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LETTER

Identification of a novel linezolid resistance gene, *cfr*(F), in *Riemerella anatipestifer*

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Highlights

- A novel linezolid resistance gene, *cfr*(F), was identified in *Riemerella anatipestifer*.
- *cfr*(F) confers cross-resistance to PhLOPS$_A$ antibiotics, including linezolid.
- *Riemerella anatipestifer* is a potential reservoir for linezolid resistance gene dissemination.

Riemerella anatipestifer is a facultative anaerobic Gram-negative bacterium that pose a significant threat to the waterfowl industry, causing significant economic losses worldwide (Tao *et al.* 2020). Due to the limited cross-protection between vaccines of different serotypes, antibiotic treatment is usually adopted for *R. anatipestifer* infection. However, multi-drug resistance limits the efficacy of antibiotics (Zhu *et al.* 2022). Bacterial ribosomes are one of the primary targets of antibiotics used in both human and veterinary medicine. Several rRNA-modifying enzymes associated with antibiotic resistance have been identified (Roberts *et al.* 1999). Among these, the free radical S-adenosyl-L-methionine (SAM) enzyme Cfr is particularly noteworthy. The Cfr proteins belongs to the free radical SAM enzyme family mediates the transfer of methyl groups to the unactivated C-H bonds. Cfr enzymes methylates the C2 and C8 carbon atoms on the A2503 residue, located in a functionally critical region of the 23S rRNA component, providing cross-resistance to phenicol (e.g., florfenicol), lincosamides (e.g., clindamycin), oxazolidinones (e.g., linezolid), pleuromutilins (e.g., thiocolistin), and streptogramin A (e.g., virginiamycin M1), resulting in the PhLOPS$_A$ phenotype (Long *et al.* 2006), In addition, Cfr-mediated methylation confers resistance to 16-membered macrolides, aminocyclitol hygromycin A, and nucleoside antibacterial agent A201A (Long *et al.* 2006; Smith and Mankin 2008; Polikanov *et al.* 2015). The *cfr* gene was first identified on a plasmid of *Staphylococcus sciuri* (Schwarz *et al.* 2000). Since then, nearly 20 different genetic backgrounds harboring this gene have been identified in isolates of *Enterococcus*, *Bacillus*, *Escherichia coli*, *Macrococcus caseolyticus*, *Pediococcus parvulus* and *Streptococcus suis* from Europe, Latin America, the United States and Asia (Roberts *et al.* 1999; Vester 2018). Homologous *cfr* genes have also been identified in *Proteus vulgaris*, *Escherichia*, and other Gram-negative bacteria (Shen *et al.* 2013; Liu X Q *et al.* 2017; Schwarz *et al.* 2021). Additionally, four *cfr*-like genes with less than 80% amino acid sequence identity to the *cfr* gene have been identified in *Clostridium*, *Enterococcus* spp. and *Clostridioides* (Roberts *et al.* 1999). According to the macrolide-lincosamide-streptogramin B (MLS) nomenclature system, maintained by Dr. Marilyn C. Roberts (http://faculty.washington.edu/marilynr/ermwebA.pdf) (Roberts *et al.* 1999), these genes are designated as *cfr*(B), *cfr*(C), *cfr*(D) and *cfr*(E).

During routine testing in our laboratory, we identified three strains of *R. anatipestifer* exhibiting a high resistance

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phenotype, despite being negative for the known florfenicol resistance genes. Among them, *R. anatipestifer* RCAD1242 (Appendix A), isolated from a sick duck in Henan Province, China, showed a typical $PhLOPS_A$ resistance phenotype, with high minimum inhibitory concentration (MIC) to florfenicol (32 mg L^{-1}), linezolid (8 mg L^{-1}), and clindamycin (256 mg L^{-1}). Genotypic analysis revealed the presence of several resistance genes, including *erm*(F), bla_{OXA}, and *tet*(X), but none could account for the observed high-level florfenicol resistance. This discrepancy prompted us to conduct whole-genome sequencing of this isolate. From the genomic data of *R. anatipestifer* RCAD1242, we identified a putative *cfr* gene and carried out experiments to assess its function. The putative *cfr* gene in RCAD1242 (locus_tag: PG338_009440) was identified using AMRfinder (Feldgarden *et al.* 2019) and the Comprehensive Antibiotic Resistance database (CARD, https://card.mcmaster.ca/) (Alcock *et al.* 2023). It is located at positions 2,020,066–2,021,157 bp on the RCAD1242 genome (GenBank accession number: CP168321.1) and encodes a protein of 363 amino acids (aa) (protein ID: XGJ26214). To characterize this protein, its amino acid sequence was aligned with other known Cfr-like enzymes using the Pairwise Sequence Alignment (PSA) tool provided by EMBOSS Needle (Madeira *et al.* 2024), and homology tree were constructed using DNAMAN software (version 8, Lynnon-Biosoft, Ontario, Canada) with default parameters. The results showed that the putative Cfr protein (XGJ26214) had the lowest similarity with Cfr(C) at 32.9% and the highest similarity with Cfr at 43.8%. The amino acid sequence identity fell below 79% with all other known Cfr sequences (Fig. 1-A and B), indicating that it represents a new *cfr*-like enzyme according to the macrolide-lincosamide-streptogramin B (MLS) nomenclature system (Roberts *et al.* 1999). This gene was designated *cfr*(F). This gene was also detected in two other *R. anatipestifer* strains (Appendix B). The susceptible *R. anatipestifer* ATCC 11845 (Appendix A) was used as the recipient strain in the natural transformation experiment; *Pasteurella multocida* DY120818 served as the exogenous expression strain. *Escherichia coli* ATCC 25922 was used as a quality control strain. The Cfr(F) protein belongs to the radical SAM enzyme family, known for a conserved cysteine-rich motif (CXXXCXXC) that coordinates a crucial iron-sulfur [4Fe-4S] cluster for catalytic activity. Our sequence alignment showed that in Cfr(F), residues C121, C125, and C128 form this critical motif (Appendix C). To confirm the functional importance of this putative catalytic site, we used seamless cloning to create three point mutants: C121A, C125A, and C128A. The deletion mutant RCAD1242Δ*cfr*(F) was constructed using the markerless deletion method (Liu M *et al.* 2017), while the complemented strain was generated *via* natural transformation (Appendix D). All aforementioned strains were subjected to MIC determination conducted in accordance with Clinical and Laboratory Standards Institute M100 guidelines (CLSI 2023). We determined MICs for several antibiotics, including florfenicol, chloramphenicol, clindamycin, linezolid, tiamulin, virginiamycin M1, these data were analyzed to assess whether the presence of *cfr*-like genes was associated with a $PhLOPS_A$ phenotype (Table 1). Strains containing pLMF03-*cfr*(F) showed elevated MICs compared to the control strain ATCC 11845-pLMF03. Specifically, the MICs for florfenicol (32 mg L^{-1}), chloramphenicol (16 mg L^{-1}), and clindamycin (256 mg L^{-1}) were 32-, 8-, and >1,024-fold higher, respectively, than those of the control strain. Additionally, the MIC for linezolid was 8 mg L^{-1} (8-fold higher), and for tiamulin was 4 mg L^{-1} (16-fold higher), and for virginiamycin M1 was >128 mg L^{-1} (>8-fold higher). The data also confirmed that *cfr*(F) confers the $PhLOPS_A$ phenotype in *P. multocida* DY120818. However, the introduction of *cfr*(F) did not confer any resistance to *E. coli* BL21(DE3). Detailed data are presented in Table 1. The MIC values for florfenicol, chloramphenicol, clindamycin, linezolid, tiamulin and virginiamycin M1 for the C121A, C125A and C128A mutants showed no significant difference from those of ATCC 11845-pLMF03 strain (Table 1). These findings suggest that the CXXXCXXC motif is essential for the enzymatic activity of Cfr(F) and its associated antibiotic resistance.

To investigate the prevalence of the *cfr*(F) gene in isolates, 573 *R. anatipestifer* isolates (collected from 1994 to 2022) kept in our laboratory were subjected to three PCR detection using the primers *cfr*(F)-F and *cfr*(F)-R (Appendix D). To further characterize the genetic context of *cfr*(F), we subjected three PCR-*cfr*-positive strains, including RCAD1242, to whole-genome sequencing (Yang *et al.* 2024). The samples were sequenced using the MGISEQ-2000 (BGI, Shenzhen, China) platform. Genome assembly was performed *de novo* using SPAdes (St. Petersburg genome assembler) (version 3.14.0, setting: -k auto -careful). The quality and completeness of the assembly were assessed using Quast (v5.2.0, default parameters), CheckM (v1.1.10, parameter: taxonomy wf), and BUSCO (v5.1.2, default parameters and flavobacteriales_odb10 database). Annotations were obtained with the NCBI Prokaryotic Genome Annotation Pipeline.

To characterize the distribution of *cfr*(F), a BLASTN search of *cfr*(F) sequences against the NR database (https://blast.ncbi.nlm.nih.gov/Blast.cgi) was performed on 22 April, 2025. The distribution analysis of the *cfr*(F) gene indicates that it exists in various habitats, two sequences were retrieved from the public database (Appendix E). We analyzed the 100 kb regions upstream and downstream of *cfr*(F) hits, which is considered sufficient to predict the majority of mobile genetic elements. We used VRprofile2 to detect known mobile genetic elements within these regions. The genetic structures of the *cfr*(F)-containing regions were compared and visualized using EasyFig v2.2.3. Notably, the presence of integrative and conjugative elements (ICE) and insertion sequences (IS) in *Sphingobacterium* sp. BN32 suggests the *cfr*(F) may be transferable *via* horizontal gene transfer, however, this gene has not been shown to be functional (Fig. 1-C). Previous studies have reported that *cfr* genes are mainly associated with plasmids and transposons (Schwarz *et al*. 2000; Deshpande *et al.* 2015; Tang *et al.* 2017; Stojković *et al.* 2019; Guerin *et al.* 2020). Furthermore, we identified several adjacent ARGs, including *erm*(F), $bla_{OXA-347}$, $bla_{OXA-209}$, bla_{OXA-10}, *tet*(X), *ere*(D), *est*T, and *lnu*(H). Notably, the *cfr*(F) gene was co-located with $bla_{OXA-347}$, $bla_{OXA-209}$, bla_{OXA-10}, *tet*(X), and *erm*(F) genes (Fig. 1-C), which are responsible for resistance

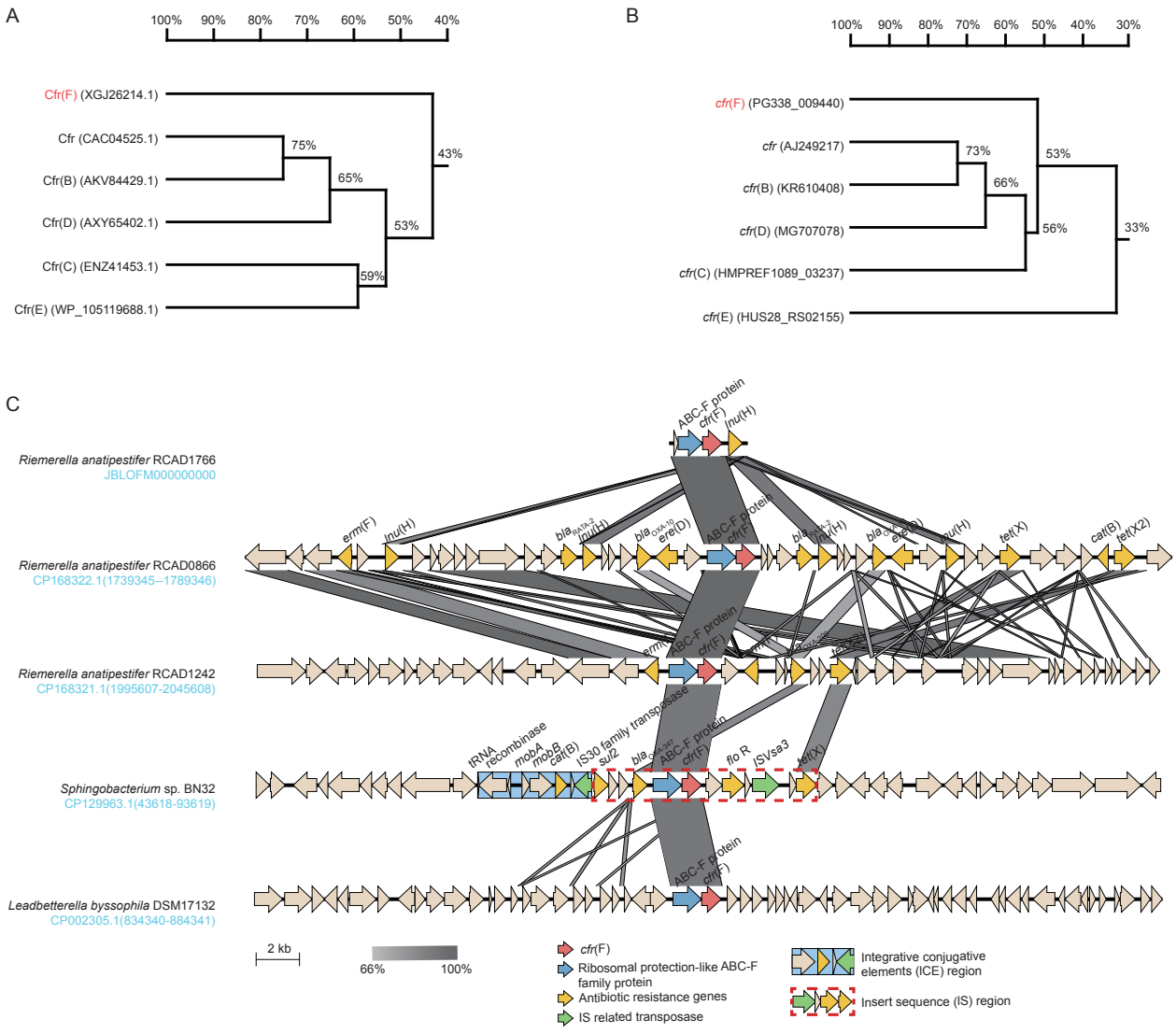


Fig. 1 Homology analysis and genetic context of the *cfr*(F) gene. A, homology tree of the *Cfr*(F) and representative Cfr proteins based on amino acid sequences. B, homology tree of the *cfr*(F) and representative *cfr* genes based on nucleotide sequences. C, genetic context of the *cfr*(F) gene, including associated resistance genes and mobile genetic elements.

to cefepime, tetracycline, erythromycin and lincomycin, respectively. While the PhLOPS$_A$ phenotype is primarily associated with Gram-positive bacteria (Schwarz *et al.* 2000; Deshpande *et al.* 2015; Candela *et al.* 2017; Stojković *et al.* 2019; Guerin *et al.* 2020), our study identified the *cfr*(F) gene in the Gram-negative pathogen *R. anatipestifer*. Although the intrinsic hydrophobic barrier of Gram-negative bacteria provides baseline protection against many antibacterial agents, the persistence of *cfr*(F) in *R. anatipestifer* is most likely driven by the extensive veterinary use of phenicols. Because *cfr*(F) confers resistance to both phenicols and oxazolidinones, the use of florfenicol in poultry may indirectly co-select for oxazolidinone resistance genes, even though linezolid is not applied in veterinary practice. This finding highlights the importance of ongoing surveillance to track the dissemination of the *cfr*(F) gene.

Further analysis of the genetic environment (100 kb) of the *cfr*(F) gene region in *R. anatipestifer* revealed no known mobile or transposable elements (Fig. 1-C). Previous genomic analyses have showed that *R. anatipestifer* harbors a variety of ARGs (Zhu *et al.* 2020; Luo *et al.* 2022). Some reports suggest that *R. anatipestifer* may serve as a natural host of the *tet*(X) gene, which is commonly found in this bacterium (Umar *et al.* 2021). Additionally, we detected *cfr*(F) in the genomes of two other clinical *R. anatipestifer* strains, RCAD0866 and RCAD1766 (Appendix B), and in one *Bergeyella* strain, RCAD1439. The corresponding GenBank accession numbers are CP168322.1, JBLOFM000000000, and JAYWXI000000000, respectively. Detailed information for these strains is provided in Appendices B and E. Although few plasmids have been reported in *R. anatipestifer*, and only small number carry antibiotic resistance genes. Plasmids are not the primary means by which ARGs are disseminated among *R. anatipestifer* isolates. Recent studies also suggest that *R. anatipestifer* may employ undiscovered mechanism to acquire ARGs (Umar *et al.* 2021). *Riemerella anatipestifer*

Table 1 *In vitro* transfer of plasmid-mediated *cfr*(F) gene

Strains	MIC (mg L^{-1})[1)]					
	FFC	CHL*	CLI	LZD	TIA	VM M1
Riemerella anatipestifer						
ATCC 11845	1	2	≤0.25	1	≤0.25	16
pLMF03	1	2	≤0.25	1	≤0.25	16
pLMF03-*cfr*(F)	32	16	256	8	4	>128
pLMF03-*cfr*(F)-C121A	1	1	≤0.25	1	≤0.25	32
pLMF03-*cfr*(F)-C125A	0.5	0.5	≤0.25	0.5	≤0.25	8
pLMF03-*cfr*(F)-C128A	0.5	0.5	≤0.25	0.5	≤0.25	32
RCAD1242	32	16	256	8	64	>128
RCAD1242Δ*cfr*(F)	1	4	2	1	32	16
RCAD1242::*cfr*(F)	32	16	128	8	64	128
Pasteurella multocida						
DY120818	0.5	0.5	64	4	4	64
pMC-Express	0.5	128	128	4	4	64
pMC-Express-*cfr*(F)	4	256	512	16	16	>128
Escherichia coli						
BL21	2	4	256	128	256	64
pET28a	2	2	128	64	128	64
pET28a-*cfr*(F)	4	4	256	64	256	64
ATCC 25922	8	8	128	>256	128	>32

[1)] FFC, florfenicol; CHL, chloramphenicol (* means chloramphenicol is the resistance marker for plasmid pMC-Express); CLI, clindamycin; LZD, linezolid; TIA, tiamulin; VM M1, virginiamycin M1.

is known for its unique natural transformation ability (Huang *et al.* 2021), which may facilitate the acquisition of ARGs from the environment, potentially contributing to the spread of resistance in this bacterium (Luo *et al.* 2022).

In conclusion, we have identified and characterized a novel gene, *cfr*(F), which confers *R. anatipestifer* with resistance to full $PhLOPS_A$ resistance profile. As this avian pathogen is widespread in poultry production and readily exchanges mobile genetic elements, it represents a potential reservoir from which linezolid resistance gene can spread to clinically important human bacteria. Our findings highlight the need for integrated One Health surveillance and prudent antimicrobial use to curb further dissemination of *cfr* mediated resistance along the animal-environment-human interfaces.

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Declaration of competing interest

The authors declare that they have no conffict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare that they did not use AI in the preparation and writing of this manuscript.

Ethical statements

This study did not involve any experiments on humans or live animals. All bacterial isolates were obtained from routine diagnostic samples, and no ethical approval was required.

Appendices associated with this paper are available at https://doi.org/10.1016/j.jia.2025.11.026

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