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MAJOR ARTICLE

First Report of a Fatal Septicemia Case Caused by *Vibrio metoecus*: A Comprehensive Functional and Genomic Study

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Background. In recent years, we have witnessed an unprecedented increase in the incidence of vibriosis due to global warming. *Vibrio metoecus* is a recently described *Vibrio cholerae*-like species that has not been associated with septicemia death in humans. During follow-up of human vibriosis, we received a blood isolate from a patient with secondary septicemia who died a few hours after admission.

Methods. Phenotypic and genotypic methods failed to identify the isolate, which could only be identified by average nucleotide identity after genome sequencing. The isolate was subjected to in vitro and ex vivo assays, complemented by comparative genomics focused on the identification of unique genetic traits. Strains and genomes from the same and related species (*V. cholerae* and *Vibrio mimicus*) were used for analyses.

Results. The isolate was the only one able to resist and multiply in human serum. Its genome contained virulence genes shared with *V. mimicus* and/or *V. cholerae*, with those associated with sialic acid degradation within pathogenicity island 2 standing out. However, it also presented a unique gene cluster, flanked by a transposase gene, putatively related to surface polysaccharide pseudosialyzation.

Conclusions. We document the first case of death caused by septicemia due to *V. metoecus* and propose that the acquisition of surface pseudosialyzation genes explains the ability of certain isolates of this species to survive in blood. Our discovery underscores the urgent need to monitor and study newly emerging pathogenic species, as climate change may be facilitating their spread and increasing the risk of serious infections in humans.

Keywords. *Vibrio metoecus*; global warming; septicemia; comparative genomics; serum resistance.

The *Vibrio* genus encompasses over 150 aquatic bacterial species thriving in diverse ecosystems, ranging from freshwater to marine environments. These bacteria are found in water both as free-swimming forms and associated with organic and inorganic particles, as well as various aquatic life forms [1]. Temperature fluctuations driven by global warming are dramatically altering the spatiotemporal distribution of these bacteria, leading to population blooms in warming coastal waters concomitant with a substantial increase in *Vibrio* infections [1, 2].

V. cholerae, native to warm aquatic environments, is particularly responsive to these climatic shifts, contributing to its prevalence in affected areas. Renowned as the primary agent behind cholera, *V. cholerae* instigates severe diarrheal outbreaks requiring stringent quarantine measures during epidemics and pandemics. Interestingly, only specific strains carrying the cholera toxin genes (TVc), transmitted by the CTX-Φ phage and belonging to serotypes O1 and O139, are responsible for these outbreaks [3, 4]. Other nontoxigenic strains (NTVc), while not cholera inducing, are increasing alarmingly [5]. They are implicated in sporadic human vibriosis cases, presenting symptoms ranging from diarrhea to more severe infections such as septicemia and necrotizing fasciitis [5].

Human vibriosis can also be caused by other *Vibrio* species, both closely and distantly related to *V. cholerae*, such as *Vibrio mimicus*, *Vibrio vulnificus*, and *Vibrio alginolyticus*. Of these, *V. vulnificus* is notably lethal, capable of causing death within 48 hours, particularly in patients with high blood iron levels [6]. Recently identified species like *Vibrio tarriae*, *Vibrio paracholerae*, and *Vibrio metoecus* add to this diversity, although information on their specific sources of infection, the forms of vibriosis they cause, and patient histories remains scarce.

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This complex variety of pathogens and associated diseases raises crucial questions about the connections between specific *Vibrio* species and the illnesses they induce. To address these questions, accurate identification of the pathogens involved is essential, followed by detailed reporting and genetic and phenotypic characterization of their virulence mechanisms compared to known *Vibrio* species.

This article discusses a fatal case of secondary septicemia caused by *V. metoecus* in Valencia (Spain). The strain was isolated from blood in pure culture and could only be correctly identified by genomic sequencing and average nucleotide identity (ANI) calculation with reference genomes. In vitro and ex vivo assays, together with comparative genomic analysis with *V. cholerae* and *V. mimicus*, revealed that this strain presented as unique features its iron-dependent ability to grow in human serum and the presence of a transposase-flanked gene cluster related to pseudosialyzation of surface polysaccharides. Consequently, our discovery underscores the urgent need to monitor and study newly emerging pathogenic species, as climate change may be facilitating not only their expansion but also that of virulence genes, further increasing the risk of serious infections in humans.

METHODS

Bacterial Strains and Growth Conditions

We received the clinical strain of *Vibrio* sp. as a pure culture on chocolate agar. To verify its ability to ferment sucrose, an isolated colony was inoculated onto thiosulphate, citrate, bile, and sucrose agar (TCBS; Condalab). For phenotypic comparative analyses, we included *V. metoecus* OP3H^T (type strain; isolated from an oyster pond in Falmouth, United States, in 2006), *V. mimicus* ATCC 33653^T (isolated from a human ear in North Carolina, United States, in 2006), *V. cholerae* CECT 652 (TVc, serotype O1, biotype El Tor, isolated from a human patient in 1980 in Louisiana, United States), and *V. cholerae* CECT8265 (NTVc, serotype non-O1/non-O139, isolated from human feces from a patient in the United Kingdom). All strains, except for *V. metoecus* OP3H^T, were derived from clinical samples. Routine culturing was performed in LB (Luria-Bertani broth), TSB (trypticase soy broth), and TSA (trypticase soy agar) from Condalab, at 28°C for 24 hours. Cultures were preserved in LB supplemented with 20% glycerol at −80°C.

Phenotypic and Genotypic Assays

Biochemical identification of the clinical *Vibrio* sp. strain was conducted using the analytical profile index (API) 20E system (BioMérieux). Genetic identification involved polymerase chain reaction (PCR) targeting *V. cholerae* and *V. vulnificus*-specific genes *toxR* and *vvhA*, respectively [7, 8]. The halotolerance of the isolates was determined in salt tolerance broth (STB) (tryptone 1% w/v + yeast extract 0.3% w/v + NaCl 0% to 8% w/v). Inoculated STB tubes were incubated at 28°C with constant shaking (110 rpm) for 48 hours. Growth was assessed by turbidity and confirmed by streaking on TSA plates. Growth kinetics for salt concentrations supporting growth (0% to 5.5% NaCl) were analyzed by culturing a selected colony in LB overnight, adjusting the absorbance to 0.5 at 625 nm, and then incubating diluted cultures in corresponding STB solutions at 28°C for 22 hours in a 96-well plate. Growth was measured as the absorbance at 625 nm.

Biofilm Formation Assay

Biofilm production was quantified after incubating the strains in 5 mL of STB 2% at 28°C for 24 hours. The biofilms were semi-quantified based on their thickness on the tube sides, treated with methanol, stained with crystal violet, and then dissolved in acetic acid for absorbance measurement at 560 nm.

Septicemic Potential Assay

To assess the septicemic capacity of the isolates and the dependence on iron, cultures in the stationary phase in LB were incubated with fresh human serum or iron-supplemented human serum as previously described [9]. Bacterial survival was quantified by counting colonies on TSA plates before and after incubation at 37°C for 4 hours.

Genomic Analysis

DNA was extracted using the GeneElute Bacterial Genomic DNA Kit (Sigma) and sequenced on Illumina Miseq and MinION Nanopore platforms. Libraries were prepared using the Nextera XT DNA Library Prep Kit for Illumina and the Oxford Nanopore PCR Barcoding Kit for MinION.

Bioinformatics Analyses

For bioinformatics, default settings were applied unless noted otherwise. We first filtered Illumina reads for a mean quality score of 20 using Prinseq [10]. This was followed by a hybrid assembly combining both Illumina and MinION reads via the Unicycler pipeline set to bold mode [11]. Assembly quality was visually inspected with Bandage [12], and completeness evaluated with BUSCO [13]. Assembly statistics were generated using Quast [14].

For comparative analyses of the clinical *Vibrio sp.* strain isolated in this study, we used the *V. mimicus* genome GCA_008464965.1 (ATCC 33654^T strain, USA, 1980), and the *V. metoecus* genome GCA_000696385.1 (OP3H^T strain, USA, 2006) from the National Center for Biotechnology Information (https://www.ncbi.nlm.nih.gov/). Due to the unavailability of the other genomes used in phenotypic tests, we utilized the TVc O1-Eltor genome GCA_003063785.1 (seventh pandemic-N16961 strain, Bangladesh, 1975) along with the *V. metoecus* genome GCF_009665275.1 and the NTVc O45 genome GCA_023168185.1.

Genomic Identification and Annotation

We calculated the ANI between the *Vibrio* sp. genome and each reference using the ANI calculator web tool [15], with a similarity of 95% or greater confirming species identity [16].

To decode the virulence mechanisms of the *Vibrio* sp. strain, we searched for mobile genetic elements like prophages and plasmids using geNomad [17]. The genome was annotated with Prokka [18] and Bakta [19], targeting virulence genes from *V. cholerae* and *V. mimicus* within specific pathogenicity islands and phage CTX-Φ. Genes for the O-antigen and capsular polysaccharide synthesis (CPS) locus were sourced from a previous study [20] (GenBank accession DQ915177), and their presence verified using Proteinortho6 [21] and hmmersearch [22]. We followed the modified methodology of a previous study [23] for annotating and assessing genes in other strains using Proteinortho.

RESULTS

In 2016, a 73-year-old man with a history of type 2 diabetes mellitus, congestive heart failure, chronic obstructive pulmonary disease, cor pulmonale, and complete arrhythmia due to atrial fibrillation treated with Sintrom was admitted to the intensive care unit with acute pain in the right lower limb, suggestive of cellulitis or fasciitis. A physical examination revealed an erythematous plaque with intense pain upon palpation and movement. The patient underwent emergency surgery for debridement with sepsis, but the immediate postoperative course was poor, marked by refractory septic shock and eventual multiorgan failure leading to death. He was treated with antibiotics including amikacin, meropenem, and linezolid.

Species Identification

The bacterium was isolated in pure culture from blood and identified as *Vibrio* sp. by matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF). It formed yellow colonies on TCBS agar, resembling those of *V. cholerae* control strains. Initial biochemical identification using the API 20E system yielded an ambiguous result, prompting further analysis. PCR tests for *V. cholerae* and *V. vulnificus* were negative. The isolate's genome was sequenced using a hybrid approach of Illumina and MinION, revealing a close genomic identity with *V. metoecus* (97.7%), distinct from *V. cholerae* and *V. mimicus* (Supplementary Table 1). It was named *V. metoecus* RIP1, from the Latin *requiescat in pace*.

In Vitro and Ex Vivo Assays

Salt tolerance assays showed that all strains grew without salt and tolerated up to 5% NaCl (Table 1), with optimal growth observed at 2% (Supplementary Figures 1–6). Upon comparing the growth across all tested salinities for each strain, it was determined that 2% NaCl provided the optimal growth conditions for all strains (Supplementary Figures 1–6). At 0% NaCl, the NTVc strain reached the stationary phase sooner (as shown in Supplementary Figure 6*A*). However, at 2% NaCl, no significant differences were observed among the strains, except for TVc, which demonstrated a lower absorbance level (Supplementary Figure 6*B*).

Biofilm production also varied with salinity; *V. metoecus* and *V. mimicus* produced significantly more biofilm up to 4.5%–5% salt, peaking at 2% (Supplementary Figure 7, and Table 1 and Table 2). ANOVA confirmed that strains of both species produced significantly more biofilm than *V. cholerae* strains. Tukey test classified the strains into 3 categories: high producers (*V. metoecus* and *V. mimicus* strains), medium producer (NTVc strain), and low producer (TVc strain) (Supplementary Figure 6*B*). Additionally, ex vivo assays showed that *V. metoecus* RIP1 was the only strain that survived in human serum (5% survival in human serum), with dramatically increased survival when iron was supplemented (300 fold). The main differences among the tested strains revealed by the in vitro and ex vitro assays are shown in Table 2.

Table 1. Growth and Biofilm Formation by *Vibrio* Strains at Different NaCl Concentrations and Incubation Times

	NaCl Concentration, %													
	0		2		4		4.5		5		5.5		6	
Strain	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h	G[a]	Bf[b] 24/48 h
TVc	+	–/–	+	–/–	+	–/–	+	–/–	+	–/–	+	–/–	+	–/–
NTVc	+	–/+	+	+/+++	+	+/++	+	–/+	+	–/+	+	–/–	–	–/–
RIP1	+	++/++	+	+++/+++	+	+/++	+	–/++	+	–/+	–	–/–	–	–/–
V. metoecus[T]	+	++/++	+	+++/+++	+	+/++	+	–/++	+	–/+	+	–/–	+	–/–
V. mimicus[T]	+	++/++	+	++/++	+	+/+	+	+/+	+	+/+	+	–/+	+	–/–

Growth in salinities up to 8% were also measured but were negative for all strains. Thus, they were not tested for biofilm production.

Abbreviations: Bf, biofilm formation; G, growth; T, type strain.

[a]Growth was assessed after 48 hours and confirmed by plating 10 µL of the culture (– no growth, + growth).

[b]The strains were incubated in salt tolerance broth, the grown broth was discarded and the thickness of the mark on the sides of the tube was measured (–, no biofilm; +, small white stains; ++, clear white mark around the media of less than 0.2 cm, +++, more than 0.3 cm).

Table 2. Main Phenotypic Differences Among the Tested Strains

Phenotype	TVc	NTVc	RIP1	*V. metoecus*^{T}	*V. mimicus*^{T}
Growth at > 5% NaCl	Yes	Yes	No	Yes	Yes
Biofilm formation at 2% NaCl	Light	Moderate	Strong	Strong	Strong
Resistance to human serum with Fe	No	No	Yes	No	No

Abbreviations: NTVc, non-toxigenic *V. cholerae* strain; T, type strain; TVc, *V. cholerae* strain with cholera toxin gene.

Table 3. Presence of Several Virulence Genes in the Studied Genomes

	V. metoecus			*V. cholerae*		
Gene/Gene Cluster	RIP1	OP3H^T	08-2459	N16961	O45	*V. mimicus* ATCC33653^T
Toxins and exoenzymes						
hlyA (hemolysin A, VCC)	+	+	+	+	+	+
rtxA (repeat-in-toxin A)	–	–	–	+	+	–
ctxA (*V. cholerae* cytotoxin)	–	–	–	+	–	–
hap (hemagglutinin protease)	+	+	+	+	+	–
prt (collagenase)	+	+	+	+	+	–
Adhesion						
gbpA (*N*-acetylglucosamine-binding protein)	+	+	+	+	+	+
MSHA (mannose-sensitive hemagglutinin) pilus	+	+	+	+	+	+
Type IV pilus	+	+	+	+	+	+
Tcp (toxin-coregulated-pilus)	–	–	–	+	–	–
Secretion systems						
T2SS	+	+	+	+	+	+
T6SS general cluster	+	+	+	+	+	+
T6SS auxiliar clusters 1 and 2	+	+	+	+	+	+
T6SSb auxiliar cluster 3	+	–	–	–	+	–
Surface antigens						
O-antigen biosynthesis and export	+	+	+	+	+	+
Capsule biosynthesis and export	+	+	+	+	+	+
Exopolysaccharide biosynthesis and export	+	+	+	+	+	+
Iron uptake						
Vibriobactin production	–	–	–	+	+	–
Vibriobactin utilization	–	–	–	+	+	–
Aerobactin production	+	+	+	–	–	+
Aerobactin utilization	+	+	+	–	–	+
hemR (hemin receptor)	+	–	+	+	+	–

Genomic Features of RIP1

De novo assembly of the hybrid genome of the RIP1 strain yielded 6 contigs with a total of 4 022 012 bp, 5 corresponding to chromosome 1 (chromosome 1, contigs 1,3–6), and 1 (contig 2) to chromosome 2, the latter obtained as a circular contig. The L50 statistic (number of contigs whose length sum makes up half of genome size) was 1, the length of that contig (N50) was 2 636 067 bp (Supplementary Table 2), and the total number of detected of coding sequences was 3553, all of them chromosomal. The only mobile genetic element found was a 10 652-bp putative prophage located on chromosome 2 that was taxonomically assigned to the family *Inoviridae* (Supplementary Table 3).

The main identified virulence genes and genomic islands are shown in Table 3, Figure 1, Figure 2, and Supplementary Material 1 and 2. We highlight the following factors.

Pathogenic Islands and Islets. All genomes, except NTVc, shared part of the *Vibrio* pathogenicity island 2 (VPI-2), in particular the *nan-nag* region located on islet B, which encodes sialic acid metabolism [24] (Supplementary Material 2). The *V. cholerae* and *V. metoecus* genomes also shared part of islet D.

Prophages. The prophage detected in the RIP1 genome of *V. metoecus* was not found in any of the other genomes, meaning that although it belongs to the same family as the CTX-Φ phage, they are different phages.

Adhesion Factors. All analyzed genomes shared genes for biogenesis of an MSHA (mannose-sensitive hemagglutinin) pilus (Table 3) and part of a *PilA*-like pilus, while those of *V. cholerae*

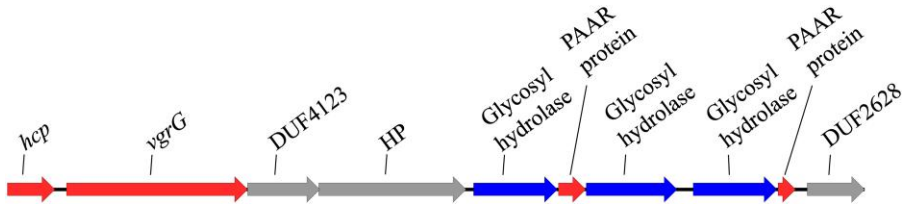

Figure 1. Type 6 secretion system (T6SS) auxiliar cluster in *V. metoecus* RIP1 and *V. cholerae* O45. The auxiliar cluster contains 10 genes (represented as arrows). Red, genes relate to T6SS; blue, glycosyl hydrolases; grey, genes with unknown functions.

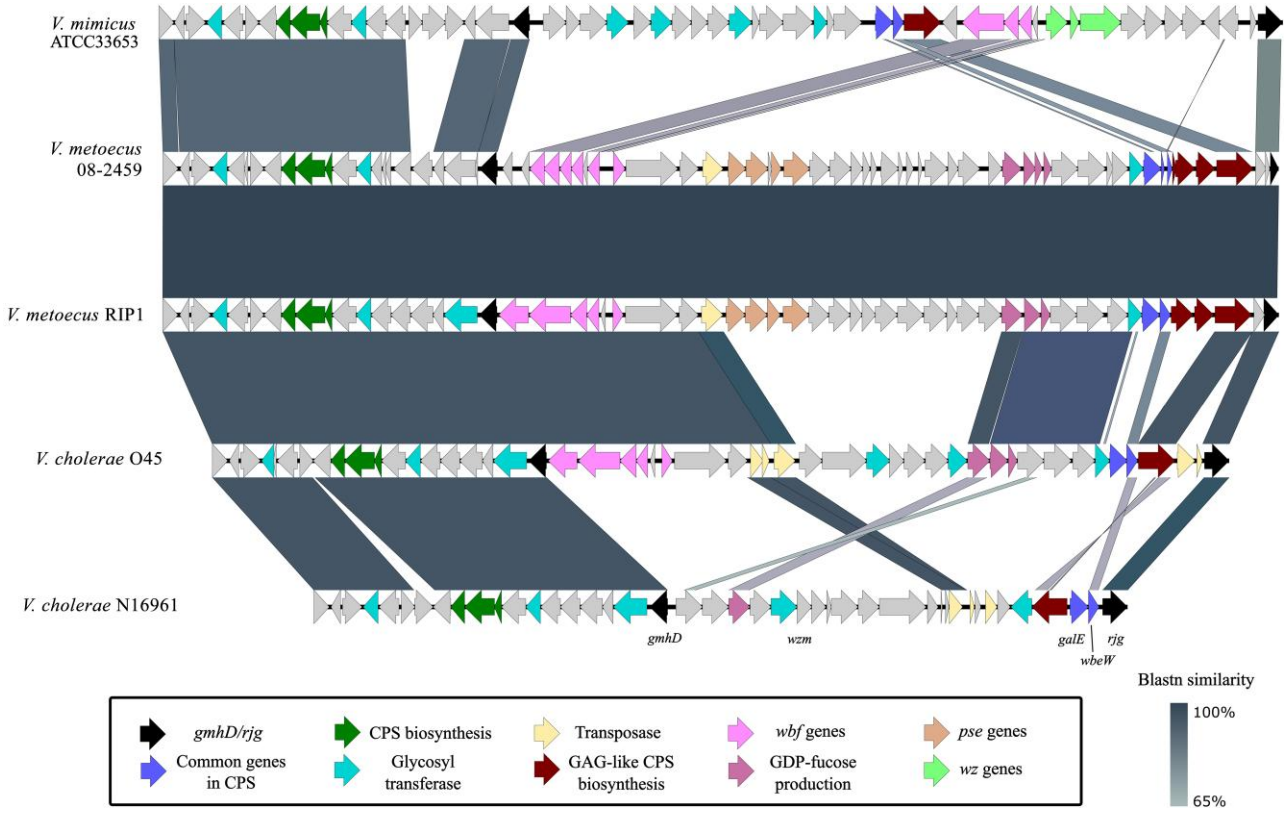

Figure 2. Capsular polysaccharide (CPS) locus and O-antigen regions comparison. The genes were annotated according to Chen et al [20].

contained genes for an additional type IV pilus, the toxin-coregulated pilus (*tcp* genes) encoded in VPI-1 (Supplementary Material 2).

Siderophores. The genomes of *V. metoecus* and *V. mimicus* also shared genes for the synthesis and transport of the siderophore aerobactin, while those of *V. cholerae* contained genes for the synthesis and transport of the siderophore vibriobactin. Notably, only the genomes of clinical isolates of *V. metoecus* and *V. cholerae* contained a common gene for a hemin receptor.

Secretion Systems. All genomes presented highly homologous gene clusters for a T2SS (type 2 secretion system) and a T6SS-1. Interestingly, an additional T6SS cluster was found only in *V. metoecus* RIP1 and *V. cholerae* O45 strains. This new set is located in chromosome 1 and comprises 10 genes, including 2 effectors for the T6SS (*hcp* and *vgrG*), 2 proline-alanine-alanine-arginine (PAAR) proteins, and 3 glycosyl hydrolases (Figure 1).

Capsule. All genomes contained a *vps* locus (*Vibrio* [exo]polysaccharide synthesis) but differed in the Ag-O/capsule biosynthetic locus (Figure 2). We distinguished 2 regions: the consensus one according to a previous study [20] (region A), which is delimited by the *gmhD* and *rjg* genes, and an additional upstream region (region B). Region B showed high homology and contained genes for hypothetical proteins, 3

capsular synthesis proteins (*cps* genes), and 3 glycosyltransferases. Region A contained 3 genes described as necessary for *V. cholerae* capsule biosynthesis [20], the *galE*, *wbeW*, and *wzm* genes, the first 2 present in all genomes and the third only in *V. cholerae* genomes. Additionally, it contained genes for enzymes that generate guanosine diphosphate fucose (GDP-fucose) from GDP-mannose [25], an unusual sugar in *Vibrio* envelopes, which were only present in the genomes of *V. metoecus* and NTVc. *V. mimicus* exclusively harbored the *wza*, *wzb*, and *wzc* genes, present in the capsular locus of certain serotypes of *V. vulnificus* [26]. The *wbf* genes, responsible for the change from O1 to O139 antigen in *V. cholerae* [27], were present in all genomes except TVc. Notably, a complete cluster of genes, flanked by a transposase for enzymes involved in pseudaminic acid (Pse) biosynthesis (*pse* genes) was only present in *V. metoecus* genomes.

DISCUSSION

In this article, we present a fatal case of human vibriosis and analyze the causative agent to understand how it caused the patient's death. The patient was diabetic and elderly and died within hours of admission. Initial clinical evaluations suggested vibriosis caused by *V. vulnificus*, but identification by MALDI-TOF pointed to an unknown *Vibrio* species. Subsequent laboratory analyses revealed that the isolate formed the typical yellow colonies of *V. cholerae* on TCBS agar and not the green ones of *V. vulnificus*, so we suspected that it might be an atypical *V. cholerae*. Then, phenotypic (API 20E) and genotypic identification (PCR) confirmed that it was neither *V. cholerae* nor *V. vulnificus*. ANI determination after genome sequencing showed that the isolate belonged to a newly emerging species very close to *V. cholerae*, that is *V. metoecus* [28]. For this reason, we included strains and genomes of *V. cholerae*, and *V. mimicus* (species very close to *V. metoecus*) and *V. metoecus* as references in the subsequent study.

V. metoecus and *V. cholerae* can coexist in similar environments, but the former is less abundant or more difficult to isolate by conventional methods [29]. Our salt tolerance and biofilm formation studies revealed that all species tested could grow and form biofilms at salinities between 0% and 5%, with optimal growth at 2%. However, *V. metoecus* stands out for forming significantly thicker biofilms at lower salinities, indicating that it is particularly adapted to sessile life in freshwater ecosystems. This adaptation is crucial, as *V. metoecus* has been isolated from the mucosal surfaces of fish in low-salinity waters close to the Mediterranean Sea near Valencia [30], where increasing temperatures may influence the incidence of vibriosis, an issue currently not included in official records in Europe.

Genomic comparisons revealed that *V. metoecus* does not possess unique genes for toxins or adhesion factors, including biofilm formation [31, 32]. In fact, all vibrios analyzed share genes for several toxins, for the biosynthesis and transport of exopolysaccharides (putatively involved in biofilm production), and for the biogenesis of MSHA and PilA pili (putatively involved in both specific adhesion and the initial steps of biofilm formation). In addition, all strains except NTVc harbor genes for sialic acid catabolism. These genes are located in a genomic islet similar to that described as islet B in the VPI-2 pathogenicity island of some *V. cholerae* strains [33]. Sialic acid is mainly found on the surface of epithelial and immune cells [34, 35] so that if the bacterium can degrade it and use it as a nutrient, it will colonize the tissue more efficiently. Furthermore, by removing sialic acid from host cells, the bacterium can alter host defenses, evade immune detection, and expose other receptors for adhesion, facilitating more efficient colonization and infection [34, 35]. Of note, this islet is flanked in *V. metoecus* RIP1 by a transposase gene, providing indirect evidence of horizontal gene transfer.

In contrast, *V. metoecus* is notable for having unique genes relative to reference strains/genomes for iron uptake and protection from the immune system, which would facilitate its systemic spread. The 2 clinical *V. metoecus* strains, but not the environmental strain, share genes for aerobactin biosynthesis and use, a heme receptor, and biosynthesis of pseudaminic acid (*pse* genes), which potentially favor its growth in blood. Aerobactin-related genes have been previously identified in *V. mimicus* [36] and in strains of *V. vulnificus*, where the presence of aerobactin and other siderophores was associated with 100-fold higher lethality than in environmental strains lacking these systems [37]. Pseudaminic acid is a sugar similar to sialic acid. The biosynthetic cluster is located within the locus for lipopolysaccharide and CPS biosynthesis, suggesting that clinical *V. metoecus* strains can pseudosialylate their surface. Sialic acid is also involved in self-recognition, and the presence of this sugar on surfaces renders the bacterium invisible to innate defenses. This same system has been previously described in other human pathogens of concern, such as *Campylobacter jejuni* [38]. Again, this cluster appears in *V. metoecus*, flanked by a transposase gene, providing further evidence of horizontal gene transfer.

Phenotypic growth assays in serum confirmed that the isolated bacterium resists human complement, especially under conditions of iron excess, an ability it shares with *V. vulnificus*, which also shows increased septicemic power in patients with iron overload [6]. Interestingly, the patient's diabetes, not elevated iron levels, was the hypothesized factor that predisposed him to fatal sepsis, highlighting the complex interactions between host factors and bacterial virulence.

Finally, the genome of our strain presents a second T6SS system, which could be involved in fighting immune system cells and DNA uptake, collaborating not only in septicemia but also in acquiring beneficial genes (such as those found in the study) from other bacteria with which it shares habitat [39, 40].

CONCLUSION

This detailed investigation of the pathogenic mechanisms of *V. metoecus* reveals, for the first time, a sophisticated strategy of immune evasion and environmental adaptation, implicating it as an important emerging pathogen. Notably, our findings suggest that the strategies for survival and growth in blood, such as surface pseudosialylation, have likely been acquired through horizontal gene transfer, further enhancing the pathogenic potential of *V. metoecus*. Understanding the pathogenic mechanisms of noncholeric vibrios is crucial in a scenario where global warming is spreading them worldwide. The proximity of the source of infection to the patient and the identification of environmental conditions conducive to *V. metoecus* proliferation suggest possible preventive measures and call for further research on the virulence and epidemiology of this pathogen, which could inform future public health strategies and clinical management of vibriosis. Our discovery also underscores the urgent need to monitor newly emerging pathogenic species, as climate change may be facilitating not only their spread but also that of virulence genes, further increasing the risk of severe human infections.

Supplementary Data

Supplementary materials are available at *The Journal of Infectious Diseases* online (http://jid.oxfordjournals.org/). Supplementary materials consist of data provided by the author that are published to benefit the reader. The posted materials are not copyedited. The contents of all supplementary data are the sole responsibility of the authors. Questions or messages regarding errors should be addressed to the author.

Notes

Data availability. This whole-genome shotgun project has been deposited at DDBJ/ENA/GenBank under the accession number JBANQT000000000. The versions described in this paper for RIP1 is the first version. This sample was collected under the BioProject accession number PRJNA1080516.

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