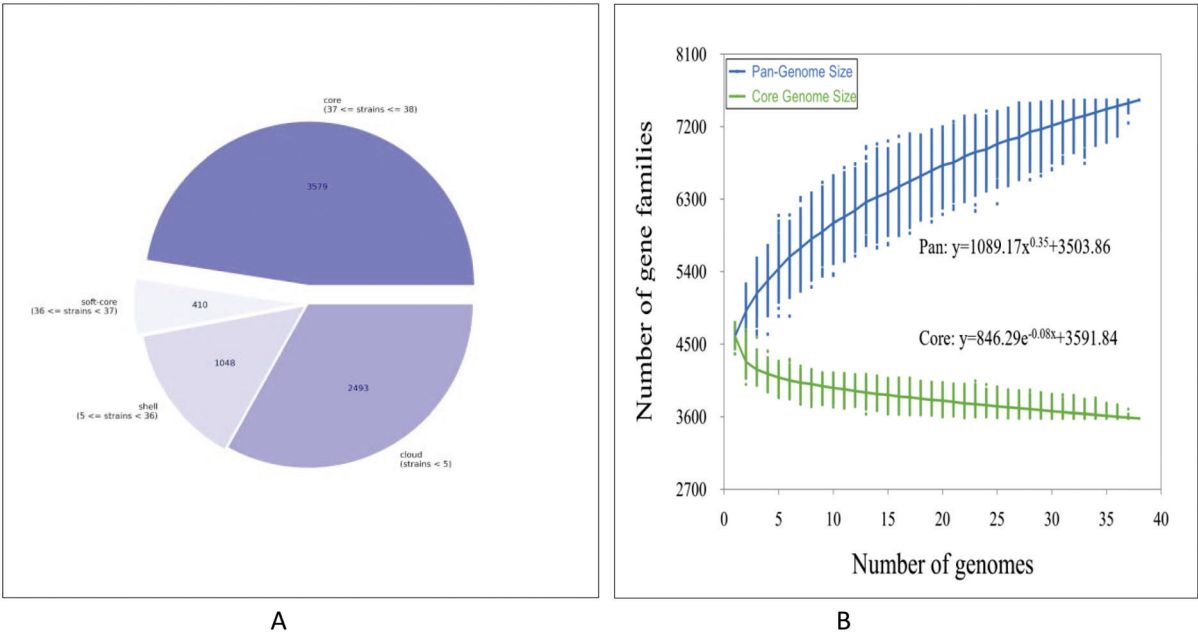


FIGURE 4 | Pan-genome evolutionary analysis.

TABLE 1 | Predicted vaccine-related outer membrane proteins in *Bordetella hinzii*.

Protein name	Host homology	Antigenicity score	Allergenicity prediction	Essential proteins	Transmembrane helices	Molecular weight (kDa)	Instability index	Aliphatic index
bamA_2	53%	0.5668	-	+	1	-	-	-
lptD	-	0.6396	-	+	0	89.2	35.72	70.44
bamD	-	0.4445	-	+	0	31	33.49	70.69

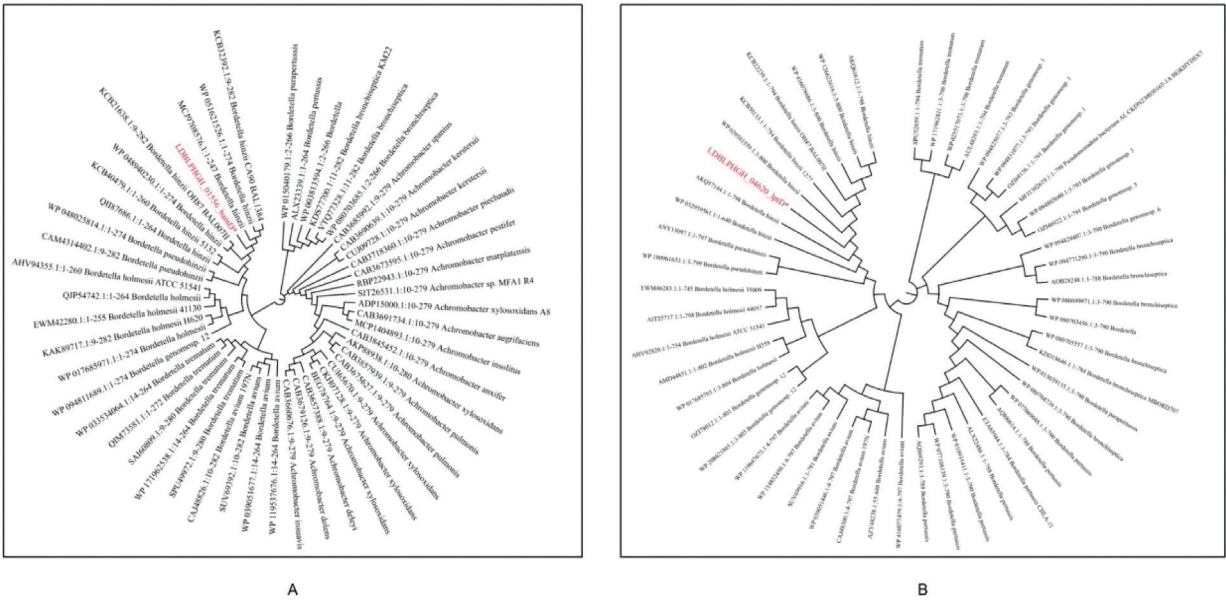


FIGURE 5 | Evolutionary analysis of bamD and lptD amino acid sequences (A: bamD; B: lptD).

poultry, pigeons, and pet birds) and rodents (e.g., mice and rats). In humans, *B. hinzii* infections are rare; are typically associated with immunocompromised states, prior SARS-CoV-2 infection, or exposure to poultry; and manifest as pneumonia with or without bacteremia [2,3]. Respiratory symptoms such as cough, rhinorrhea, nasal congestion, and fever vary in severity, according to the infecting strain and host immune status.

Genomic analysis of 38 global *B. hinzii* strains revealed a genome size of 4.8 Mbp (range: 4.70–4.84 Mbp) and GC content of ~67.2%. All strains carried 18 antimicrobial resistance (AMR) genes, including the efflux pump systems *MexAB-OprM*, *MdsABC*, and *CeoAB-OpcM*, as well as the β -lactamase gene *HBL-1*. Genes encoding major efflux pumps (*MexAB-OprM*, *MdsABC*, *CeoAB-OpcM*, and *HBL-1*) were universally identified in the studied genome. Although the presence of these genes provides a genetic basis for efflux-mediated resistance, conferring actual resistance to antibiotics, such as β -lactams, aminoglycosides, or fluoroquinolones, typically requires elevated expression and functional activity of these pumps. Therefore, our bioinformatic detection indicated a potential resistance mechanism but not a confirmed resistance phenotype. Future studies should focus on quantifying the expression and functional contributions of these efflux systems under relevant conditions [4] to highlight the risks of acquired resistance. Virulence profiling identified the *bplA-L* gene cluster (encoding adhesins critical for host cell invasion, functionally analogous to *B. pertussis* filamentous hemagglutinin [5]) in all strains. The high prevalence of the *BvgAS* two-component system (97.4%) underscored its central role in virulence regulation, thereby mirroring its function in *B. pertussis*, in which it mediates adaptive shifts between environmental and host-associated states by modulating toxin (e.g., pertussis toxin) and adhesin expression [6].

Among the 38 strains, 128 ISs were identified, including IS481 (a diagnostic marker for *B. pertussis* [7]) in seven strains. This finding prompts clinical concern, because PCR-based *B. pertussis* assays targeting IS481 might yield false positives in individuals exposed to *B. hinzii*, thus necessitating strain typing for accurate diagnosis. Additionally, 55 prophage types were detected, including human-derived strains uniquely carrying Entero_HK022. As a temperate phage, Entero_HK022 integrates its genome into the host bacterium's chromosome, becoming a prophage. During this process, certain genes carried by the phage, if expressed by the host bacterium, can directly confer new traits upon the host. The high frequency of Erwini_phiEt88 (52.63%) suggests that lysogenic conversion might modulate host metabolism, although functional validation through multi-omics approaches is required.

Phylogenetic SNP analysis divided the 38 strains into six clades and two monophyletic groups, with ≤ 250 SNP differences within clades and distinct host

clustering: human-derived strains were concentrated in clades 1, 3, and 5, whereas animal-derived strains were dispersed across other clades (Fig 3). The close phylogenetic relationship between the Fuzhou, China, isolate (GCA_033956105) and a Switzerland-derived strain (GCA_000657695), despite the geographic distance, might reflect international poultry trade or migratory bird routes [2,3]. However, because sampling bias (22 U.S. strains vs. 2 Chinese strains) was likely to have led to underestimation of Asian genetic diversity, expanded surveillance is warranted.

Pan-genome analysis revealed an open genome structure (Heaps' law exponent $B = 0.35$) comprising 7,530 genes: 3,579 core (47.53%), 410 soft-core (5.44%), 1,048 shell (13.92%), and 2,493 cloud (33.11%). The high proportion of cloud genes suggested horizontal gene transfer-driven adaptability (e.g., antibiotic resistance or host interaction factors), whereas the conserved core genes were likely to maintain essential metabolic and virulence pathways. These findings support the "ecological generalist" hypothesis, wherein *B. hinzii* balances core and accessory genomes to thrive in diverse hosts [8]. Functional annotation of accessory genes (e.g., COG/KEGG) is needed to elucidate niche-specific adaptations.

From the core genome, *lptD* (lipopolysaccharide transport protein) and *bamD* (β -barrel assembly machinery component) emerged as promising vaccine candidates, because of their outer membrane localization, high antigenicity, and absence of host homology. Antibodies targeting *lptD* disrupt outer membrane integrity, a strategy validated in *Neisseria* vaccines [9,10], whereas *bamD* immunization has shown efficacy in *Escherichia coli* models [11]. However, *B. hinzii*-specific animal infection models are required to evaluate protective efficacy. Targeting efflux pumps (e.g., *MexB*) might decrease antibiotic resistance, but cross-reactivity risks warrant careful assessment. The pathogenicity of *Bordetella sinnei* is the result of the combined effect of its basal virulence genes, environmental adaptability, and acquired drug resistance. In the future, further mechanistic verification on the basis of gene-edited animal models and clinical multi-omics will be necessary [12–16].

Limitations and future directions

This study has several limitations, as follows: (1) geographic and host sampling biases might have led to omission of critical evolutionary lineages; (2) AMR genotype-phenotype correlations lacked antimicrobial susceptibility testing; and (3) virulence gene mechanisms require *in vitro/in vivo* validation. Future work should integrate multi-omics (transcriptomics and proteomics) to dissect resistance/virulence networks and use CRISPR-Cas9 mutagenesis in functional studies. Monitoring of environmental *B. hinzii* populations and antibiotic resistance gene dissemination would be valuable to inform strategies to mitigate zoonotic transmission.

CONCLUSION

This study, synthesizing genomic, AMR, virulence, and vaccinology perspectives, highlights *B. hinzii*'s public health risks and research value. By addressing current gaps and proposing integrative approaches, this work lays a foundation for targeted interventions and One Health-driven surveillance.

DATA AVAILABILITY STATEMENT

The data supporting this study are available within the article.

ETHICS STATEMENT

No direct interactions with human or animal subjects were involved. Therefore, ethical approval and informed consent were not required.

AUTHOR CONTRIBUTIONS

Jin Rui and Mao Junjie: Conceptualization, Writing—original draft. Deng Yuhang, Wu Wenwei, and Liang Xuechen: Methods, Formal analysis. Zheng Enhui and Li Quwen: Supervision, Funding acquisition. All authors approved the final manuscript.

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CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

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