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Emergence and genomic characterization of a carbapenem- and colistin-resistant Enterobacter chengduensis isolate carrying blaNDM-1 in Northeast China

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Abstract

Background: *Enterobacter chengduensis* was first isolated from human blood samples in China in 2015, and was defined as a new species of the *Enterobacter* genus in 2019. To date, research on this species remains limited and there have been no reports of similar bacteria isolated from northeastern China. In this study, we investigated the molecular characteristics of *E. chengduensis* in a tertiary hospital.

Results: We identified a multidrug-resistant (MDR) *Enterobacter chengduensis* strain with sequence type (ST) 414.WGS data showed that this isolate carried 5 virulence-associated factors clpK1, fyuA, irp2, nlpI and terC and 15 antimicrobial resistance genes (ARGs), including β-lactam resistance genes (comprising extended-spectrum β-lactamase and carbapenemase-encoding genes),aminoglycoside resistance genes, and fluoroquinolone resistance genes. Our findings enhance the understanding of *E. chengduensis.*.

Conclusion: These findings enhance our understanding of *E. chengduensis*. Early surveillance and the strict implementation of infection control measures are critical to reducing infections and curbing transmission.

Introduction

Enterobacter spp rank third among clinical *Enterobacteriaceae* bacteria, followed by *Escherichia coli* and *Klebsiella*[1]. *Enterobacter chengduensis* was initially isolated from a blood sample in 2015 and later identified as a member of the *Enterobacter cloacae complex* (ECC)[2 3]. Owing to the insufficient resolution of traditional biochemical phenotypic identification techniques, the vast majority of clinical microbiology laboratories in China and abroad cannot accurately classify ECC, indicating that specific species within the complex cannot be reliably differentiated in traditional clinical testing[4].

The number of *Carbapenem-resistant Enterobacteriaceae* (CRE) has shown a continuous upward trend in most regions[5], largely attributable to the widespread clinical application of carbapenems for managing severe infections. In addition, carbapenem-resistant *Enterobacter cloacae complex* (CRECC) has emerged as a critical priority in global efforts to combat antimicrobial resistance.[6] In China, CRECC is mainly mediated by metallo β-lactamases (MBLs), among which *NDM-type* enzymes, especially the *blaNDM-1* are the most prevalent.[7]

In this study, we successfully isolated a strain of *E. chengduensis* YBHECC-C1 from a clinical specimen. Antimicrobial susceptibility testing (AST) verified that the isolate displayed resistance to carbapenems and colistin, which are widely acknowledged as last-resort agents for severe clinical infections. To elucidate the molecular characteristics of this strain, we performed whole-genome sequencing (WGS). To the best of our knowledge, this represents the first report of clinically recovered *E. chengduensis* in Northeast China.

Materials and Methods

Bacterial isolate collection and identification

The clinical isolate, YBHECC-C1, was collected from the lower respiratory tract of the Department of Infectious Diseases in 2024. Preliminary bacterial identification and antimicrobial susceptibility testing were performed using the VITEK 2 Compact automated system (bioMérieux, France). This isolate was stored at -80°C and prior to testing, the frozen stock was revived by streaking onto blood agar plates and incubating overnight (16–24 h) at 37°C. Genomic DNA was extracted using a commercial kit (Tiangen Biotech, Beijing, China) and submitted to Novogene Co., Ltd. for WGS. DNA libraries were constructed with an insert size of approximately 350 bp and sequenced on an Illumina platform in paired-end 150-bp (PE150) mode.

Antimicrobial susceptibility testing (AST)

The minimum inhibitory concentrations (MICs) of commonly used antibiotics were determined by broth microdilution on commercial susceptibility plates (Wenzhou Kangtai Biotechnology Co., Ltd.) containing cation-adjusted Mueller-Hinton broth (CAMHB), including imipenem (IPM), meropenem (MEM), ertapenem (ETP), ceftazidime (CAZ), cefepime (FEP), ceftazidime-avibactam (CZA), aztreonam-avibactam (AZA), piperacillin-tazobactam (TZP), aztreonam (ATM), levofloxacin (LVX), amikacin (AMK), sulfamethoxazole-trimethoprim (SXT), tigecycline (TGC), and polymyxin B (PB). Cefiderocol (CFDC), susceptibility was separately tested using iron-depleted CAMHB (ID-CAMHB). *Escherichia* coli ATCC 25922 was used as the quality control strain for AST to ensure accuracy of the results. All procedures were performed in strict accordance with reference methods described by the Clinical and Laboratory Standards Institute (CLSI)[8]. TGC was interpreted using the breakpoints recommended by the U.S. Food and Drug Administration (FDA). PB was interpreted according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints (version 14.0). The remaining drugs were interpreted following the CLSI M100 Performance Standards for Antimicrobial Susceptibility Testing, 36th edition[9].

Whole genome sequencing and analysis

Genomic DNA was extracted using a DNA extraction kit (Tiangen Biotech, Beijing, China). The purified DNA was subjected to WGS at Novogene Co., Ltd. DNA was fragmented to approximately 350 bp using a Covaris sonicator and then processed for end repair, A-tailing, and Illumina adapter ligation. Sequencing was conducted on the Illumina platform using a paired-end 150 bp (PE150) strategy.

Molecular characteristic analysis

The assembled contigs were submitted to the Center for Genomic Epidemiology (CGE) server (https://cge.food.dtu.dk/) for downstream analyses. The acquired antimicrobial resistance genes (ARGs), sequence type (ST), and plasmids were identified using ResFinder, MLST, and PlasmidFinder, respectively. The virulence factors were screened using the VFDB database (http://www.mgc.ac.cn/VFs/). Circular genome maps were generated using Proksee (https://proksee.ca) to visualize the genomic architecture, and the identified ARGs were mapped onto the assembly.

Phylogenetic Tree Construction

To clarify the phylogenetic position of the local isolate YBHECC-C1, we downloaded all genome sequences annotated as *E. chengduensis* from the NCBI database(as of 1 May 2026). Average nucleotide identity (ANI) was calculated using FastANI v1.33 for all retrieved genomes, with the type strain WCHECL-C4 (GCA_001984825.2) as the reference. This yielded a final set of 77 high-confidence genomes, including the local isolate YBHECC-C1. Core genome alignment was then performed using Parsnp v1.7.4 from the Harvest suite, and core-genome single-nucleotide polymorphisms (cgSNPs) were extracted. Based on the resulting cgSNP alignment, a maximum-likelihood (ML) phylogenetic tree was reconstructed using IQ-TREE v3.1.2. The optimal nucleotide substitution model was automatically selected by the built-in Model Finder, and branch support was assessed with 1000 ultrafast bootstrap replicates. The final tree was visualized and annotated using Itol (iTOL: Interactive Tree Of Life).

Results

Precise Species Identification and MLST Analysis

The initial VITEK 2 result identified the isolate as a member of ECC, to further clarify the taxonomic position of strain YBHECC-C1 within the ECC, ANI analysis was performed based on WGS data, through pairwise comparisons against the type strains of the major species within the ECC. As shown in Figure 1, strain YBHECC-C1 exhibited the highest genetic similarity to the type strain of *E. chengduensis*, with an ANI value of 99.7%. Based on phylogenetic clustering and ANI metrics, the strain was identified as *E. chengduensis*. We utilized an online service (https://tygs.dsmz.de/) reached the same conclusion[10]. On this basis, we further performed MLST analysis and the results showed that the strain belonged to ST414.

Phenotypic Screening for Common Antibiotics and Detection of Antimicrobial Resistance Genes

AST revealed that the strain had a multidrug-resistant phenotype (Table 1). It was resistant to all the tested carbapenems (IPM, MEM, and ETP), monobactams (ATM), fluoroquinolones (LVX), sulfonamides, CZA, and PB, while retaining susceptibility to AZA, AMK, TGC, and CFDC.

Aside from the carbapenemase gene *blaNDM-1*, the strain carried a variety of other resistance genes (Figure 2), including the broad-spectrum β-lactamase gene *blaTEM-1B*, the OXA-type β-lactamase gene *blaOXA-1*, the AmpC β-lactamase (ACT family) gene *blaACT-6*, and the ESBL genes *blaCTX-M-15* and *blaSHV-12*. Aminoglycoside resistance genes included aac(3)-IIa, ant(2")-Ia, and aadA2b. The bifunctional gene aac(6')-Ib-cr was also detected, which can mediate aminoglycoside modification and reduce susceptibility to certain fluoroquinolones. Fluoroquinolone resistance further involved qnrB1. The genome additionally harbored sul1 (sulfonamide), dfrA14 (trimethoprim), tet(A) (tetracycline), and fosA (fosfomycin). Meanwhile, we detected that this strain carried five virulence factors: clpK1, fyuA, irp2, nlpI, and terC (Figure 2).

Table 1 Antimicrobial Susceptibilities of YBHECC-C1

Antimicrobial Agent (Abbr.)	MIC (mg/L)	Interpretation
Imipenem (IPM)	16	R
Meropenem (MEM)	16	R
Ertapenem (ETP)	8	R
Ceftazidime (CAZ)	>64	R
Cefepime (FEP)	>32	R
Cefiderocol (CFDC)	4	S
Ceftazidime/Avibactam (CZA)	>64	R
Aztreonam/Avibactam (AZA)	<0.125	S
Piperacillin/Tazobactam (TZP)	>256	R
Aztreonam (ATM)	>128	R
Levofloxacin (LVX)	2	R
Amikacin (AMK)	4	S
Trimethoprim/Sulfa (SXT)	>32	R
Tigecycline (TGC)	0.25	S
Polymyxin B (PB)	>64	R

R, resistant; S, susceptible.

Maximum-likelihood phylogenetic tree

To determine where YBHECC-C1 sits phylogenetically within *E. chengduensis*, we built a maximum likelihood tree using core genome SNP data from 77 high quality genomes (Figure 3). In the resulting phylogeny, both the local isolate YBHECC-C1 and the type strain WCHECL-C4 were positioned within the core phylogenetic clade. YBHECC-C1 clustered with strain GCA_017312015.1, isolated from the Chinese PLA General Hospital, into the same lineage with strong bootstrap support. The terminal branch leading to YBHECC-C1 was notably longer than that of its closest relative.

Discussion

Previous identification based on DNA-DNA hybridization (DDH) with a 70% cut-off or 16S rRNA sequencing has often been insufficient to discriminate between closely related species within ECC. [4] This species has been recovered from both clinical and environmental sources, highlighting its potential for transmission across both the clinical and environmental compartments[11 12]. This study employed whole-genome ANI analysis to precisely identify strain YBHECC-C1 as *E. chengduensis* (ANI > 99%), thereby underscoring the necessity of WGS for accurate bacterial taxonomy. In the phylogenetic tree, YBHECC-C1 has undergone a relatively greater degree of sequence divergence within the local clade, at least under the cgSNP based framework.

E. chengduensis, a novel species of the *Enterobacter* genus, was first described in 2015 when Wu et al. recovered strain WCHECl-C4 from a human blood sample in China.[2] Importantly, the present study is the first to isolate a strain of *E. chengduensis* from sputum samples in the local region, which exhibited substantial molecular epidemiological divergence from the contemporarily prevalent ECC strains circulating locally. According to our surveillance data, the dominant ECC during the same period was ST171. In stark contrast, only strain YBHECC-C1 belonged to ST414.

AST revealed that the strain was resistant to all the tested carbapenems, and we suspect the presence of the *blaNDM-1* carbapenemase is likely the underlying mechanism, and further analysis pointed to a plasmid-mediated origin for this resistance. IncX3 plasmids are the most common *blaNDM-1* carriers in *Enterobacterales* worldwide, largely due to their high conjugation efficiency and broad host range[13]. Within the ECC, IncX3 also dominates [14 15], Fu et al. reported that *blaNDM-1* in *E. chengduensis* was carried exclusively by IncC plasmids, which differs from our findings[16].WGS data indicated that the strain harboured an IncM1 plasmid. However, due to the abundant presence of insertion sequences surrounding the *blaNDM* gene, Illumina short-read sequencing could not span these highly repetitive regions, resulting in fragmented plasmid assemblies rather than a complete circular structure. Therefore, while depth-of-coverage analysis strongly infers the location of *blaNDM-1* on the IncM1plasmid, resolving the precise plasmid architecture and complete genetic context will require future investigations utilizing long-read sequencing or conjugation assays.

The strain was also resistant to aztreonam, which can be attributed to the extended-spectrum β-lactamase gene *SHV-12*[17]. The active site of *SHV-12* efficiently binds to and hydrolyzes oxyimino-substituted β-lactam antibiotics, including third-generation cephalosporins and aztreonam [18 19]. However, this strain was susceptible to the combination of aztreonam and avibactam. Avibactam potently inhibits *SHV-12*, whereas aztreonam is intrinsically stable against MBL present in this strain. These factors protect aztreonam from hydrolysis and restore its activity together [20]. Previous studies have demonstrated that mutations and overexpression of *SHV-12* result in a reduction in the susceptibility of *Enterobacterial* isolates to AZA, which warrants equal attention in clinical practice and antimicrobial resistance surveillance[21]. In contrast, ceftazidime is highly susceptible to MBL-mediated hydrolysis. Avibactam targets only class A, C, and D serine β-lactamases and shows no activity against class B MBLs. Consequently, avibactam cannot protect ceftazidime from hydrolysis by *BLANDM-1*, which leads to resistance to ceftazidime-avibactam [22].

Strain YBHECC-C1 exhibited high resistance to colistin, with an MIC of > 64 μg/mL. No plasmid-mediated *mcr* family colistin resistance genes were detected by genomic analysis. Therefore, we focused our investigation on chromosomal polymorphisms [23]. No SNPs or short indels were found in the coding regions of *pmrA, pmrB*, or the arn operon[24 25]. When we mapped the YBHECC-C1 reads to the full-length mgrB CDS (144 bp), the entire query sequence was continuously covered (Supplementary Fig. 1), and no SNPs or short indels were called against the reference. Soft-clipping was frequently observed, which is expected given that the reads are longer than the short reference sequence and that short seed matches tend to produce such artifacts. Per-base coverage did not drop in the middle of the gene, arguing against IS-mediated insertional inactivation of mgrB. Taken together, the short-read data do not support *mcr* acquisition, classical coding mutations in *pmrAB/arn*, or IS disruption of mgrB as the mechanism underlying colistin resistance in YBHECC-C1. Regulatory changes, other chromosomal loci, or alterations at the expression level remain formally possible and represent limitations of this study.

Although the tetracycline efflux pump gene *tet(A)* was detected, tigecycline can evade recognition and efflux by typical tetracycline efflux pumps owing to its tert-butylglycylamino side chain at C9 [26 27]. Thus, the presence of *tet*(A) did not compromise tigecycline susceptibility. We did not detect any known tigecycline-specific resistance determinants, including plasmid-mediated *tet*(X) variants or mutations in RND-type efflux pumps like *tmexCD-toprJ* [28 29]. This suggests that the strain lacks functional tigecycline resistance mechanisms; therefore, tigecycline should remain effective against this multidrug-resistant *E. chengduensis* isolate.

Conclusion

Our study characterized a multidrug-resistant *E. chengduensis blaNDM-1*-ST414 strain, which shared the same phylogenetic origin with domestic isolates. These findings suggest the emergence of a rare and newly described ECC species in clinical settings, in addition to the frequently encountered *E. cloacae* and *E. hormaechei*, presenting new challenges for nosocomial infection prevention and control. Although this bacterial strain exhibited multidrug resistance, it remained sensitive to CFDC, AZA, AMK, and TGC. The detection of *E. chengduensis* in both human and environmental settings suggests that it exhibits metabolic plasticity and environmental resilience. To curb the rising trend of CRECC, surveillance should monitor resistance profiles and clonal transmission in clinical isolates while also tracking environmental strains to interrupt cross-niche transmission.

Declarations

Ethics approval and consent to participate

This study was reviewed and approved by the Ethics Review Committee of the Yanbian University Hospital (approval no. 20260013). Only ECC isolates identified during routine clinical diagnosis were used. Therefore, the requirement for informed consent was waived by the Ethics Committee in accordance with the “Ethical Review Measures for Biomedical Research Involving Humans” and the “Regulations of the People’s Republic of China on the Administration of Human Genetic Resources.” This study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The whole-genome sequence data of *E. chengduensis* strain YBHECC-C1 have been deposited in the NCBI database under BioSample accession number SAMN57088929https://dataview.ncbi.nlm.nih.gov/object/PRJNA1447783 or https://zenodo.org/records/21526462?token=eyJhbGciOiJIUzUxMiJ9.eyJpZCI6ImEzNGM4MDVlLWE1OTgtNDY1Mi1hMTNjLTc1NzMyNTFmMzgzYiIsImRhdGEiOnt9LCJyYW5kb20iOiJiYjZkMmM4MI Any qualified independent researcher can obtain the complete raw sequencing reads and assembled genome datasets by sending a formal reasonable request to the corresponding author.

Competing interests

The authors declare that they have no conflict of interest.

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Authors' contributions

XYZJ analyzed the characteristics of the data, constructed the prediction model, and drafted the manuscript. MJB enhanced the visual presentation of the figures. JX was involved in the experimental design. JCM reviewed the manuscript, and approved the submitted version.All authors read and approved the final manuscript.

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Figures

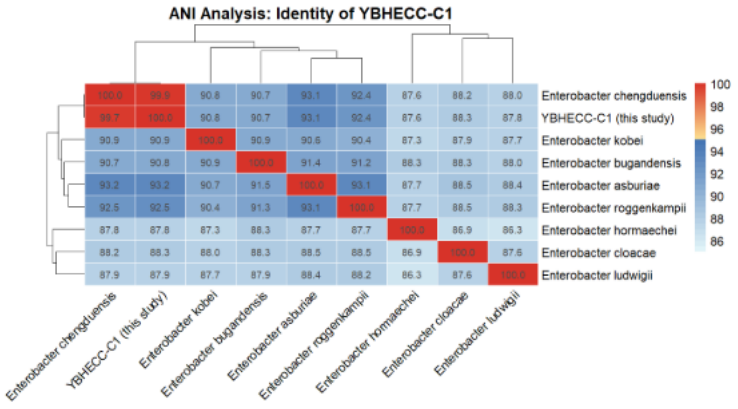


Figure 1

Average Nucleotide Identity (ANI) heatmap of strain YBHECC-C1 and other species of *Enterobacter cloacae complex*.

The color scale represents pairwise ANI percentage identity, ranging from blue (low similarity) to red (high similarity). The numbers within the cells indicate specific ANI values (%).

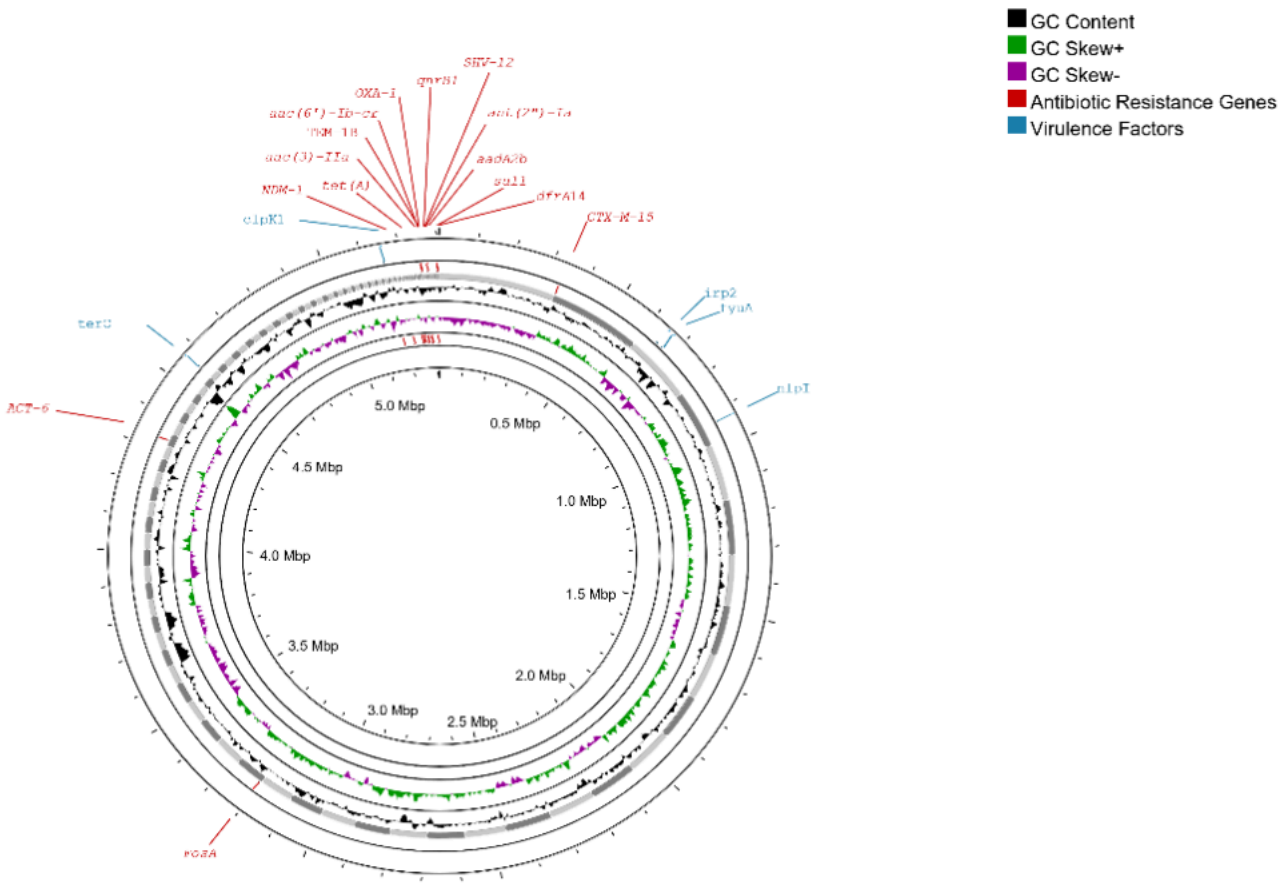


Figure 2

Identification of resistance genes and virulence factors in YBHECC-C1.

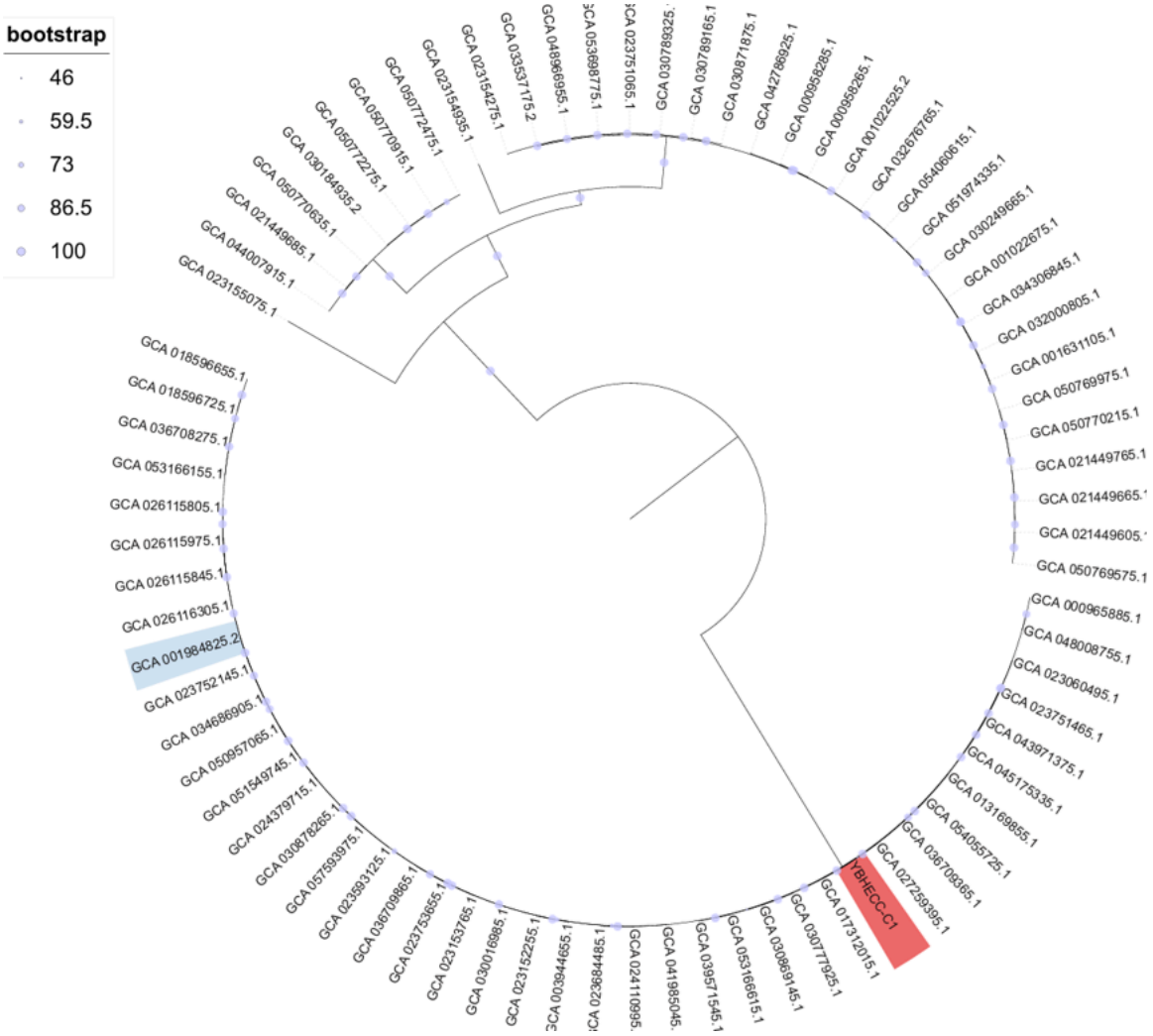


Figure 3

Intraspecific phylogenetic analysis of *E. chengduensis*

Supplementary Files

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- SupplementaryFig.1ReaddepthprofileoftheYBHECCC1mgrBlikelocus..png