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Vibrio parahaemolyticus Serovar O3:K6 as Cause of Unusually High Incidence of Food-Borne Disease Outbreaks in Taiwan from 1996 to 1999

CHIEN-SHUN CHIOU,[1]* SHIH-YUAN HSU,[2] SHIOU-ING CHIU,[3] TIEN-KUEI WANG,[3] AND CHENG-SHUN CHAO[4]

The Third Branch Office, Center for Disease Control, Taichung City 403,[1] The Fourth Branch Office, Center for Disease Control, Kaohsiung City 813,[2] The Division of Bacterial Diseases, Center for Disease Control, Taipei 115,[3] and The Sixth Branch Office, Center for Disease Control, Hualien 970,[4] Taiwan

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The occurrence of food-borne disease outbreaks in Taiwan increased dramatically in 1996, and the incidence has since remained elevated. This increase in outbreaks is correlated with a high rate of isolation of *Vibrio parahaemolyticus*, which caused between 61 and 71% of the total outbreaks for the period 1996 to 1999. By serotyping, 40 serovars were identified from 3743 *V. parahaemolyticus* isolates, of which O3:K6 was the most frequently detected. The O3:K6 serovar could have emerged in Taiwan as early as October 1995 and at that time accounted for only 0.6% of the *V. parahaemolyticus* infections. This level increased suddenly to 50.1% in 1996 and reached a peak (83.8%) in 1997. Comparison of the outbreak profiles for the etiology groups indicates that the high incidence of food-borne disease outbreaks during 1996 to 1999 can be attributed to the extraordinarily high O3:K6 infections. In 1999, the O3:K6 serovar was still prevalent, and accounted for 61.3% of all *V. parahaemolyticus* infections. Due to its extraordinarily high infection frequency and its capability to spread globally, this organism needs to be intensively monitored internationally.

Vibrio parahaemolyticus is an important etiologic agent of seafood-borne gastroenteritis and has become a leading cause of food-borne disease outbreaks in Taiwan and Japan (9, 12). In Taiwan, food-borne disease data have been systematically collected by the Department of Health since 1986, with an average of 85 outbreaks per annum having been recorded during the period from 1986 to 1995 (12). From this surveillance, we noticed that the incidence of food-borne disease outbreaks increased suddenly in 1996 and has remained high since this time. In addition, we have observed a correlation between this increase in outbreaks and a high isolation rate for *V. parahaemolyticus*. In this study, we investigated the causes of this increase by analyzing the etiologic agents responsible for food-borne disease outbreaks occurring in Taiwan from 1995 to 1999.

Specimens such as stool, rectal swab, and nose swab were collected by local health authorities from patients and food handlers involved in the food-borne disease outbreaks and sent to the laboratories of the Center for Disease Control, where bacterial pathogen examinations were conducted. Briefly, the specimens were streaked in selective differential media described for *Vibrio cholerae* and *V. parahaemolyticus* (7), *Salmonella* spp. (16), *Staphylococcus aureus* (5), *Bacillus cereus* (13), *Shigella* spp. (14), *Aeromonas* spp. (15), and *Escherichia coli* O157:H7 (6). Suspected colonies were initially identified by biochemical tests with triple sugar iron agar (Eiken Chemical Co., Tokyo, Japan), SIM medium (Eiken), and lysine iron agar (Difco Laboratories, Detroit, Mich.). For screening *E. coli* O157:H7, suspected *E. coli* colonies were tested with Clig agar slants (Kyokuto, Tokyo, Japan) instead of triple sugar iron agar. After the screening tests, suspected isolates were further identified with API biochemical test kits (bioMérieux, l'Etoile, France), i.e., API 20E for *V. cholerae, V. parahaemolyticus, Salmonella* spp., *Shigella* spp., and *Aeromonas* spp., API ID32 Staph for *S. aureus*, and API 50CH for *B. cereus*. Serologic tests for identification and serotyping were performed routinely with the antisera of O antigens and H antigens for *E. coli* O157:H7 (Difco) and *Salmonella* spp. (Denka, Seiken Corp., Tokyo, Japan), the antisera of O1 and O139 antigens for *V. cholerae* (Denka), the antisera of O antigens for *Shigella* spp. (Denka), the antisera of K antigens for *V. parahaemolyticus* (Denka), and the antisera of enterotoxins (SEA to SEE) for *S. aureus* (Denka). Some isolates of serovars K6, K8, K10, K12, K63, and K68 of *V. parahaemolyticus* were also serotyped with antisera of O antigens (Denka). Laboratory procedures of the Centers for Disease Control and Prevention, Atlanta, Ga., were followed for examination of *Clostridium botulinum* (2).

Epidemiological data relating to the food-borne disease outbreaks investigated were obtained from standardized case report forms filled in by the county public health authorities. These reports included the dates, times, and locations of suspected food ingestion, the suspected foods, the persons who consumed the food and were poisoned, and basic information about each patient's sex, age, date and time of onset, residency, clinical symptoms, and medical treatment.

The etiology of a food-borne disease outbreak was confirmed based on comparisons of the clinical symptoms, disease incubation period, implicated foods, and laboratory findings from the specimens from the implicated persons.

Table 1 presents the etiologies of food-borne disease outbreaks occurring in Taiwan during the period from 1995 to 1999. A total of 850 outbreaks were reported, and specimens from these outbreaks were examined by the Center for Disease Control. Bacterial agents were responsible for 610 outbreaks (71.8%), with the remainder (28.2%) due to chemical or unknown agents. Among the bacterial agents, *V. parahaemolyti-*

* Corresponding author. Mailing address: The Third Branch Office, Center for Disease Control, 4F 103 Minchuan Rd., Taichung City 403, Taiwan. Phone: 886-4-2225196. Fax: 886-4-2221917. E-mail: nipmcsc@cdc.gov.tw.

TABLE 1. Etiologies of food-borne disease outbreaks occurring in Taiwan from 1995 to 1999

Etiology	No. of outbreaks (%) in (yr):					Total no. of outbreaks (%)
	1995	1996	1997	1998	1999	
Bacterial agents[a]	72 (59.5)	130 (73.4)	185 (76.4)	122 (73.9)	101 (69.7)	610 (71.8)
Vibrio parahaemolyticus	54 (44.6)	119 (67.2)	171 (70.7)	110 (66.7)	88 (60.7)	542 (63.8)
Salmonella spp.	8 (6.6)	10 (5.6)	7 (2.9)	8 (4.8)	11 (7.6)	44 (5.2)
Staphylococcus aureus	7 (5.8)	2 (1.1)	6 (2.5)	4 (2.4)	2 (1.4)	21 (2.5)
Bacillus cereus	1 (0.8)	0 (0.0)	1 (0.4)	3 (1.8)	1 (0.7)	6 (0.7)
Vibrio cholerae[b]	4 (3.3)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	5 (0.6)
Shigella sonnei[c]	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	1 (0.1)
Aeromonas sp.	0 (0.0)	1 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.1)
Chemical and unknown agents	49 (40.5)	47 (26.6)	57 (23.6)	43 (26.1)	44 (30.3)	240 (28.2)
Total outbreaks	121	177	242	165	145	850

[a] All specimens were examined for the bacterial pathogens *Vibrio* spp., *Salmonella* spp., *S. aureus, B. aureus, Shigella* spp., *E. coli* O157:H7, and *Aeromonas* spp.
[b] Non-O1, non-O139 strains.
[c] Imported case.

cus, Salmonella spp., and *S. aureus* accounted for 63.8, 5.2, and 2.5% of the total outbreaks, respectively. *B. cereus, V. cholerae* (non-O1, non-O139 strains), *S. sonnei*, and *Aeromonas* sp. combined caused only 1.5% (13) of all bacterial outbreaks. The outbreak caused by *S. sonnei* was imported. No *E. coli* O157:H7 and *C. botulinum* outbreaks were identified during this period.

Compared to 1995, outbreaks increased abruptly from 121 to 177 cases in 1996 and have remained high since this time. The highest incidence of disease outbreaks was in 1997, with a total of 242 outbreaks occurring. The number of outbreaks due to chemical and unknown agents continued to remain steady, ranging from 43 to 57, but outbreaks caused by bacterial agents fluctuated from 72 to 185. This fluctuation in bacterial outbreaks is related to the number of *V. parahaemolyticus* outbreaks. Outbreaks caused by this agent range from 54 (44.6%) to 171 (70.7%) over the period from 1995 to 1999. In contrast, six other bacterial agents, including *Salmonella* spp. and *S. aureus*, caused 13 to 20 outbreaks. These data indicate that the high incidence of food-borne disease outbreaks during 1996 through 1999 can be attributed to *V. parahaemolyticus*.

To investigate whether a particular *V. parahaemolyticus* serovar was involved in the sudden increase in food-borne disease outbreaks, the K serotypes of the recovered *V. parahaemolyticus* isolates were analyzed. Furthermore, some isolates of several major K serovars were also serotyped with antisera of O antigens. Table 2 presents the prevalence of *V. parahaemolyticus* K serovars in food-borne disease outbreaks in Taiwan from 1995 to 1999 (inclusive). A total of 3,743 *V. parahaemolyticus* isolates were recovered and serotyped. Of these, 3,729 isolates were grouped into 40 K serovars and 14 isolates were nontypeable. K6 (O3:K6), K12 (O4:K12), K8 (O4:K8), K10 (O4:K10), and K63 (O3:K63) were ranked as the leading serovars, accounting for 82.6% of the total *V. parahaemolyticus* isolates. The O3:K6 serovar was the most prevalent over this time period. There were 2,234 isolates of O3:K6 identified in 351 of the 542 *V. parahaemolyticus* outbreaks over the period investigated. In 1995, O3:K6 was identified in a single outbreak with three isolates recovered, accounting for 0.6% of the *V. parahaemolyticus* identified in that year. However, in 1996, 372 O3:K6 isolates were identified in 66 outbreaks, accounting for 50.1% of the *V. parahaemolyticus*. The highest incidence of O3:K6 occurred in 1997 and accounted for 83.8% of the total *V. parahaemlyticus* isolates detected. The incidence of O3:K6 infections decreased in 1998, but this serovar still accounted for 71.5% of the *V. parahaemolyticus* and 61.3% in 1999. These data indicate that the prevalence of O3:K6 is correlated with the increase in total *V. parahaemolyticus* outbreaks in Tawian.

Serovars O4:K8 and O4:K10 caused 107 (19%) outbreaks over the study period. The number of these outbreaks caused by these two serovars showed no significant change during these 5 years. An O4:K68 serovar appeared for the first time in 1997 and had caused 22 outbreaks by 1999. This serovar is rarely implicated in food poisoning, but it had caused six outbreaks in Japan in 1998 (9). The future trend of *V. parahaemolyticus* O4:K68 infection must be carefully watched.

To determine the cause of the high incidence of food-borne disease outbreaks, we compared the outbreak profiles of the classified groups. That is, we compared the yearly total outbreaks with the agent responsible for that outbreak. The agents were classified into etiological groups, either chemical and unknown agents, or bacterial agents (*V. parahaemolyticus*, non-*V. parahaemolyticus*, O3:K6 , and non-O3:K6). Figure 1 depicts the profiles of the total outbreaks during the study period and also of the six etiology groups. The outbreak profiles of bacterial agents, *V. parahaemolyticus*, and O3:K6 coincide with the total outbreaks. In contrast, the outbreak profiles for all other groups showed no correlation with the total outbreak profiles. This profile analysis indicates that the O3:K6 *V. parahaemolyticus* was the cause of the unusually high incidence of food-borne disease outbreaks over the period from 1996 to 1999.

Figure 2 depicts the numbers of monthly food-borne disease outbreaks caused by the *V. parahaemolyticus* O3:K6 serovar, mixed serovars (consisting of the O3:K6 serovar and other non-O3:K6 serovars), and non-O3:K6 serovars and compares these with the average temperature in Taiwan. *V. parahaemolyticus* outbreaks were high between May and September for each year investigated. However, frequent outbreaks caused by *V. parahaemolyticus* were also reported in April and October. The occurrence of *V. parahaemolyticus* food-borne disease outbreaks can be correlated to temperature. Outbreaks tend to be more prevalent in late spring to early autumn and less prevalent in the cooler months of winter. Figure 2 also shows that the first *V. parahaemolyticus* O3:K6 serovar was identified in October 1995. The same serovar was identified again in April 1996 and has become more prevalent over time.

This study has investigated the causes behind a high incidence of food-borne disease outbreaks from 1996 to 1999 in Taiwan by analyzing the possible etiologic agents. We present data indicating that a single bacterial agent, *V. parahaemolyticus*, has caused 61 to 71% of the total food-borne disease

TABLE 2. Prevalence of the *V. parahaemolyticus* K serovars in food-borne disease outbreaks in Taiwan from 1995 to 1999

Serovar	No. of isolates (no. of outbreaks) in (yr):					Total no. of isolates (no. of outbreaks)
	1995	1996	1997	1998	1999	
K3	4 (1)	1 (1)	8 (3)	6 (2)	7 (2)	26 (9)
K4		9 (4)	19 (5)	8 (4)	2 (2)	38 (15)
K5	1 (1)		6 (2)		1 (1)	8 (4)
K6	3 (1)	372 (66)	990 (146)	523 (75)	346 (63)	2234 (351)
K7	1 (1)	13 (6)		1 (1)	1 (1)	16 (9)
K8	62 (12)	59 (20)	57 (11)	16 (8)	55 (11)	249 (62)
K9	4 (2)	13 (4)	6 (3)	6 (3)	6 (2)	35 (14)
K10	11 (4)	23 (2)	7 (5)	92 (20)	80 (14)	213 (45)
K11	3 (2)	3 (1)			8 (2)	14 (5)
K12	264 (11)	7 (4)	8 (2)	5 (2)		284 (19)
K13	2 (2)		2 (1)			4 (3)
K15	22 (5)	20 (6)	2 (2)	29 (2)	2 (2)	75 (17)
K17	1 (1)				1 (1)	2 (2)
K18	31 (3)	1 (1)	1 (1)			33 (5)
K19		20 (7)				20 (7)
K22	12 (3)	18 (1)	1 (1)			31 (5)
K25				5 (2)	4 (2)	9 (4)
K28					1 (1)	1 (1)
K29	13 (4)	12 (5)	3 (1)	2 (1)	4 (1)	34 (12)
K32		2 (1)				2 (1)
K33		1 (1)				1 (1)
K37			4 (2)	1 (1)		5 (3)
K38	2 (1)	26 (9)	2 (2)		9 (2)	39 (14)
K41	23 (7)	24 (7)	3 (2)	9 (2)		59 (18)
K44			1 (1)			1 (1)
K45				1 (1)		1 (1)
K46			1 (1)			1 (1)
K48			1 (1)	4 (4)	1 (1)	6 (6)
K53			3 (3)	1 (1)	1 (1)	5 (5)
K54		1 (1)	7 (2)			8 (3)
K55	1 (1)	15 (5)	2 (2)			18 (8)
K56	32 (4)	26 (12)	14 (7)	2 (2)		74 (25)
K57			2 (1)	1 (1)		3 (2)
K58	12 (1)					12 (1)
K59				1 (1)		1 (1)
K60			3 (1)			3 (1)
K63	18 (6)	72 (18)	8 (2)	1 (1)	13 (2)	112 (29)
K66			1 (1)			1 (1)
K68			11 (3)	17 (10)	18 (9)	46 (22)
K69	3 (2)	2 (1)				5 (3)
Nontypeable		2 (1)	8 (1)		4 (2)	14 (4)
Total	526 (54)	742 (119)	1,181 (171)	731 (110)	564 (88)	3,743 (542)
%K6	0.6 (1.9)	50.1 (55.5)	83.8 (85.4)	71.5 (68.2)	61.3 (71.6)	59.7 (64.8)

outbreaks in Taiwan during a 5-year period. By using the technique of outbreak profile analysis based on etiology groups, we have demonstrated that infections with a single *V. parahaemolyticus* serovar, O3:K6, were responsible for the unusually high incidence of food-borne disease from 1996 to 1999.

Infections caused by *V. parahaemolyticus* are usually associated with diverse serovars, but this situation has altered in recent years. Okuda et al. (11) first reported that a new strain of *V. parahaemolyticus* (O3:K6 serovar) had emerged. This serovar first emerged in February 1996 in Calcutta, India, and was responsible for 50 to 80% of the *V. parahaemolyticus* infections during the surveillance period of February to August 1996. Soon, it appeared that the emergence of this new serovar was a pandemic. The serovar appeared in several Asian countries, including Taiwan, Bangladesh, Laos, Japan, Korea, and Thailand (8), as well as in North America (3, 4). In Japan, O3:K6 has been identified as the major serovar in *V. parahaemolyticus* food-borne disease outbreaks in recent years (9). This strain has recently also caused two outbreaks associated with the consumption of raw seafood in the United States (3, 4). In Taiwan, the O3:K6 serovar may have appeared as early as October 1995, and the incidence has increased since April 1996 (Fig. 2). Our data show that this serovar has caused 50.1 to 83.8% of the *V. parahaemolyticus* infections during the 1996-to-1999 period, in contrast to only 0.6% in 1995. In 1999, the O3:K6 serovar still accounted for 61.3% of the *V. parahaemolyticus* infections in Taiwan, indicating that the number of infections with this serovar is still high. The O3:K6 serovar is currently dominant in the Asian region and has also appeared on the North American continent (3, 4). The origins of this serovar are unknown, and it may have spread to other regions of the world. This suggests that an intensive monitoring program for this organism is needed globally to monitor its spread and help understand its origins.

The genome of numerous O3:K6 isolates has been studied by several molecular techniques, including the arbitrarily

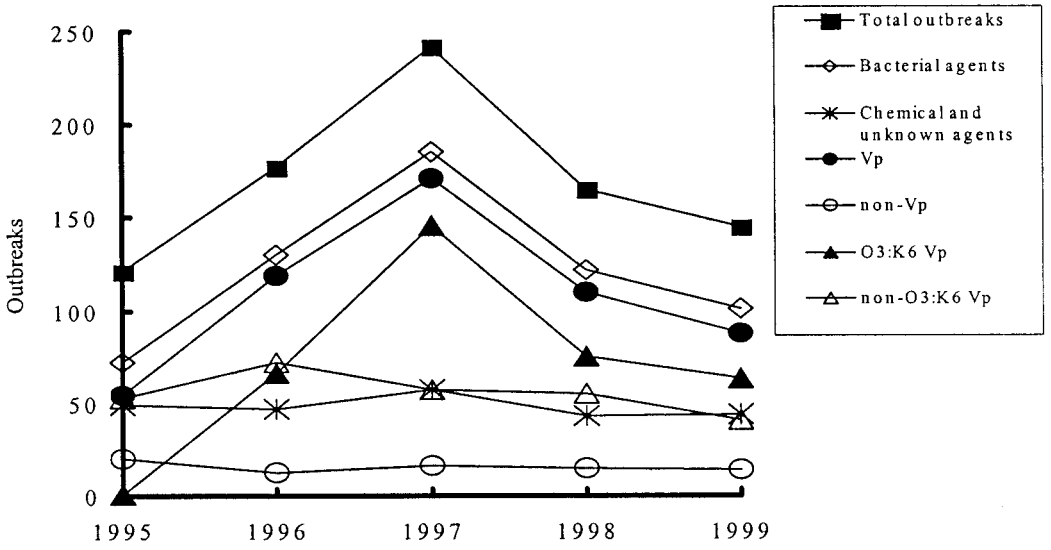

FIG. 1. Outbreak profiles of the total outbreaks and the six etiology groups from 1995 to 1999. The etiology groups include bacterial agents, chemical and unknown agents, *V. parahaemolyticus* (Vp), non-*V. parahaemolyticus* (non-Vp) (consisting of *Salmonella* spp., *S. aureus*, *B. cereus*, *V. cholerae*, *S. sonnei*, and *Aeromonas* sp.), O3:K6 V. *parahaemolyticus* (O3:K6 Vp), and non-O3:K6 *V. parahaemolyticus* (non-O3:K6 Vp).

primed PCR method (8, 11), ribotyping (1), pulsed-field gel electrophoresis analysis (PFGE; 1, 17), and *tox*RS sequence analysis (8). These studies conclude that the O3:K6 strain which emerged recently in Asia and the United States actually belongs to a single clone. However, Bag et al. (1) reported that a certain degree of genomic reassortment has occurred among the O3:K6 strains. Molecular typing of O3:K6 isolates recovered in Taiwan and several other Asian countries has recently been conducted by Wong and colleagues (17). From this study, eight different but genetically closely related PFGE pulsotypes were identified. In accordance with other studies (1, 8), Wong et al. demonstrated that the newest O3:K6 strains which caused pandemics in many Asian countries, including Taiwan, originated from a common ancestor with minor genomic changes.

The factors which influenced the emergence of the O3:K6 serovar are of great scientific interest. Two recent studies show that, compared to other O3:K6 strains isolated before 1996 and compared to non-O3:K6 reference strains, the recent O3:K6 strains show no significant differences in the levels of thermostable direct hemolysin, antibiotic susceptibility, and survival rate under the same environmental stresses (i.e., extreme temperatures, low pH, and high salinity) (11, 17). Recently, Nasu et al. (10) found that these newly emerged O3:K6 strains have

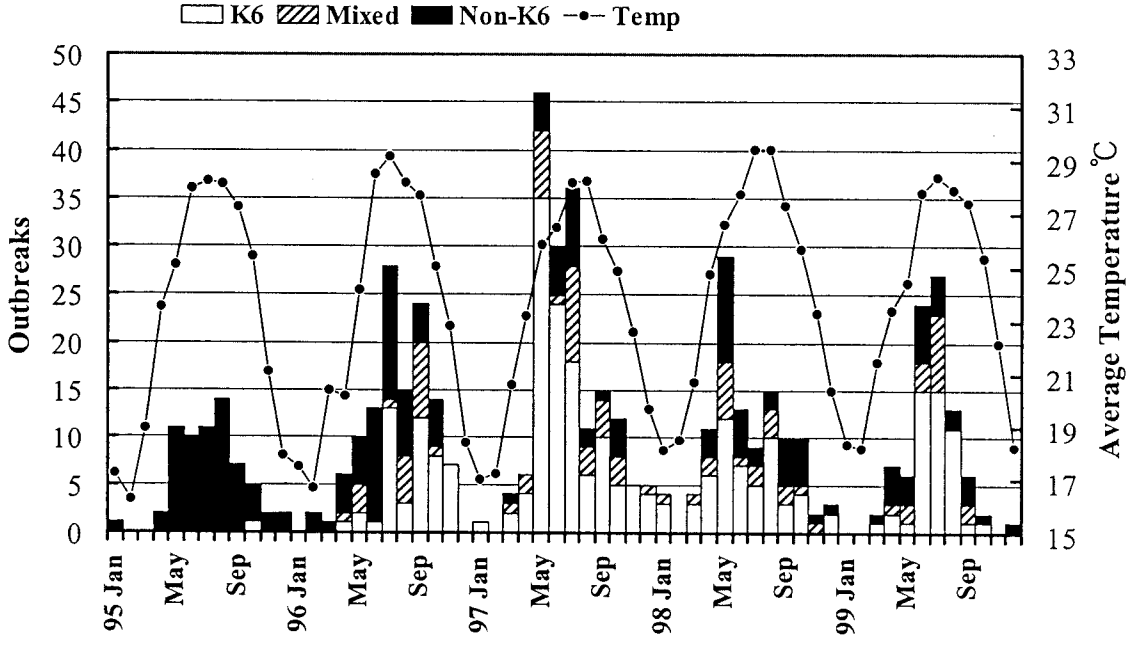

FIG. 2. Monthly food-borne outbreaks caused by *V. parahaemolyticus* O3:K6 serovar, mixed serovars (consisting of O3:K6 serovar and other non-O3:K6 serovars), and non-O3:K6 serovars versus the average temperature in Taiwan from 1995 to 1999.

a filamentous phage (f237) in common. The genome of f237 contains 10 open reading frames (ORFs). The ORF8 encodes a protein with a similar combination of motifs to a *Drosophila* adhesive protein encoded by *plx* (18). These authors point out that if ORF8 encodes a protein that has an adhesive function, the recent *V. parahaemolyticus* O3:K6 strains that possess ORF8 could be more adhesive, possibly adhering to host intestine cells or to the surface of marine plankton. This adhesiveness could account for the high potency of *V. parahaemolyticus* O3:K6 infection. Indeed, this adhesiveness hypothesis could account for the high incidence of O3:K6, but other possibilities, such as alternative virulence factor(s), competitive capability with other *V. parahaemolyticus* strains, and its population distribution in the natural environment, also need to be investigated.

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