(*3*), including our case, have identified the presence of worms in the colon. This unique location of echinostome flukes in humans might be a distinguishing feature for *E. cinetorchis* infection.

Freshwater snails are first as well as second intermediate hosts for *E. cinetorchis* (*1*). Large-sized snail species in particular (e.g., *Cipangopaludina*) and freshwater fish are potential sources of human infections. Our patient reported that she had sold snails and freshwater fish on the street and often consumed them raw or undercooked. Thus, the infection source for our patient might have been 1 or both kinds of intermediate hosts.

In countries where human echinostome infections are found, physicians should include echinostomiasis among the differential diagnoses of diseases causing nonspecific gastrointestinal problems. Public education regarding the hazards associated with consuming raw or undercooked snails or fish in these regions also would be useful in reducing *E. cinetorchis* infections.

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Vibrio mimicus Lineage Carrying Cholera Toxin and *Vibrio* Pathogenicity Island, United States and China

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Vibrio mimicus bacteria have caused sporadic cases and outbreaks of cholera-like diarrhea throughout the world, but the association of lineages with such events is unexplored. Genomic analyses revealed *V. mimicus* lineages carrying the virulence factors cholera toxin and toxin co-regulated pilus, one of which has persisted for decades in China and the United States.

Vibrio mimicus bacteria are native to aquatic environments but have the potential to cause diseases in animals and humans, such as gastroenteritis

and cholera-like diarrhea (*1*). In *Vibrio cholera* bacteria, the main toxigenic factor is cholera toxin (CTX), which is encoded by *ctx*A and *ctx*B genes and is part of the bacteriophage CTXΦ. The acquisition of CTXΦ phage by *V. cholerae* bacteria was associated with the toxin coregulated pilus (TCP), which is involved in intestinal colonization and aggregation. This virulence factor is encoded in an operon in the *Vibrio* pathogenicity island 1 (VPI-1), and *tcp*A is the main structural subunit of that pilus. In addition to TCP, VPI-1 also harbors the *acf*A-D operon, which also plays a role in colonization (*2*).

In 2004, the largest documented foodborne outbreak of *V. mimicus* occurred in Thailand, in which 306 persons experienced symptoms including diarrhea, abdominal pain, and vomiting (*3*), but the virulome associated with these strains was not verified. In 2010, in the United States, a cluster of severe diarrheal diseases was caused by *V. mimicus* strains carrying CTX (*4*). In 2019, *V. mimicus* bacteria caused a seafood-associated outbreak in Florida (USA), in which the patients experienced severe diarrhea, although the strains were CTX-negative (*5*). However, the virulome of most genomes analyzed (n = 33) was not explored, leaving a gap regarding the association of the strains or lineages with virulence factors. To fill this gap, we analyzed 44 *V. mimicus* genomes, 35 from GenBank and 9 environmental genomes from Brazil and Japan that we sequenced by using an Illumina Hiseq 2500, assembled by using SPAdes 3.15.2 (https://github.com/ablab/spades), and then analyzed by using Abricate (http://github.com/tseemann/abricate) and the Comprehensive Antibiotic Resistance Database (https://card.mcmaster.ca) and the

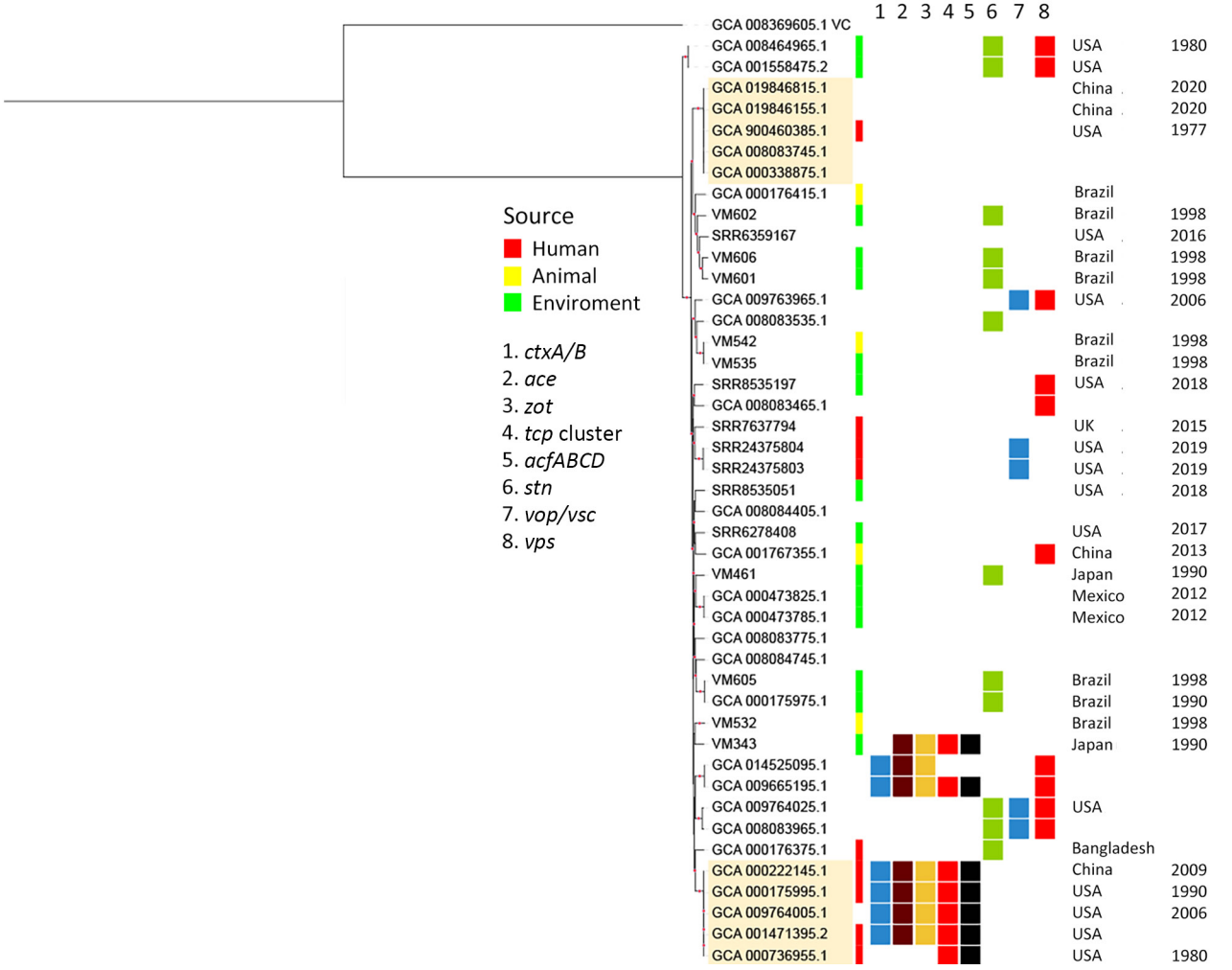

Figure. Maximum-likelihood phylogenetic tree of 44 *Vibrio mimicus* bacteria genomes from the United States and China. The best evolutionary model (general time reversible plus base frequencies plus ascertainment bias correction plus FreeRate model with 8 categories) was selected on the basis of the Bayesian information criterion. The beige highlighted clusters represent the main ones sharing genomes from China and the United States. Red circles on branches represent >70% bootstrap.

Virulence Factor Database (http://www.mgc.ac.cn/VFs/main.htm).

The phylogenetic analysis based on the core genome revealed clusters; the 2 main clusters had genomes that had been circulating in China and the United States for decades (Figure). These 2 clusters are characterized by distinct virulomes; 1 co-harbored *ctx*A, *ctx*B, *ace*, *zot*, TCP, and *acf*A-D (mainly clinical genomes), whereas the other did not have any of those genes. The lineage carrying those virulence genes has persisted for ≥3 decades (1980–2009), infecting persons in China and the United States. The other lineage, which lacks these virulence factors, also was identified in China (2020) and the United States (1977 [human source]). Another interesting cluster of genomes is the one that covers Brazil (1998 [animal source]) and the United States (2016 [environmental source]).

Our analysis indicates that *V. mimicus* lineages are disseminated and persist in distinct sources in space and time. In addition, another set of 3 related genomes (VM343, GCA_014525095.1, and GCA_009665195.1) also possessed CTX, TCP, or both, which suggests loss or partial acquisition of the pathogenicity islands of these elements. Of note, other genomes belonging to the same lineage (Figure) appear to have acquired the *ctx*A or *ctx*B genes from different sources; GCA_000175995.1, GCA_001471395.2, and GCA_009764005.1 (United States) possessed the *ctx*B2 genotype (El Tor [Australia]), whereas GCA_000222145.1 (China) had the *ctx*B1 genotype (classical [strain 569B]). Regarding the TCP cluster, analyses in blastn (https://blast.ncbi.nlm.nih.gov) revealed that all *tcp*A sequences, except for VM343, were identical and differed from the classical and El Tor genotypes. The *tcp*A allele carried by most genomes has also been characterized in nonpandemic clinical *V. cholerae* strains from the United States (*6*), whereas the *tcp*A allele of VM343 (Japan [environmental source]) is unique.

We identified several other virulence genes in the genomes (Appendix, https://wwwnc.cdc.gov/EID/article/30/8/24-0252-App1.pdf). We highlight the presence of the heat-stable enterotoxin gene (NAG-ST), identified throughout the phylogeny, and gene clusters related to exopolysaccharide production (*vps*) and the type III secretion system (T3SS) (*vop*, *vsc*, and *vcr*). We identified T3SS only in genomes that did not carry CTX, TCP, or both, including those from the 2019 outbreak in Florida (*5*), but 2 closely related genomes (1 of which was identified in the United States) co-carried the T3SS and NAG-ST genes. Because T3SS is a syringe-like protein secretion apparatus, the co-occurrence of this system with a diarrhea-associated toxin could increase the pathogenicity of these strains.

Our findings show that *V. mimicus* strains are spread throughout the world and that some of them carry a virulome comparable to that of *V. cholerae* bacteria. The virulome of environmental and clinical strains is apparently not heterogeneous (*5*), even with the analysis of the new environmental genomes. However, those findings may represent just the tip of the iceberg, given the bias regarding the locality of available genomes. Therefore, more genomic data must be generated to determine whether specialized clinical strains of *V. mimicus* exist, as they do for *V. cholerae* bacteria. Furthermore, the environmental and clinical genomes possessed a set of common virulence genes, suggesting that environmental strains have the potential to cause disease in humans. Because *V. mimicus* already possesses an intrinsic virulome, with the potential to cause disease, the acquisition of virulence determinants such as CTX, TCP, or both could specialize certain lineages, as revealed in our analysis by the clinical lineages that carry these determinants.

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The *Vibrio mimicus* whole-genome sequences from this study were deposited in GenBank under the accession nos. JAZHPO000000000 (VM343), JAZHPP000000000 (VM461), JAZHPQ000000000 (VM532), JBAKBZ000000000 (VM535), JAZHPR000000000 (VM542), JBAKBY000000000 (VM601), JBAKBX000000000 (VM602), JBAKBW000000000 (VM605), and JAZHPS000000000 (VM606).

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Fecal Microbiota Transplantation for Severe Infant Botulism, China

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Infant botulism in a 4-month-old boy in China who continued to excrete toxins for over a month despite antitoxin therapy was further treated with fecal microbiota transplantation. After treatment, we noted increased gut microbial diversity and altered fecal metabolites, which may help reduce intestinal pH and enhance anti-inflammatory capabilities.

Infant botulism is caused by ingesting *Clostridium botulinum* spores and characterized by symmetric descending paralysis, which can progress to respiratory failure in severe cases (*1*). Specific therapies include intravenous administration of botulism immune globulin (BIG-IV or BabyBIG, https://infantbotulism.org/general/babybig.php) or botulinum antitoxin. However, BIG-IV and BabyBIG are unavailable in some countries, including China (*2*). Even after clinical signs have been alleviated through antitoxin therapy, some children may continue to excrete *C. botulinum* and its neurotoxin (BoNT) in their feces over a prolonged period, heightening the potential for relapse and transmission to others (although relatively rare) (*3*,*4*). Hence, effective treatments that promote clearance of intestinal *C. botulinum* spores are needed. During March–May 2021, we treated severe infant botulism in a 4-month-old boy in China who had continued excreting toxins for >1 month after clinical signs disappeared after antitoxin therapy. The study was approved by the Ethics Committee of Beijing Children's Hospital (2023-E-149-R).

Five days before admission to Beijing Children's Hospital (Beijing, China), the previously healthy infant exhibited intermittent fever, lethargy, poor appetite, and constipation, followed by respiratory distress. After intubation and mechanical ventilation in the emergency department, the patient was admitted to the pediatric intensive care unit. During examination, his pupils were dilated (≈4 mm) and had sluggish light reflexes but no signs of meningeal irritation. In addition, his muscle strength and tone were low. A series of tests excluded central nervous system infections, metabolic disorders, and other potential causes. By day 3 of hospitalization, the diagnosis of infant botulism was confirmed by detection of BoNT nucleic acid (serotype B) in fecal samples. Subsequently, we were able to obtain botulism antitoxin (monovalent type B) and administer it by intravenous injection of 2 mL (5,000 IU) 2×/day. Substantial improvement in clinical signs was observed by day 7 of hospitalization, and complete resolution was achieved by day 15.

Nevertheless, through day 33, multiple fecal samples tested for botulinum nucleic acid by real-time