Vibrio parahaemolyticus and *Vibrio vulnificus* in South America: water, seafood and human infections

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Summary

The bacterial species, *Vibrio parahaemolyticus* and *Vibrio vulnificus*, are ubiquitous in estuaries and coastal waters throughout the world, but they also happen to be important human pathogens. They are concentrated by filter-feeding shellfish which are often consumed raw or undercooked, providing an important potential route of entry for an infective dose of these bacteria. *Vibrio parahaemolyticus* can cause abdominal cramping, nausea, diarrhoea, vomiting, chills and fever. *Vibrio vulnificus* can cause similar gastrointestinal-related symptoms, but can also spread to the bloodstream, resulting in primary septicaemia, and it can also cause disease via wound infections. The objective of this article is to summarize, for the first time, the incidence and importance of *V. parahaemolyticus* and *V. vulnificus* in South America, in environmental waters and seafood, especifically molluscan shellfish, as well as human infection cases and outbreaks. It appears that infections from *V. parahaemolyticus* have been more strongly related to shellfish ingestion and have been more frequently reported on the Pacific coast of South America. Conversely, *V. vulnificus* has been more frequently acquired by water contact with open wounds and its presence has been more heavily reported along the Atlantic coast of South America, and while documented to cause serious mortality, have been relatively few in number. The impacts of El Nino Southern Oscillation (ENSO) have been observed to cause an increase in *V. parahaemolyticus* outbreaks on the Pacific coast of South America. The implementation of a regulated monitoring approach, along with the use of faster, more accurate and virulence-specific detection approaches, such as PCR confirmation, should be considered to detect the presence of pathogenic *Vibrio* strains in environmental and seafood samples for protection of public health. Furthermore, improved clinical surveillance with suspected cases should be implemented. This review highlights the need for more research and monitoring of vibrios in South America, in water, shellfish and clinical samples.

Introduction

The proportion of foodborne disease derived from consumption of raw and undercooked seafood worldwide is considerable, reaching above 80 000 illnesses, 500 hospitalizations and 100 deaths each year in the United States (Altekruse *et al.* 2000; Iwamoto *et al.* 2010; Westrell *et al.* 2010; Schaeffer *et al.* 2013). While bacterial, viral, algal and parasitic pathogens, particularly those stemming from faecal contamination, can contribute to seafood-borne illness, the pathogenic bacteria in the genus *Vibrio* have been garnering some recent headlines, as outbreaks and infections caused by these marine bacteria are increasing in number (Baker-Austin *et al.* 2012; Martínez-Urtaza *et al.* 2013), especially for raw and undercooked seafood.

In the United States, foodborne *Vibrio* infections are on the rise, even when numbers of infections from other foodborne bacterial pathogens are decreasing (CDC 2013a). The CDC produces an annual food safety report, which contains updates about foodborne infections including those caused by *Vibrio*. The most recent report presents some dour statistics. Most strikingly, the frequency rate of foodborne *Vibrio* infection, recorded during the 2013–2014 period has increased 173% as compared to the previous decade (CDC 2013a). Alarmingly, this trend is not restricted to the US. The geographic areas for which *Vibrio* diseases are being reported is expanding, even to locations unaccustomed to these infections, a phenomenon most likely brought about by warming ocean temperatures (Martínez-Urtaza *et al.* 2010; Baker-Austin et al. 2012; Levy 2015).

Vibrio spp. are ubiquitous along estuaries and coastal waters throughout the world (Urakawa and Rivera 2006). While the majority of these bacteria are harmless, several species can potentially infect humans or other animals and cause serious disease (Colwell 2005). Of those, *Vibrio cholerae*, *Vibrio vulnificus* and *Vibrio parahaemolyticus* are the most important pathogens. The pathogens, *V. vulnificus* and *V. parahaemolyticus*, can cause waterborne diseases, but are particularly dangerous when combined with a filter-feeding vector, such as molluscan shellfish. Filter-feeding molluscs pump the surrounding water over their gills, simultaneously obtaining oxygen and food. *Vibrio* spp. are often found attached to particles and as these particulates are passed over the sieve-like gills of filter-feeding molluscs, they are strained out of the water and retained (Ward and Shumway 2004; Froelich *et al.* 2013). This filtration ultimately can concentrate the number of *V. vulnificus* and *V. parahaemolyticus* in shellfish up to 100-fold of that found in the overlaying water (DePaola *et al.* 2003). Because some seafoods, especially oysters, are commonly eaten raw or only lightly cooked, this can provide a route of entry for a significant dose of live, potentially pathogenic *Vibrio* bacteria. *Vibrio* pathogens can also be associated with other seafood, such as shrimp, fish, clams, mussels or octopus (Oliver *et al.* 1983; Normanno *et al.* 2006; Yamamoto *et al.* 2008; García *et al.* 2009; Rodgers *et al.* 2014; Rodríguez-Camacho *et al.* 2014). In the United States, even though *Vibrio* diseases are reportable to the CDC, there are a large number of unreported cases, and approx. 84 000 Americans are estimated to contract a foodborne *Vibrio* infection every year (CDC 2013b).

Vibrio parahaemolyticus infections cause symptoms that are typical of enteric viruses such as norovirus, and bacterial pathogens such as *Salmonella* spp. Symptoms of infection with *V. parahaemolyticus* can include abdominal cramping, nausea, diarrhoea, vomiting, chills and fever (Yeung and Boor 2004). Some *V. parahaemolyticus* strains are sufficiently virulent to cause outbreaks, in which large numbers of people can be affected (Martínez-Urtaza *et al.* 2005, 2013). *V. vulnificus* can cause similar symptoms, but can also be far more grievous, with infections that can spread to the bloodstream, resulting in primary septicaemia (Ratner 1987; Oliver 2006). After a short incubation, in as little as 24 h, the patient can experience dangerously low blood pressure, blistering skin lesions along the extremities, organ failure and death (Jones and Oliver 2009). These types of infections predominate in subpopulations with compromised immune systems (such as people with liver disease, or diabetes). Interestingly, *V. vulnificus* can also cause serious infections through entry of the pathogen into an open wound, and can rapidly proceed to the point of requiring amputation of limbs or, ultimately, death (Horseman and Surani 2011). *Vibrio vulnificus*, which has a fatality rate approaching 50%, is the most fatal foodborne pathogen in the United States. Thus, *V. parahaemolyticus* results in the highest number of cases, while *V. vulnificus* cases are more severe and cause the most deaths.

While the United States has some of the best *Vibrio* disease epidemiology data available, reporting requirements only began in 2007. Other countries, with fewer historical infections, have large numbers of shellfish harvested and consumed on an annual basis but a relatively low level of data are available. In South America, for example, shellfish and finfish aquaculture are large and economically robust industries, corresponding to 7·6% of the world's production (FAO 2013). Yet, despite the occurrence of both *V. vulnificus* and *V. parahaemolyticus* along the Pacific and Atlantic coasts of South America, these bacteria are not part of any formal or official monitoring program for shellfish production in any of the South American countries. Only *V. parahaemolyticus* is regulated for seafood in Brazil and Peru, but not at shellfish production areas, as these regulations are focused on ready to eat products. There may be limited transmission of *Vibrio*-caused disease from nonmolluscan aquaculture and seafood products in South America, but the presentation of information here is focused on the well-known vectors of *Vibrio*-caused disease, molluscan shellfish. The objective of this article is to review and summarize the importance of *V. parahaemolyticus* and *V. vulnificus* in South America, across both environmental waters and seafood, as well as to document in a single publication, the reported cases and outbreaks caused by pathogenic *V. parahaemolyticus* and *V. vulnificus* pathogens. Through this summary, we present information indicating the importance of developing coordinated monitoring strategies for these pathogens into the future.

Shellfish aquaculture in South America

South America is the fourth largest continent in the world with twelve countries and three major territories: the Falkland Islands, the Galapagos Islands and the French Guiana. South America is surrounded by the Pacific and Atlantic Oceans, and by the Caribbean Sea. Bolivia and Paraguay are the only landlocked countries.

Aquaculture in South America produces a significant amount of food and contributes substantially to local economies. The continent contributes 7·6% (12 307 208 tons) of the world fishery production, with 80% of the total supplied by three countries: Peru, Chile and Brazil (FAO 2013). Bivalve mollusc production has always been remarkable in Chile and Peru, and in other countries, like Brazil, production has been on the rise (FAO 2013). Shellfish production is important for jobs and economic growth throughout the year and also serves as an alternative source of income for fishermen during closed fishing seasons. And even if *V. vulnificus* and *V. parahaemolyticus* are not known to be pathogenic to shellfish, the possible impact of *V. parahaemolyticus* on shrimp populations via early mortality syndrome and their pathogenicity to finfish has now been noted (De Schryver *et al.* 2014).

Vibrio parahaemolyticus occurrence in South America

In 1971, Argentina reported the occurrence of *V. parahaemolyticus* in mussels (Casellas *et al.* 1977), which was the first report in South America and until now it is the only report of the bacterium in that country. In 1975, Brazil reported the first human case of *V. parahaemolyticus* (Hofer 1983) in South America. Since then, many reports of *V. parahaemolyticus* in environmental samples, seafood and marine animals have been published, and isolated cases and outbreaks of *V. parahaemolyticus* have been observed in some South American countries.

The spread of *V. parahaemolyticus* in environmental samples (Fig. 1) and the occurrence of human cases and years reported along the South America have been documented for the recent decades (Fig. 2). It is important to observe that the spread of the disease coincides with El Niño Southern Oscillation (ENSO) years and location, reaching the Peruvian coast in 1993 and again in 1997, when the cases spread to Chilean coast (Figs. 1 and 2).

Records of *V. parahaemolyticus* in South American marine water and animal samples are mainly found in Brazil, with only a few reports in Chile, Peru, Colombia and Venezuela. Table 1 lists published reports, from 1971 until 2015, of *V. parahaemolyticus* in environmental and seafood samples from South America. Despite the existing reports of the bacterial presence in environmental and seafood samples, only Brazil and Peru have instituted national legislation establishing a maximum allowed number of *V. parahaemolyticus* in seafood. Peru requires nondetectable *V. parahaemolyticus* in 25 g for all seafood (Peru 2008), while Brazilian legislation established a maximum limit of 10^3 CFU g^{-1} in ready-to-eat seafood, which includes raw oysters (Brasil 2001), while the limit in the United States is equal to or $>1 \times 10^4$ CFU g^{-1} (Kanagawa positive or negative) for ready-to-eat fishery products with minimal cooking by the consumer (FDA 2011). Interestingly, even though numerous *V. parahaemolyticus* outbreaks have been reported, Chile does not establish maximum limits for *V. parahaemolyticus* in seafood. There is only a requirement for cold transportation of molluscan shellfish (Chile 1996).

Some strains of *V. parahaemolyticus* are especially virulent and instead of causing single sporadic cases, they are responsible for outbreaks (Martínez-Urtaza *et al.* 2004, 2013), occurring when the frequency of cases of a disease is above what was expected in a defined community, geographical area or season (WHO, 2015). These outbreak strains can be identified by serotyping of the capsular (K) and lipopolysaccharide (O) antigens (Parveen and Tamplin 2013). Two serotypes are of particular importance, O4:K12 and O3:K6 and have been implicated in recent outbreaks and have worldwide presence.

The first report of *V. parahaemolyticus* infection in South America occurred in 1975 in Brazil, and was reported as isolated watery diarrhoea in a 6-year-old child from Ceara, Brazil (Hofer 1983). The strain was serotyped as O5:K17, Kanagawa-positive. There are no epidemiological data available, except that the local population was known to eat salt-cured marine and freshwater fish (Hofer 1983; dos Santos and Vieira 2013). Even though there are other *V. parahaemolyticus* serotypes such as the above mentioned O5:K17 in South America, O3:K6 and O4:K12 serotypes are worth mentioning as they are related to outbreaks in South America and also in other continents as cited previously (Bhuiyan *et al.* 2002; Chao *et al.* 2011; Martínez-Urtaza *et al.* 2013; Powell *et al.* 2013).

Serotype O4:K12 and O4:KUT are of concern today in Europe and the United States (Martínez-Urtaza *et al.* 2013; Haendiges *et al.* 2015). They have been shown to be more virulent than other pathogenic *V. parahaemolyticus* strains and they have caused large outbreaks in the United States in 1997, 2004 and 2013 (Martínez-Urtaza *et al.* 2013; Newton *et al.* 2014). In South America, except for a report in Brazil from an outbreak in 1989 (Magalhães *et al.* 1991) and one report from Kanagawa-negative environmental sample (Pereira *et al.* 2004), all findings from this serotype occurred on the Pacific coast. Serotype O4:K12 has been recovered from environmental and seafood samples in South America since 1984, when

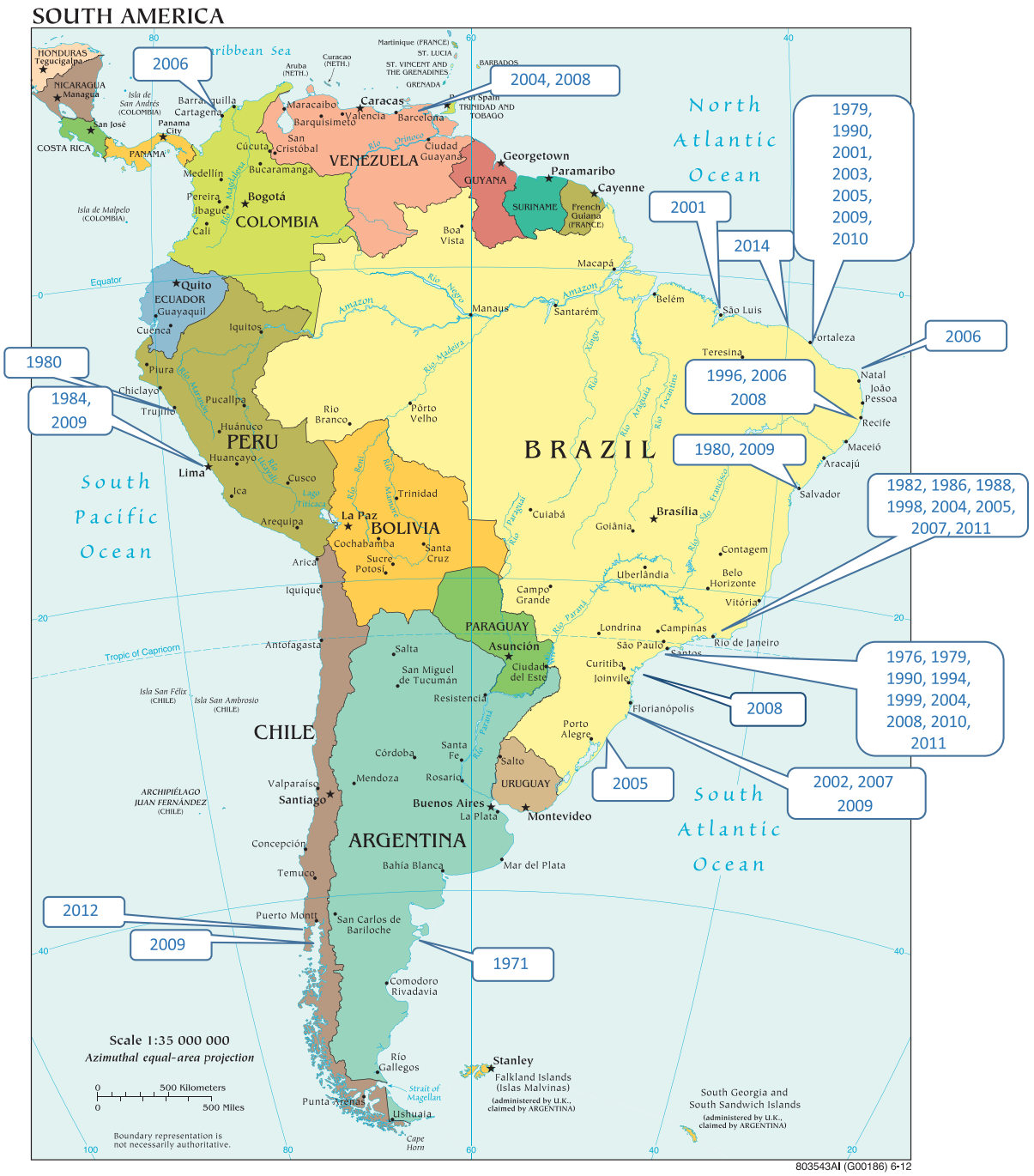

Figure 1 Map of *Vibrio parahaemolyticus* reports in South American environmental samples with tags showing the year of occurrence. Colour figure can be viewed at wileyonlinelibrary.com.

it was found in ceviche samples in Peru (Guevara-Duncan *et al.* 1989). Since then, it has been mainly found in outbreaks in Peru and Chile in 1997 and 2004, respectively, at the same time when this serotype caused outbreaks in the United States (González-Escalona *et al.* 2005; Gil *et al.* 2007). The outbreak that occurred in Chile in 2004 affected approx. 1500 people, mainly in Puerto Montt, a region characterized by cold coastal water and one of the main shellfish-producing areas in Chile (González-Escalona *et al.* 2005). It is important to mention that the Pacific coast was under the influence of ENSO phenomena in both the 1997–1998 and 2004–2005 years, with warmer water all along the coastline, favouring the growth of the bacteria.

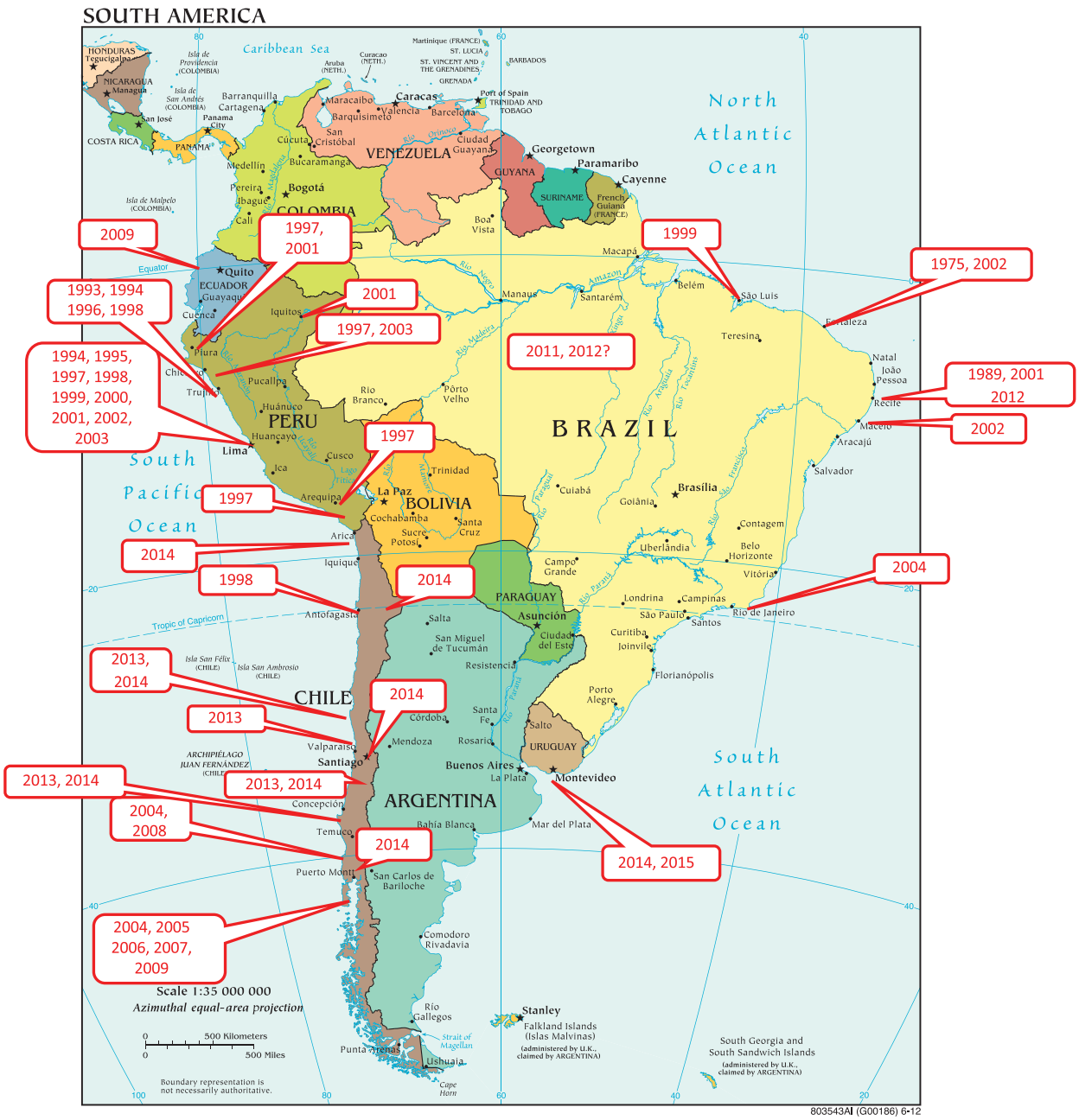

Figure 2 Map of *Vibrio parahaemolyticus* cases and outbreaks in South America, with tags showing the year of occurrence. Colour figure can be viewed at wileyonlinelibrary.com.

Strain O3:K6 has been implicated in outbreaks worldwide until since 1996 and has been typically found associated with other serotypes such as O1:K38, O3:K29, O4:K8, O2:K3 and O4:K8 (Okuda *et al.* 1997; Wong *et al.* 2000). This situation changed in 1996, after an atypical increase in *V. parahaemolyticus* O3:K6 infections in India (Velazquez-Roman *et al.* 2013) when the serotype was detected alone causing disease. In the same year, this clone rapidly spread throughout Southeast Asian countries (Okuda *et al.* 1997; Chowdhury *et al.* 2000), and also to South America, particularly in Peru (Martínez-Urtaza *et al.* 2008). It was then reported in the following years in outbreaks and isolated cases in the Atlantic and Gulf coasts of the United States (Okuda *et al.* 1997; Chowdhury *et al.* 2000; Matsumoto *et al.* 2000). Recently, similar reports were obtained from Europe (Martínez-Urtaza *et al.* 2013), Africa (Ansaruzzaman *et al.* 2005), and North, Central and South America

Table 1 Reports of *Vibrio parahaemolyticus* in environmental and seafood samples in South America

Year	Country	State/Region	Matrix/Temp/Salinity	Serotype	Reference
1971	Argentina	Chubut	Mussel		Casellas *et al.* (1977)
1976	Brazil	São Paulo	Oyster	O5:K17, 05:K47, O3:K45, O1:K32, O11:K5, O3:K33	Leitão *et al.* (1976)
1979	Brazil	São Paulo	Oyster		Gelli *et al.* (1979)
1979	Brazil	Ceará	Fish		Hofer and Silva (1974)
1980	Peru	Trujillo	Crab and water		Bocanegra *et al.* (1981)
1980	Brazil	Bahia	Molluscs, shellfish, crustaceans, fish		Franca *et al.* (1980)
1982	Brazil	Rio de Janeiro	Oyster and water	O1:K32, O1:K33, 02:K28, O3:K29, O3:K*, O4:K13, O4:K*, O6:K61, O5:K*, O8:K20, O11:K15	Rodrigues and Hofer (1986)
1984	Peru	Lima	Ceviche	O4:K33, O1:K33, O2:K28, **O4:K12**, O5:K17, O3:K30, O1:K33, O7:K19, O2:K22, O3:K33, O11:K61.	Guevara-Duncan *et al.* (1989)
1986	Brazil	Rio de Janeiro	Fish	O2:K28	Hofer and Silva (1986)
1988	Brazil	Rio de Janeiro	Squid		Lima *et al.* (1994)
1990	Brazil	Ceará	Lobster	Serogroups K.	Vieira and Iaria (1993)
1990	Brazil	São Paulo	Mussel/Temp: 23–23·5°C/ Salinity: 32–33‰		Matté *et al.* (1994)
1990	Brazil	São Paulo	Oyster		Rojas *et al.* (2011)
1994	Brazil	Santa Catarina	Mussel/Temp: 23–28·5°C/ Salinity: 35–36·5‰	O5:K30, OND:K30, OND:K17, OND: KND, OND:K39, OND:K22, O1:K30, O3:K17, O5:KND, OND:K13, OND: K34, OND:K11	Archer and Moretto (1994)
1994	Brazil	São Paulo	Mussel		Matté *et al.* (2007)
1998	Brazil	Rio de Janeiro	Oyster and mussel	O10:K*,O1:K*, O5:K17, O8:K*, O2: K28, O10:K69, O2:K3, O3:K57, O3: K72, O11:K*, O2:K*, O4:K*, O4: K42, O10:K52, O11:K19, O1:K32, O3:K33, O4:K34, O5:K47, O11: K34, O1:K12, O1:K33, O2:K25, O2: K30, O3:K*, O3:K5, **O3:K6**, O3: K30, O3:K31, O3:K36, **O4:K12**, O5:K25, O6:K*, O8:K11, O8:K39, O8:K41, O9:K*, O10:K7, O10:K25, O10:K31, O10:K60, O11:K22, O11: K36, O11:K40	Pereira *et al.* (2004)
1999	Brazil	Pernambuco	Oyster and water/Temp: 26–30°C/Salinity: 27–42‰		Lira *et al.* (2001)
1999	Brazil	São Paulo	Oyster and water/Temp: 19–28°C/Salinity: 16–21‰		Ristori *et al.* (2007)
2001	Brazil	Maranhão	Clam and mussel/Temp: 30–32°C/Salinity: 9–23‰		Serra *et al.* (2001)
2001	Brazil	Ceará	Oyster Salinity: 3‰		Sousa *et al.* (2004)
2002	Brazil	Santa Catarina	Oyster, mussel and water/ Temp: 20–28°C/Salinity: 29–36‰		Silva (2003)
2003	Brazil	Ceará	Oyster		Barros *et al.* (2003)
2003	Brazil	Ceará	Crab		Vieira *et al.* (2004)
2004	Venezuela	Sucre	Mussel		Grau *et al.* (2004)
2004	Brazil	Rio de Janeiro	Mussel		Lafisca *et al.* (2008)
2004	Brazil	São Paulo	Tuna		Chen (2004)
2004	Venezuela	Sucre	Clam and mussel		Muñoz *et al.* (2008)
2005	Brazil	Rio de Janeiro	Marine mammals		Pereira *et al*. (2007a)
2005	Brazil	Rio Grande do Sul	Marine mammals		Pereira *et al*. (2007a)

(Continued)

Table 1 (*Continued*)

Year	Country	State/Region	Matrix/Temp/Salinity	Serotype	Reference
2005	Brazil	Ceará	Shrimp and water		Costa (2006)
2006	Brazil	Pernambuco	Shrimp and water		Mendes *et al.* (2009)
2006	Colombia	Cartagena	Oyster		López *et al.* (2010)
2007	Brazil	Rio de Janeiro	Mussel		Pereira et al. (2007b)
2007	Brazil	Santa Catarina	Oyster/Temp: 23–24°C/ Salinity: 33–34‰		Ramos (2007)
2007	Brazil	Rio Grande do Norte	Shrimp		Melo *et al.* (2011)
2007	Brazil	Santa Catarina	Oyster/Temp: 18–29°C		Ramos *et al.* (2012)
2008	Brazil	São Paulo	Water/Temp: 20–32°C/ Salinity: 19–32‰		Markman (2008)
2008	Brazil	Paraná	Water/Temp: 16–35°C/ Salinity: 17–27‰		Markman (2008)
2008	Brazil	Pernambuco	Water/Temp: 23–29°C/ Salinity: 8–35‰		Markman (2008)
2008	Venezuela	Sucre	Clam		Muñoz *et al.* (2008)
2009	Peru	Lima	Fish/Temp: 20°C	**O3:K6**	Aliaga *et al.* (2010)
2009	Chile	Región de los Lagos	Molluscan shellfish	**O3:K6**, O3:KUT	García *et al.* (2009)
2009	Brazil	Santa Catarina	Oyster and water/Temp: 21–28°C/Salinity: 31–34‰		Ramos *et al.* (2014)
2009	Brazil	Santa Catarina	Oyster and water/Temp: 24°C/Salinity: 12–36‰	O1:K1, O1:K25, O1:K41, O1:K69, O1:KUT, O2:K3, O2:K28, **O3:K6**, O3:K30, O4:K34, O4:K63, O5:K61, O6:K4, O6:K6, O6:K18, O6:K46, O7:K7, O7:K19, O8:K20, O8:K39.	Ramos (2012)
2009	Brazil	Bahia	Oyster		Rodrigues and Carvalho-Filho (2011)
2009	Brazil	Ceará	Oyster		Vieira *et al.* (2011)
2010	Brazil	São Paulo	Oyster/Temp: 14–28°C/ Salinity: 5–30‰		Costa Sobrinho *et al.* (2010)
2010	Brazil	São Paulo	Oyster		Costa Sobrinho *et al.* (2011)
2010	Brazil	Ceará	Oyster		Vieira *et al.* (2010)
2011	Brazil	São Paulo	Mussel, oyster		Rojas *et al.* (2011)
2011	Brazil	Rio de Janeiro	Mussel		Oliva (2012)
2012	Chile	Puerto Montt	Molluscan shellfish		Aranda *et al.* (2015)
2014	Brazil	Piauí	Shrimp		Muratori *et al.* (2014)

Serotypes in bold are the pandemic *V. parahaemolyticus* serotypes. Temp refers to seawater temperature.

(Daniels and MacKinnon 2000; González-Escalona *et al.* 2005; Velazquez-Roman *et al.* 2013). Unfortunately, only a handful of reports from South America have included *V. parahaemolyticus* serotyping, which does not allow us to trace the frequency of outbreak strains along the South American coast temporally. Considering the serotyped samples, it is noted that serotype O3:K6 is still a public health concern in South America and it has been observed more frequently in outbreaks and environmental samples than the O4:K12 serotype.

The first report of *V. parahaemolyticus* pandemic strain O3:K6 in South America was one isolated case in Trujillo, Peru in 1996 in a 6-month-old baby, and occurred simultaneous to the outbreak in Calcutta in February 1996. However, the first Peruvian outbreak related to the O3:K6 strain occurred in Lima, in 1998, which coincided with a strong ENSO occurrence and the strain was found to be similar to that found in the Calcutta outbreak in 1996 (Gil *et al.* 2007). In Chile, the first report occurred in Antofagasta, in 1997 and the serotype has been present on the Chilean coast since then (González-Escalona *et al.* 2005; Dabanch *et al.* 2009; García *et al.* 2009). In 2005, there were nearly 11 000 cases reported by the Ministry of Health of Chile, with more than 95% of the cases caused by serotype O3:K6 (García *et al.* 2009). This strain was also found to cause gastroenteritis cases in Brazil in

Pernambuco, Alagoas and Ceará in 2002 (Leal *et al.* 2008). These states are within the tropical region, in northeast Brazil, characterized by warm waters and high tourism activity. Unfortunately, there is information about the number of cases only in the Ceará outbreak, with 26 reported cases between guests of two hotels, and in which *V. parahaemolyticus* O3:K6 was found in 45% of the cases. The bacterium was not isolated from any food sample (FUNASA 2002), which could have been due to imprecise detection methodology or to inadequate food sample maintenance. This outbreak in Ceará highlights the importance of an adequate surveillance system, not only to generate more accurate data about foodborne disease outbreaks but also to ensure the correct treatment is administered. There were also outbreaks in 2003 and 2004 in Brazil, with no information available about location or serotype (Brasil 2014).

Notably, most of the recent reports do not discuss serotype information. Beyond the absence of information on *V. parahaemolyticus* serotypes in the reports referenced in Table 1, there is also a lack of information on salinity and seawater temperature. These two environmental parameters are critical to understanding the ecology of the species. *V. parahaemolyticus* growth is favoured by warmer temperatures, between 5 and 43°C, with an optimum temperature of 37°C, and by moderate salinity, a range of 0·5–10‰, with optimum growth between salinity of 1·5–3·0‰ (WHO and FAO 2011). These environmental parameters are often observed on South American coasts, especially in estuarine areas or after tropical rainfalls. Moreover, the ENSO oscillation that occurs along the Pacific coast brings warm seawater to this area that can favour the presence of the bacteria on the South American west coast and increase the occurrence of cases and outbreaks.

A review of the data shows a broad variation in salinity and temperature in South America. Seawater temperature on the Atlantic coast varied between 14·4°C (Costa Sobrinho *et al.* 2010) and 35°C (Markman 2008), which is within the range of the survival and growth of the species. Salinity ranged between 3‰ (Sousa *et al.* 2004) to 41·6‰ (Lira *et al.* 2001). The remarkably higher salinity data found (41·6‰) was recorded in an estuarine area on the Brazilian northeast coast, with a water temperature of 30°C. The shellfish samples contained around 10^3 CFU g^{-1} of sucrose-negative colonies on TCBS agar, which indicates the presence of *V. parahaemolyticus*. Approximately 60% of those isolates were confirmed to be *V. parahaemolyticus* by biochemical tests (Lira *et al.* 2001). It is important to note that even under extreme conditions, such as high salinities, researchers were able to quantify the species, showing that it can be a potential health hazard to molluscan shellfish consumers.

Since there is no official monitoring program for *V. parahaemolyticus* in shellfish production areas or seawater in South American countries, the data presented in this review are not uniform in respect to the methodology used, temporal or spatial coverage, and do not provide enough information to establish relationships between bacterial abundance and environmental parameters. These factors are needed to perform predictive analyses.

Among the studies found in this review, two of them, from the same author, deserve to be mentioned for robust monitoring and the inclusion of environmental parameters, serotyping and PCR confirmation for thermostable direct haemolysin (*tdh*) and *tdh*-related haemolysin, *trh.* genes. The presence of one or both of these genes is typically associated with host cell cytotoxicity (Nishibuchi and Kaper 1995; Broberg *et al.* 2011). Ramos (2007, 2012) studied the prevalence of vibrios in oysters and seawater in Santa Catarina, the main oyster production area in Brazil. *Vibrio parahaemolyticus* was found as the most prevalent *Vibrio* species in both studies. Samples were examined for potential virulence by assaying for the presence of *tdh* and *trh* genes. There were strains positive for *tdh* and *trh* genes, but no positive strain was found on Wagatsuma agar. The haemolysis caused by some *V. parahaemolyticus* strains on this medium is called 'Kanagawa phenomenon' and it is positively related to human pathogenicity. It was found that *tdh* is responsible for the Kanagawa phenomenon (Honda *et al.* 1980), although there are cases of Kanagawa-negative and *tdh*-positive strains (Vieira *et al.* 2011; Ramos 2012). Ramos (2012) found no correlation between environmental parameters and the presence of *V. parahaemolyticus* when the sampling occurred in a temperature range between approx. 21 and 28°C, during summer and spring seasons. In another study, Ramos (2007) collected samples all year and there was a positive correlation with the incidence of *Vibrio* and seawater temperature, ranging from 18 to 29°C. Ramos (2012) identified the presence of pandemic *V. parahaemolyticus* serotype O3:K6 among 83 *V. parahaemolyticus* strains collected and found ca. 37% as *trh* positive and 5% as *tdh* positive. The *trh* and *tdh* genes were found occurring simultaneously in 4·3% of oyster samples and in 5% of seawater samples. Although, all the strains found were Kanagawa negative.

Regrettably, none of the most recent reports published after 2009 serotyped *V. parahaemolyticus* in environmental sampling or in clinical cases. This information is crucial to trace pathogenic serotypes and to establish a control plan to reduce the number of cases and outbreaks. Table 2 shows outbreaks and isolated cases generated by *V. parahaemolyticus* in SA countries in chronological order. The last report of disease caused by

Table 2 Reports of *Vibrio parahaemolyticus* incidence in isolated human cases and outbreaks in South America

Year	Country	State/Region	Cases/Outbreak information	Strain identification/serotype	Reference
1975	Brazil	Ceará	Isolated case	O5:K17	Hofer (1983)
1989	Brazil	Pernambuco	Outbreak	**O4:K12**, O1:K56, O3:K5, O3:K58, O3:KUT, O4:K4, O4:K10, O4:K53, O5:KUT, O10:KUT	Magalhães *et al.* (1991)
1993	Peru	Trujillo	Outbreak	O2:K3, O4:K8, OUT:KUT	Gil *et al.* (2007)
1994	Peru	Lima, Trujillo	Outbreak	OUT:K3, O2:K3, O2:KUT, O4:K8	Gil *et al.* (2007)
1995	Peru	Lima	Outbreak	**O4:K12,** OUT:K46	Gil *et al.* (2007)
1996	Peru	Trujillo	Outbreak	**O3:K6**, O4:K8, OUT:K8.	Gil *et al.* (2007)
1997	Peru	Lima, Arequipa	Outbreak	**O3:K6, O4:K12**	Gil *et al.* (2007)
1997	Peru	Lima, Cajamarca, Lambayeque, Monqueagua	Outbreak	**O3:K6**	Martínez-Urtaza *et al*. (2008)
1998	Peru	Lima	Outbreak	**O3:K6**	Martínez-Urtaza *et al*. (2008)
1998	Chile	Antofagasta	Outbreak	**O3:K6**, O1:K56	González-Escalona *et al.* (2005)
1998	Chile	Antofagasta	Outbreak		Córdova *et al.* (2002)
1998	Peru	Lima, Trujillo	Outbreak	**O3:K6,** O3:K68, O3:K58, O4:K8, **O4:K12,** O11:KUT, O11:K15, OUT:KUT	Gil *et al.* (2007)
1998	Peru	Lima	Outbreak		Ibarra *et al.* (1999)
1999	Peru	Lima, Lambayeque	Outbreak	**O3:K6,** O3:KUT	Martínez-Urtaza *et al*. (2008)
1999	Brazil	Maranhão	Wound		Rodrigues *et al.* (2001)
1999	Peru	Lima	Outbreak	**O3:K6**	Gil *et al.* (2007)
2000	Peru	Lima	Outbreak	**O3:K6, O4:K12**	Gil *et al.* (2007)
2001	Peru	Lima, Lambayeque, Iquitos	Outbreak	**O3:K6**	Martínez-Urtaza *et al*. (2008)
2001	Peru	Lima	Outbreak	O6:K18	Gil *et al.* (2007)
2001	Brazil	Pernambuco	Outbreak	O3:KUT	Leal and Franco (2008)
2002	Peru	Lima	Outbreak	**O3:K6**	Martínez-Urtaza *et al*. (2008)
2002	Brazil	Pernambuco	Isolated case and outbreak samples (26 cases/9 confirmed O3:K6, due to raw crab leg	**O3:K6**	Leal *et al.* (2008); FUNASA (2002)
2002	Brazil	Ceará	Outbreak	**O3:K6**	Leal and Franco (2008)
2002	Brazil	Alagoas	Isolated case	**O3:K6**	Leal and Franco (2008)
2003	Peru	Lima, Cajamarca	Outbreak	**O3:K6**	Martínez-Urtaza *et al*. (2008)
2003	Brazil		2 outbreaks		Brasil (2014)
2004	Brazil		1 outbreak		Brasil (2014)
2004	Chile	Puerto Montt	Outbreak	**O3:K6, O4:K12**	González-Escalona *et al.* (2005)
2004	Chile	Región de los Lagos	Outbreak/1500 cases	**O3:K6**	García *et al.* (2009)
2005	Chile	Región de los Lagos	Outbreak/3725 cases	**O3:K6**	García *et al.* (2009)
2006	Chile	Región de los Lagos	Outbreak/1083 cases	**O3:K6**	García *et al.* (2009)
2007	Chile	Región de los Lagos	Outbreak/477 cases	**O3:K6**	García *et al.* (2009)
2008	Chile	Puerto Montt	Outbreak/1153 cases	**O3:K6,** O3:KUT, OUT:KUT	García *et al.* (2009)
2008	Chile	Puerto Montt	Isolated case, septicemia		Dabanch *et al.* (2009)
2009	Ecuador		Isolated case	OUT:K29	Ottaviani *et al.* (2013)
2009	Chile	Región de los Lagos	Outbreak/441 cases	**O3:K6,** O3:KUT	García *et al.* (2009)
2011	Brazil		1 outbreak		Brasil (2014)
2012	Brazil		2 outbreaks		Brasil (2014)
2013	Chile	Coquimbo, Valparaíso, Maule, Biobío, Los Ríos	31 outbreaks/383 cases Jan-April)		Chile (2014, 2015)

(Continued)

Table 2 (*Continued*)

Year	Country	State/Region	Cases/Outbreak information	Strain identification/serotype	Reference
2014	Chile	Arica y Parinacota, Atacama, Coquimbo, Metropolitana, Maule, Biobio	5 outbreaks/26 cases and 3 isolated cases (Jan-April)		Chile (2015)
2014/ 2015	Uruguay	Rio de la Plata Estuary	3 cases wound infection (cellulitis)		T. Camou (personal communication)
2015	Chile	Arica y Parinacota, Tarapaca, Coquimbo, Valparaíso, Metropolitana, Maule, Los Rios	8 outbreaks/60 cases and (Jan-April)		Chile (2015)

UT, untypeable.
Serotypes in bold are the pandemic *V. parahaemolyticus* serotypes. Temp refers to seawater temperature.

serotype O3:K6 in South America was in Chile (García *et al.* 2009) in 2009, the same year the serotype was found in environmental samples from Brazil (Ramos 2012). However the lack of serotyping information from the strains found after 2009 does not allow us to identify the presence this *V. parahaemolyticus* serotype in recent years in South America.

Although primary septicaemia caused by *V. parahaemolyticus* is rare, it can occur in individuals with underlying chronic illness (Parveen and Tamplin 2013). In South America, Dabanch *et al.* (2009) reported the first case of septicaemia due to *V. parahaemolyticus* in Puerto Montt, in Chile, which occurred in 2008.

Vibrio parahaemolyticus human cases are more frequent on the Pacific coast of South America, and the majority of the reports coincide with ENSO occurrences. Furthermore, the spread of *V. parahaemolyticus* infections from Peru to Chile follows the same course observed during ENSO currents, with warm waters towards south, which is the opposite of normal Peru Current route, as seen in Figs. 1 and 2. From 1993 until 1997, *V. parahaemolyticus* cases were concentrated on the Peruvian coast (Gil *et al.* 2007; Martínez-Urtaza *et al.* 2008). After 1997, the bacterium spread to Chile, where it is still causing outbreaks (Córdova *et al.* 2002; González-Escalona *et al.* 2005; Dabanch *et al.* 2009; García *et al.* 2009, Chile 2014, 2015). These facts highlight the influence of ENSO on the spread and escalation of the disease along South American Pacific coast. Despite all of the cases and outbreaks in Peru and Chile, there are only a few publications about the presence of *V. parahaemolyticus* in environmental samples along the Pacific coast. Thus, the area in which the most cases have occurred is the region in which there is the least amount of available environmental data.

All of the cases reported above resulted from isolated research efforts, and most likely are a vast underrepresentation of the real number of infections. These data are an indication of the factors that can affect the patterns of disease in South American countries.

V. parahaemolyticus is not specifically listed as a notifiable disease in any country in South America, yet all foodborne or diarrheal diseases are required to be reported, which should be inclusive of *V. parahaemolyticus*. However, the epidemiological surveillance system on the continent is inaccurate, with a high percentage of foodborne disease outbreaks classified as being from unspecific causes. Thus, cases of diarrhoea caused by *V. parahaemolyticus* on the continent could be underestimated and underreported. This leads to weak food inspection programmes regarding these bacteria as there are not enough data to justify costly control and warning systems.

Vibrio vulnificus occurrence in South America

The presence of *V. vulnificus* in environmental and seafood samples in South America is less documented than *V. parahaemolyticus*. It is mostly reported in studies focused on *V. parahaemolyticus*, in which they also detected *V. vulnificus*, all from the Atlantic and Caribbean coasts, as seen in Fig. 3, where reports from environmental and human cases are shown.

Vibrio vulnificus has been recorded in South America since 1982 when it was found in estuarine water samples in Rio de Janeiro State in Brazil, but there was no information on temperature or salinity (Rodrigues and Hofer 1986). However, as with *V. parahaemolyticus*, *V. vulnificus* samples taken from different sites and seasons, and analysed through differing methodologies, do not allow analysis of its prevalence temporally in South America. Table 3 presents *V. vulnificus* records of environmental and seafood samples from South America countries. Seawater temperature and salinity ranges are listed when

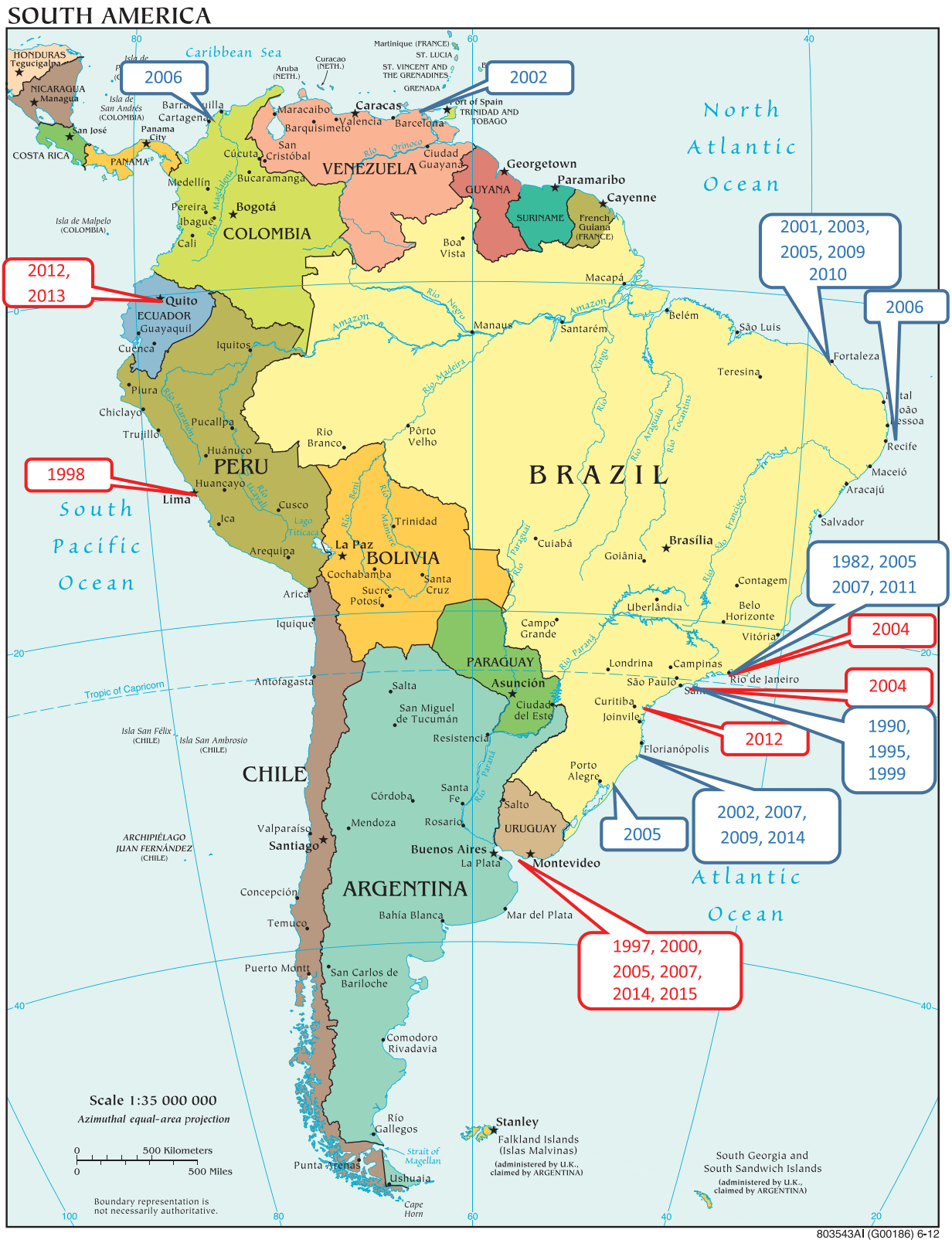

Figure 3 Map of *Vibrio vulnificus* reports from environmental samples (blue tags), and cases and outbreaks (red tags) in South America, with tags showing the year of occurrence. Colour figure can be viewed at wileyonlinelibrary.com.

present in the article cited. The methodology used by different authors is rarely the same, and was potentially inadequate to detect the bacteria, especially since most of the studies were based only on microbiological and biochemical identification, without molecular confirmation.

Higher prevalence rates of *V. vulnificus* can be found in seawater with a salinity range between 5 and 20‰ (Parveen and Tamplin 2013), the consensus is that *V. vulnificus* has a maximum environmental salinity tolerance of about 25‰ (Kaspar and Tamplin 1993; Motes *et al.* 1998; Arias *et al.* 1999; Macián *et al.* 2000; Wetz *et al.* 2008; Froelich *et al.* 2012). While *V. vulnificus* can survive at higher salinities, it becomes difficult to isolate and is considered to be rare (Froelich *et al.* 2012, 2015; Staley *et al.* 2013). According to Motes and DePaola (1996) and Audemard *et al.* (2011), keeping oysters bathed in high salinity (>30‰) water is able to reduce the level of *V. vulnificus* in oyster meat. The relationship between vibrios and salinity has been identified by some studies (Johnson *et al.* 2010; Reyes-Velázquez *et al.* 2010; Igbinosa *et al.* 2011), while others did not (Singleton *et al.* 1982; Nigro *et al.* 2011; Costa Sobrinho *et al.* 2014), demonstrating that the relationship with salinity may be variable and complex (Johnson *et al.* 2012). Although, as seen in Table 3, salinity in South America is found to be, in general, higher than 25‰, which could explain the lower prevalence of *V. vulnificus* compared to the United States. In Brazil, Ramos (2007) found *V. vulnificus* in oyster and seawater samples with salinities ranging between 33 and 34‰. Silva (2003) and Ramos (2012) also found *V. vulnificus* in environmental samples in Brazil where salinity was as high as 36‰, which is above the normal upper limit of salinity expected to permit the presence of *V. vulnificus*, although none of the authors tested the presence of potentially pathogenic strains and there are no cases of infections reported for this area (Ramos *et al.* 2012; Froelich and Noble 2014).

Table 3 Reports of *Vibrio vulnificus* incidence in environmental and seafood samples in South America

Year	Country	State/Region	Matrix	Temp and Salinity	Reference
1982	Brazil	Rio de Janeiro	Water		Rodrigues and Hofer (1986)
1990	Brazil	São Paulo	Mussel	Temp.: 23–23·5°C Salinity: 32–33‰	Matté *et al.* (1994)
1995	Brazil	São Paulo	Molluscan shellfish, shrimp		Garcia-Moreno and Landgraf (1997)
1999	Brazil	São Paulo	Oyster and water	Temp.: 19·2–28°C Salinity: 16–21‰	Ristori *et al.* (2007)
2001	Brazil	Ceará	Shrimp		Nascimento *et al.* (2001)
2002	Brazil	Santa Catarina	Molluscan shellfish, water	Temp.: 19·2–28°C Salinity: 29–36‰	Silva (2003)
2002	Venezuela	Sucre	Mussel		Grau *et al.* (2004)
2003	Brazil	Ceará	Oyster		Barros et al. (2003)
2005	Brazil	Ceará	Water		Costa (2006)
2005	Brazil	Rio de Janeiro	Marine mammals		Pereira *et al.* (2007a)
2005	Brazil	Rio Grande do Sul	Marine mammals		Pereira *et al.* (2007a)
2006	Brazil	Pernambuco	Shrimp and water		Mendes *et al.* (2009)
2006	Colombia	Cartagena	Oyster		López *et al.* (2010)
2007	Brazil	Rio de Janeiro	Mussel		Pereira *et al.* (2007b)
2007	Brazil	Santa Catarina	Oyster	Temp.: 22·27–23·57°C Salinity: 33·13–34·03‰	Ramos (2007)
2007	Brazil	Santa Catarina	Oyster	Temp.: 18–29°C	Ramos *et al.* (2012)
2009	Brazil	Santa Catarina	Oyster and Water	Temp.: 20·9–27·5°C Salinity: 30·7–33·9‰	Ramos *et al.* (2014)
2009	Brazil	Santa Catarina	Oyster and Water	Temp.: 24·3°C Salinity: 12–36‰	Ramos (2012)
2009	Brazil	Ceara	Oyster		Vieira *et al.* (2011)
2010	Brazil	Ceara	Frozen oyster		Costa *et al.* (2013)
2010	Brazil	Ceara	Oyster		Vieira *et al.* (2010)
2011	Brazil	Rio de Janeiro	Mussel		Oliva (2012)
2014	Brazil	Santa Catarina	Oyster	*vcg*-C and *vcg*-E strains Temp.: 22·2°C Salinity: 34·18‰ (*Vcg*-C) 34·83‰ (*Vcg*-E)	Raszl (2016)

Temp refers to seawater temperature.

Because of the difficulty in differentiating *V. vulnificus* from other *Vibrio* sp. on culture media, polymerase chain reaction (PCR) is typically employed to confirm presumptive colonies. The gene *vvhA* can be used to confirm *V. vulnificus* presence, but it does not differentiate potentially pathogenic and nonpathogenic strains. There are other genes, including the virulence-correlated gene (*vcg*) (Warner and Oliver 2008) that provide some indication of potential pathogenicity. Only two studies used PCR to confirm the presence of *V. vulnificus* in environmental samples from South America. Oliva (2012) used the gene *vvhA* to confirm *V. vulnificus* in mussels and Raszl (2016) used the genes *vcg-E* and *vcg-C* and confirmed the presence of a pathogenic strain in oyster samples from Brazil. All other studies were based on culture media alone, mainly Thiosulphate Citrate Bile Salts Sucrose Agar (TCBS), and/or biochemical tests. Ramos (2012) analysed oysters and seawater samples and collected data on salinity, temperature and rainfall, although there was no PCR confirmation for *V. vulnificus*. Ramos (2012) found a positive correlation between *V. vulnificus* presence between seawater samples and seawater temperature, and also with weekly precipitation levels from the week antecedent to sampling, which agrees with studies from Høi *et al.* (1998), Strom and Paranjpye (2000), Lhafi and Kühne (2007) and Blackwell and Oliver (2008). But there was no correlation found between *V. vulnificus* and salinity in seawater samples as has been found previously in Brazil (Ramos 2007; Ristori *et al.* 2007) and in other areas of the world (Høi *et al.* 1998; Blackwell and Oliver 2008; Parveen *et al.* 2008).

In some studies, *V. vulnificus* was found to be highly prevalent, as in the study performed by Matté *et al.* (1994) who found *V. vulnificus* in 17% of mussels tested, and Garcia-Moreno and Landgraf (1997) found 55% of samples from mussels, oysters and shrimp tested. Moreover, Nascimento *et al.* (2001) found *V. vulnificus* in 35% of shrimp samples. Costa *et al.* (2013) found 11·4% *V. vulnificus* presumptive in samples from frozen oysters, which were confirmed through biochemical tests. However, in other studies, the prevalence is low as 4% (Pereira *et al.* 2007b; Ramos 2007). It is difficult compare quantitative data from the scientific articles reporting *V. vulnificus* in environmental samples because of variable methodologies.

Given the reports of *V. vulnificus* from environmental water and seafood samples, there are relatively few clinical cases reported in South America. Some countries that have had confirmed *V. vulnificus* cases, such as Uruguay, Ecuador and Peru, did not previously have any report from environmental samples or in seafood. Therefore, any correlative analysis between environmental and clinical samples is impossible. Infections cases of *V. vulnificus* in South America are reported only in Brazil, Uruguay, Ecuador and Peru. The first case was diagnosed in 1997 in Uruguay. All the reported cases in South America showed in Fig. 3 are detailed in Table 4.

Severe sepsis has been described as the most common presentation of *V. vulnificus* infection. It is generally characterized by bacteraemia without an evident focus of infection. Symptoms typically occur within 7 days, and they start with an abrupt onset of fever and chills, followed by metastatic infection characterized by cutaneous lesions, usually on the lower extremities or the trunk. Ibarra *et al.* (1999) reported on *V. vulnificus* in three cases of acute diarrhoea in Peru in 1998. One of the patients discussed had a co-infection with *V. cholera* O1, but there was no follo-up information available and it is not possible to know if the symptoms evolved to sepsis. Diarrhoea is not a common symptom caused by *V. vulnificus* but there are some reported cases of patients presenting gastroenteritis, characterized by vomiting, diarrhoea, and abdominal pain, with a stool culture

Table 4 Reports of *Vibrio vulnificus* incidence in isolated human cases and outbreak in South America

Year	Country	State/Region	Case/outbreak information	Reference
1997	Uruguay	Río de la Plata Bay	Water contact, sepsis. Isolated case.	Carrerou-Perreng (2001)
1998	Peru	Lima	Acute diarrhoea. 3 cases.	Ibarra *et al.* (1999)
2000	Uruguay	Maldonado	Water contact, sepsis. Isolated case.	Chicheff *et al.* (2001)
2001	Chile	Imported case from El Salvador	Ingestion of raw oyster. Sepsis. Isolated case.	Poblete *et al.* (2002)
2004	Brazil	São Paulo	Molluscan shellfish consumption. Isolated case.	Araujo *et al.* (2007)
2004	Brazil	Rio de Janeiro	Wound infection. Isolated case.	Brack *et al.* (2004)
2005	Uruguay	Rio de la Plata Bay	Water contact. Wound infection. Isolated case.	T. Camou (not published)
2007	Uruguay	Rio de la Plata Bay	Water contact. Wound infection. Isolated case.	T. Camou (not published)
2012	Brazil	Paraná	Probably by molluscan shellfish consumption. Sepsis.	França *et al.* (2013)
2012	Ecuador	Quito	Isolated case. Sepsis.	Villacrés *et al.* (2013)
2013	Ecuador	Ibarra	Isolated case. Sepsis.	Villacrés *et al.* (2013)
2014	Uruguay	Río de la Plata Bay	Water contact. Wound infection. Isolated case.	T. Camou (not published)
2014/2015	Uruguay	Punta del Este	Water contact. Outbreak, 4 cases, 2 deaths.	Uruguay (2015)

yielding *V. vulnificus*, negative blood cultures, and no skin lesions or septicaemia caused by *V. vulnificus* (Klontz *et al.* 1988; Horseman and Surani 2011). Gastrointestinal symptoms often precede fever, chills and cutaneous manifestations. Cutaneous lesions may progress to necrotic ulcers, necrotizing fasciitis, necrotizing vasculitis or myonecrosis. Septic shock can occur in more than 50% of the cases. Hypotension during the first 12 h or leucopenia is often associated with a very poor prognosis. It has also been reported mental status changes characterized by lethargy or disorientation in half the patients (Horseman and Surani 2011).

The single case reported in Chile was recorded as being originated in El Salvador, a Central American country. A 53-year-old man, with diabetes and chronic liver disease, returned from a trip to El Salvador and had eaten raw oysters. Notwithstanding the rapid diagnosis, the man died after 68 h of hospitalization (Poblete *et al.* 2002). Despite all the reports about *V. parahaemolyticus* in Chile, there is no information about the presence of *V. vulnificus* in environmental samples as well as in human cases, in this country, nor in El Salvador, from where the infection was supposed to be acquired.

In 2013, Ecuador had reported two cases of *V. vulnificus* that resulted in sepsis during summer season 2012/2013. Both patients were men with previously documented health disorders (diabetes and aplastic anaemia respectively). One of the patients presented a gastrointestinal disease prior to sepsis. Both cases resulted in death and there was no history of molluscan shellfish consumption, but it is intriguing that the cases occurred away from the coast (in Quito and in Ibarra). The authors confirmed the strains through the *rrs* gene (Villacrés *et al.* 2013).

In Brazil, regardless of the number of reports indicating the presence of *V. vulnificus* in seawater, in shellfish and in marine mammals (Garcia-Moreno and Landgraf 1997; Pereira *et al.* 2007a,b; Ramos 2007, 2012; Costa *et al.* 2013); there are only three infection cases reported, and two of them occurred in 2004 in areas that are close to one another. The first case reported in Brazil was related to an injury caused by a wood fragment, in a 53-year-old man, in Rio de Janeiro state. The case evolved to septicaemia and the death occurred 5 days later (Brack *et al.* 2004). The second case was acquired by seafood ingestion (mussels and octopus) in the coast of São Paulo state, in an 86-year-old man, with a history of pancreatic, and hepatic and renal chronic disorders. The symptoms started with gastroenteritis and evolved to sepsis. The patient died after 12 days, due to fungaemia caused by *Candida albicans* (Araujo *et al.* 2007). The third case in Brazil occurred in Paraná State, in 2013. The patient was a 39-year-old man, who was admitted to the hospital for elective liver transplantation due to an ulcerative colitis and sclerosing cholangitis. The man had been in the coast 1 day before being admitted in the hospital. Before the surgery, the symptoms of primary septicaemia began and despite the diagnosis and the treatment applied, he died 32 h after the onset of the symptoms (França *et al.* 2013).

Uruguay is the country in South America with the higher number of *V. vulnificus* cases, which occurred all in the same area and were related to seawater contact. This country reported the first case of *V. vulnificus* infection in South America, in 1997, in the estuarine area of Río de la Plata (Carrerou-Perreng 2001). The patient, a 60-year-old diabetic man, reported to be fishing at Río de la Plata estuary the day before, letting both legs for hours into the water. The infection evolved to sepsis and caused the death of the patient even with bilateral leg amputation. Another case occurred in 2000, in Maldonado Department, also in the estuarine area of Río de la Plata, where a 57-year-old woman with diabetes using weekly immunosuppressive medication, and who had a surgical wound not completely scarred, and had been in seawater 24–36 h before the first symptoms occurred. She presented wound infections and also gastroenteritis, but she survived after 12 days of intensive treatment in hospital (Chicheff *et al.* 2001). Other cases occurred in Uruguay in 2005, 2007 and 2014/2015 summer season, all from the same area, the cases from 2005 and 2007 evolved to sepsis and death, while in 2014/2015 summer season, two of four patients died as a consequence of the infection (Uruguay 2015). The information from 2005 and 2007 is not published, they were obtained from personal communication with Dr. T. Camou, from the Ministry of Public Health from Uruguay.

There were two outbreaks of *V. vulnificus* in South America, three cases occurring in Peru, without follow-up of the patients (Ibarra *et al.* 1999) and another outbreak with four cases in Uruguay, as reported by the Ministry of Health in the 2014/2015 summer season in Punta Del Este, resulting in two deaths (Uruguay 2015). All cases from Uruguay occurred in the same area, an estuary that can present salinity rates around 20‰ and seawater temperature that can be as high as 25°C in summer seasons, as reported by Piola *et al.* (2003). There is no information about the presence of *V. vulnificus* in environmental samples from Uruguay, specifically from Rio de la Plata estuarine water where the infection cases occurred. Summer seawater temperature and salinity in Río de la Plata are permissive for *V. vulnificus*, yet more studies are needed to determine if one or both parameters are related to these infections or if there are other factors that could influence the presence of pathogenic strains in the area. An increase in *V. vulnificus* cases in

South America since 2012 has been observed, which could be related to an increase in the concentration of the bacteria, but it also could be evidence of increased surveillance (personal communication, T. Camou, Ministry of Public Health of Uruguay 2015).

Except for the gastrointestinal cases from Peru, all the reports of *V. vulnificus* human cases occurred on the Atlantic coast. Comparing the distribution maps from *V. parahaemolyticus* and *V. vulnificus* in South America (Figs. 1–3), it can be seen that the only places where both bacteria were reported causing infection were Peru, Ecuador and Uruguay. It also appears that there could be some factor that affects the distribution of pathogenic strains of *V. parahaemolyticus* or *V. vulnificus* in some areas, which is not observed for environmental strain distribution. These factors could be related to seawater temperature, oceanic currents, salinity or even with association of *Vibrio* pathogenic strains with other marine organisms, and also could be due to bacterial genetic characteristics, but it is difficult to establish since there is not a continuous monitoring and not all the countries had published environmental data, besides this is an assumption that needs to be further studied with environmental data and genotyping information.

Climate impact on *Vibrio* populations

Vibrio parahaemolyticus and *V. vulnificus* occurrence in the estuaries and along the coastlines of South America is ubiquitous, and therefore, they are also commonly found in shellfish grown in these areas (Pereira *et al.* 2004; Markman 2008; Muñoz *et al.* 2008; Ramos *et al.* 2014; Aranda *et al.* 2015). In the United States, temperature and salinity have been determined to be fundamental predictors of *V. parahaemolyticus* and *V. vulnificus* abundance. For a recent review of how environmental factors affect the concentration of *V. parahaemolyticus* and *V. vulnificus* in oysters, see Froelich and Noble (2016). Salinity along the South American coast varies with precipitation and proximity to estuaries. For example, the Amazon River outflow can cause a decrease in the salinity that is observed more than 300 km from the mouth of the river (Dias 2007).

Moreover, ENSO, which happens every 3–4 years, causes an increase in seawater temperatures on the Pacific coast, as well as an increase in precipitation levels in tropical and equatorial areas on both coasts of South America. These temperature increases and precipitation associated with salinity decreases could play a major role in *V. parahaemolyticus* and *V. vulnificus* proliferation and, consequently, enhance the probability of cases and outbreaks generated by these bacteria. The hypothesis of ENSO role spreading waterborne diseases as a long-distance corridor between Asia and the Americas has also been presented by Martínez-Urtaza *et al.* (2016). Besides ENSO, decreases in sea surface salinity in the western and central equatorial Pacific can happens due to low salinity water that is brought in by anomalous eastward surface currents, and to a lesser extent due to excess rainfall in the Pacific Ocean (Zhu *et al.* 2014). According to Latif and Grotzner (2000) the effects of ENSO can also impact the Atlantic coast, often with a lag period of 6 months. The area also has another similar phenomenon, the Equatorial Atlantic oscillation, which happens ca. every 30 months. However, as water temperatures on the Atlantic coast are warm, the impact of ENSO and the Equatorial Atlantic oscillation on the Equatorial and Tropical Atlantic coast can be lesser than those observed for ENSO on the Pacific coast in relation to *Vibrio* sp.

Another consequence of ENSO is the spread of *V. parahaemolyticus* from other continents to the Pacific coast of South America. According to Martínez-Urtaza (2011), the 1997 *V. parahaemolyticus* outbreak in Peru coincided with an ENSO phenomenon. The authors had analysed data from *V. parahaemolyticus* strains from human cases in Peru between 1994 and 2005, and also studied the distribution and environmental parameters of these cases. The strains from the 1997 outbreak were identified as being serotype O3:K6 and showed a close correspondence with the arrival and circulation of 1997 ENSO along the Pacific coast of South America. Moreover, the increase in surface seawater temperature from 18 to 23°C increased the risk of infection by 600-fold (Martínez-Urtaza *et al.* 2008).

ENSO also could explain *V. vulnificus* cases in Peru in 1998 (Ibarra *et al.* 1999; Martínez-Urtaza *et al.* 2016), a year notable for the elevated strength of ENSO (CPTEC/INPE 2015). More studies are needed, although to confirm the influence of the Peru Current and/or ENSO on *V. vulnificus* abundance in South America to determine if it is not being monitored in the area, and it could be an underreport case. Adopting a policy of notification would allow countries to better monitor the prevalence of *V. vulnificus,* and to establish control plans in order to prevent and to inform people at high risk for infection. It also would permit doctors to better diagnose and to give safety recommendations to their patients.

Overall conclusions

This review describes the presence and distribution of *V. parahaemolyticus* and *V. vulnificus* in seawater, shellfish and other marine animals, as well as clinical samples from South America. It appears that infections of *V. parahaemolyticus* have been more strongly related to seafood ingestion and have been more frequently

reported on the Pacific coast. Conversely, *V. vulnificus* is more frequently acquired by water contact with open wounds and its presence has been more heavily reported along the Atlantic coast.

The impacts of ENSO have been observed as an increase in *V. parahaemolyticus* outbreaks on the Pacific coast of South America. However, more studies are required to confirm the importance of environmental factors that are affecting the presence, concentrations and virulence of the bacteria in these areas of the continent.

Vibrio vulnificus human cases have been restricted mostly to the southeastern and southern areas of the Atlantic coast of South America, with the only exception being the report of three gastroenteritis cases in Peru and two cases in Ecuador. Uruguay had the highest number of cases in South America, but the number of cases in all of South America is low compared with the numbers of reported infections in the United States, where, according to CDC (Ratner 1987), there are approx. 96 *V. vulnificus* cases reported annually.

Peru, Ecuador and Uruguay are the only countries where human cases from both *V. vulnificus* and *V. parahaemolyticus* were recorded. Even considering possible underreporting from other areas, and the concomitant presence of environmental strains of both bacteria along the Atlantic and Caribbean coasts, the distribution of pathogenic strains should be further studied. More research is necessary in order to determine if there are environmental or genetic factors that explain the distribution of *V. vulnificus* and *V. parahaemolyticus* pathogenic strains. It is evident that the continent experiences environmental conditions that are permissive for both *V. vulnificus* and *V. parahaemolyticus*. In the face of climate change, a formal monitoring program should be instituted to quantify *Vibrio* sp., and to develop models that permit predictions of environmental conditions that are favourable for the pathogenic forms of these bacteria. This monitoring would also serve to gather sufficient information to determine the impact of ENSO and other global climate phenomena on the abundance of *V. parahaemolyticus* and *V. vulnificus*. Eventually, this would allow for the establishment of control plans to minimize new cases and reduce the impact of outbreaks, especially for *V. parahaemolyticus* in the Pacific coast during years when ENSO is forecasted to be stronger than normal such as in the spring of 2016.

The use of faster, more accurate, and virulence-specific detection approaches, such as PCR confirmation, should be considered to detect the presence of pathogenic *Vibrio* strains in environmental and seafood samples for protection of public health.

In summary, the published reports and the environmental characteristics from South America cannot be disregarded, there is a demonstrated risk of *V. parahaemolyticus* and *V. vulnificus* infection from the water and seafood. Although the total numbers of cases are very low, the morbidity, mortality and close association of these cases with seafood production and water warrant an improved monitoring and surveillance framework for all countries in South America for *V. parahaemolyticus* and *V. vulnificus*, in order to predict protect public health and prevent outbreaks. This study highlights the need for more research and a formal monitoring program of *Vibrios* in South America, both in environmental and clinical samples, particularly in the countries with the worst sets of cases as Uruguay for *V. vulnificus*, and Chile for *V. parahaemolyticus*. A continent-wide, culture-based monitoring program with collection of salinity and temperature information would be a start, and then for the mentioned countries with the most cases, a pathogenic strain-tracking system could be implemented initially in a basic way (conventional PCR). So, in order to maintain the economic, cultural and societal roles of aquaculture, public health and food safety should be closely monitored and controlled.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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