Reexamining El Niño and Cholera in Peru: A Climate Affairs Approach

IVÁN J. RAMÍREZ

Division of Social Sciences, New College of Florida, Sarasota, Florida

SUE C. GRADY

Department of Geography, Michigan State University, East Lansing, Michigan

MICHAEL H. GLANTZ

Consortium for Capacity Building, University of Colorado, Boulder, Colorado

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ABSTRACT

In the 1990s Peru experienced the first cholera epidemic after almost a century. The source of emergence was initially attributed to a cargo ship, but later there was evidence of an El Niño association. It was hypothesized that marine ecosystem changes associated with El Niño led to the propagation of *V. cholerae* along the coast of Peru, which in turn initiated the onset of the epidemic in 1991. Earlier studies supported this explanation by demonstrating a relationship between elevated temperatures and increased cholera incidence in Peru; however, other aspects of El Niño–Southern Oscillation (ENSO) and their potential impacts on cholera were not investigated. Therefore, this study examines the relationship between El Niño and cholera in Peru from a holistic view of the ENSO cycle. A ''climate affairs'' approach is employed as a conceptual framework to incorporate ENSO's multidimensional nature and to generate new hypotheses about the ENSO and cholera association in Peru. The findings reveal that ENSO may have been linked to the cholera epidemic through multiple pathways, including rainfall extremes, La Niña, and social vulnerability, with impacts depending on the geography of teleconnections within Peru. When the definition of an ENSO event is examined, cholera appears to have emerged either during ENSO neutral or La Niña conditions. Furthermore, the analysis herein suggests that the impact of El Niño arrived much later, possibly resulting in heightened transmission in the austral summer of 1992. In conclusion, a modified hypothesis with these new insights on cholera emergence and transmission in Peru is presented.

1. Introduction

It has been almost 20 years since Peru experienced the first cholera epidemic after almost a century. The outbreak, associated with the El Tor strain of *V. cholerae*, began in January 1991 along several coastal cities in Peru and then spread rapidly to neighboring South and Central American countries, infecting approximately 400 000 people in the first year of the onset (PAHO 1991; MINSA 1994, p. 43). Over the next decade, an estimated 1.2 million cases of cholera were documented in the region. Approximately 52.0% (703 000) of all cholera cases occurred in Peru (PAHO 2008).

While previous studies have shown that the diffusion of cholera in Peru was due to poor water and sanitation system capacities (PAHO 1991; MINSA 1994, p. 44; Tauxe et al. 1995; Seas and Gotuzzo 1996), the cause of the initial outbreak is still unknown. The source of emergence was initially attributed to infected persons or contaminated waste water dispelled from a cargo ship (Gangarosa and Tauxe 1992, p. 353). However, these explanations were later challenged by another hypothesis that linked the cholera epidemic to El Niño–Southern Oscillation (ENSO) (Epstein et al. 1993; Colwell 1996; Mourino-Perez 1998). ENSO, which includes El Niño (warm phase) and La Niña (cold phase), is an important source of climate variability in the Latin American region, well known for ecosystem and societal

Corresponding author address: Iván J. Ramírez, Division of Social Sciences, New College of Florida, 5800 Bay Shore Road, Sarasota, FL 34234.
E-mail: iramirez@ncf.edu

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impacts, particularly in Peru (Lagos and Buizer 1992; Glantz 2001a; Caviedes 2001). Therefore, it was proposed that marine ecosystem changes associated with El Niño led to the propagation of *V. cholerae* along the coast of Peru, which in turn initiated the onset of the epidemic in 1991 (Colwell 1996).

In earlier studies, some evidence in Peru suggested that air and water temperature changes contributed to cholera incidence via its ecological impacts on vibrios. For example, it was shown that elevated temperatures were positively correlated with diarrheal disease (Salazar-Lindo et al. 1997; Checkley et al. 2000; Lama et al. 2004) and the presence of *V. cholerae* in water sources in Peru (Franco et al. 1997; Speelmon et al. 2000; Lipp et al. 2003). It was also reported that cholera incidence was strongly associated with air and seawater temperatures during the 1997/98 El Niño (Speelmon et al. 2000; Gil et al. 2004). More recently, researchers demonstrated how ocean–atmosphere interactions influenced the transport of vibrios along the coast of Peru (Martinez-Urtaza et al. 2008).

Still, despite these advances in potential evidence, El Niño's link with cholera in Peru remains unclear. This is largely due to previous studies, which were limited in their examination of the ENSO cycle. For example, the empirical evidence so far is mainly based on temporal investigations in coastal Lima. Given the variability of ENSO's influence within Peru, the temperature–cholera association may have differed by geography (e.g., coast versus jungle). Moreover, the strongest evidence is based on one El Niño event in 1997/98. El Niño's impact on the initial outbreak has yet to be examined (Salazar-Lindo et al. 2008). Furthermore, the El Niño–cholera hypothesis is based on a period when there was disagreement about the timing of El Niño (WMO 1999; Glantz 2001a, p. 21). Conceivably, this raises questions about the definition of an ENSO event (Trenberth 1997), and the coincidence between the physical impacts of ENSO and the emergence of cholera in Peru. In addition, the potential impacts of rainfall extremes and La Niña have not been explored in relation to cholera, even though the former is an important teleconnection, and the latter is also associated with health effects in Peru (MINSA 1999a). Finally, how social factors of vulnerability, especially at the time of emergence, may have contributed to the impact of ENSO on cholera is not yet known.

Therefore, the purpose of this study is to explore these gaps of knowledge in the literature by examining the relationship between El Niño and cholera in Peru from a holistic view of ENSO. Our goal is to better understand this association by considering the many ways in which ENSO may have been linked to cholera emergence and transmission in Peru. Another goal is to provide new insights on the hypothesis in order to stimulate new avenues for potential research. As a conceptual framework, we employ a ''climate affairs'' approach to widen the scope of analysis in relation to previous studies. Climate affairs is a multidisciplinary concept used to understand climate–society interactions worldwide (Glantz and Adeel 2000; Glantz 2003; CCB 2011) and, in particular, societal impacts associated with ENSO (Glantz 2001a,b). From a climate affairs orientation, we incorporate ENSO's multidimensional nature to generate new hypotheses about the ENSO and cholera association in Peru. In addition to a literature review we also describe data about the epidemic and ENSO collected during fieldwork in Peru in 2008 and 2009. These data were obtained from documents including health data and bulletins, newspaper archives, and reports from Peruvian nongovernmental and governmental institutions.

Following this introduction is a description of the climate affairs approach and its application in this study. The second section is a review of the El Niño–cholera hypothesis in Peru. The third section recounts the current evidence in support of the hypothesis and a link. Here, our focus is the literature in Peru. The fourth section reexamines the relationship between El Niño and cholera in Peru using a climate affairs approach. We begin by revisiting the temperature association and then explore the definition of an ENSO event, which includes an assessment of El Niño's timing and impacts in Peru; rainfall extremes; the La Niña factor; and social vulnerability. In the final section, we summarize our findings and conclude by presenting a modified hypothesis with new insights on cholera emergence and transmission in Peru.

2. A climate affairs approach

Climate affairs is a holistic approach to understanding the many facets of climate (e.g., averages, extremes, variability, and change) and how societies interact with climate phenomena (Glantz and Adeel 2000; Glantz 2003; CCB 2011). Its basic components encompass, but are not limited to, (a) climate science, (b) climate impacts, (c) climate economics, (d) climate politics and policy, and (e) climate ethics. Thus, it emphasizes the importance of multidisciplinary efforts across physical and social sciences and humanities. It also recognizes local and regional knowledge to understanding climate–society interactions (Glantz 2003). The concept evolved from the collaborative research and program activities of Dr. Michael H. Glantz, beginning in 1974 at the Environmental and Societal Impacts Group at the National

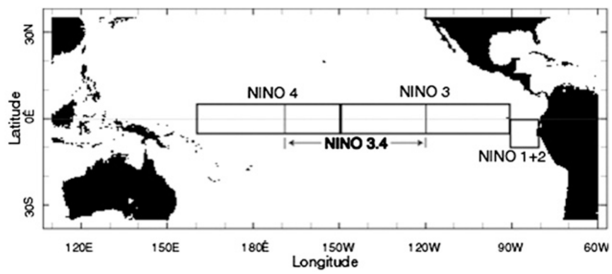

FIG. 1. Map of Niño regions. Source: NOAA (http://www1.ncdc.noaa.gov/pub/data/cmb/enso/Niño-regions.gif).

Center for Atmospheric Research (NCAR). It later came to fruition as a research, training, and education initiative in 2003. Currently, it continues to be implemented through the Consortium for Capacity Building and its partners at the University of Colorado at Boulder.

Until now, the application of climate affairs has generally focused on capacity building efforts with universities, research institutes, multilateral organizations, and governments around the world. Among these activities, the transfer of knowledge about ENSO, including forecasting, sectoral impacts, and societal responses, has been a central theme (Glantz 1997, 2001b, 2002). In this study, the concept of climate affairs is utilized as a research lens to highlight and integrate various aspects of ENSO that may have been important for cholera emergence and transmission in Peru. Specifically, we draw upon the following elements of ENSO knowledge: ENSO science, impacts, economics, politics, and ethics.

a. *ENSO science*

ENSO is a quasi-periodic phenomenon, composed of warm (El Niño), cold (La Niña), and neutral phases and ocean–atmosphere interactions that are basinwide (i.e., equatorial Pacific Ocean) and local (e.g., Peru). ENSO neutral refers to ''average'' sea surface temperature (SST) conditions in the equatorial Pacific that do not qualify as warm or cold phases (NOAA 2011). ENSO is monitored across the equatorial Pacific Ocean basin in four delimited Niño regions known as Niño-1+2, Niño-3, Niño-3.4, and Niño-4 (Fig. 1). Interpreting the characteristics of phases can vary depending on the definition of an event, the variables chosen to measure ENSO, and the Niño region of analysis (Trenberth 1997; IRI 2008). Therefore, careful attention should be paid to how events are characterized because they may affect the interpretation of quantitative associations between ENSO and health outcomes (Kovats et al. 2003).

b. *ENSO impacts*

ENSO's influence on local climate (e.g., temperature and rainfall teleconnections) can vary within different regions of Peru and by season. Geographically, the most notable effects are storms and rains along the northern coast, an area that is typically arid, or below average rains in the southern and central Andes (Lagos et al. 2008; SENAMHI 2009). Although probable, teleconnections are not deterministic because each event is unique and can vary in how it develops from one period to another (Wyrtki 1975). However, it is possible to foresee some potential effects on ecosystems and societies based on direct and indirect observations of the environment, statistical assessments, computer modeling, and historical reports of past events (Glantz 2001a, 163–173; McPhaden et al. 2006). In Peru, they may include marine ecosystem changes due to the disruption of upwelling processes, terrestrial ecosystem changes (e.g., vegetation, insects, and animals), impacts on fisheries and agriculture, and damaged built environment due to flooding (e.g., energy, water, and sanitation systems) (Glantz 2001a). When ecosystem change co-occurs with collapse of infrastructure, human exposure to infectious diseases can increase during ENSO events (Gueri 1984; Valverde 1998; PAHO 1998a; Kovats et al. 2003).

c. *ENSO economics, politics, and ethics*

ENSO impacts on society are contingent not only on the geographic location of teleconnections, but also on social, economic, and political factors of vulnerability that contribute to climate-related disasters (Glantz 2003, p. 253; Cutter et al. 2009). In Peru, the government and its population faced a number of challenges when cholera emerged that included humanitarian emergencies (UNDRO 1990a), economic restructuring, and an energy crisis (El Tiempo 1991; Youngers 2000), not to mention the high rates of population deprivation that existed at the time (PAHO 1991). Assuming El Niño's impacts were felt in Peru in 1991, in addition to triggering the epidemic as has been suggested, climate may also have exacerbated the severity of cholera transmission. It is important to highlight this context in the explanation of the El Niño and cholera association because it places the cholera epidemic at the intersection of ENSO and society interactions, crossing disciplinary boundaries (McPhaden et al. 2006). Tangentially, it also alludes to potential issues of ''climate justice'' (e.g., equity of impacts and differential vulnerabilities) that may arise from these interrelationships (Glantz and Jamieson 2000; Ramírez 2012).

3. The El Niño–cholera hypothesis

Since the emergence of cholera in Peru, El Niño and climate have been associated with cholera transmission.

The link was first proposed by Epstein (1992) in a letter to the editor at *The Lancet*. According to Epstein, once *V. cholerae* was introduced to the coastal waters of Peru, it harbored among algae and plankton blooms and proliferated because of ''warming,'' possibly due to El Niño or global climate change. Furthermore, it was thought that human activities (e.g., eutrophication due to untreated sewage waste and agricultural activities) had also contributed to ''enhanced'' blooms. Thus, it was suggested initially that cholera emerged as a result of climate and human-related processes (Epstein 1993; Epstein et al. 1993, 1994). As the mode of transmission, Epstein proposed that human consumption of shellfish that feed on blooms was the initial pathway (Epstein 1992, 1993). While El Niño seemed a likely reason for the epidemic, it was also thought that further investigations were needed before causation could be deduced.[1]

In 1996 El Niño was hypothesized, with greater confidence and explanation, as the most important driving factor in the epidemic (Colwell 1996). Motivating this postulation were three ideas. First, the passing ship hypothesis was dismissed on the grounds that a single ship could not explain multiple entry points along the Peruvian coast. Second, there was growing evidence of positive relationships among plankton, *V. cholerae*, and cholera outbreaks in Bangladesh (Colwell 1996); furthermore, *V. cholerae* was shown to survive freely in estuarine environments, lying dormant when environmental conditions are less favorable and increasing in concentrations when conditions are optimal (Colwell and Spira 1992; Colwell 1996). Third, there were reports of an ''extraordinary'' El Niño, which lasted from 1990 to 1995 (Trenberth and Hoar 1996), which was believed to coincide with the cholera epidemic. Therefore, it was proposed that an El Niño influenced sea surface temperature anomalies in the equatorial Pacific Ocean, which in turn promoted the abundance of plankton blooms (harboring bacteria) that led to cholera transmission at multiple locations on the coast (Epstein 1992; Colwell 1996; Seas et al. 2000). It was also suggested that nutrient runoff (due to heavy rains) may have contributed to the rise in blooms. The mechanism by which blooms invaded the inland coast was intrusion of warm waters (Colwell 1996), either by rising sea levels and/or storm surges with the arrival of El Niño. Furthermore, it was suggested that the original source of *V. cholerae* was contaminated plankton from Asia, which may have been transported via eastward-flowing ocean currents induced by El Niño (Colwell 1996; Mourino-Perez 1998).

In 2000, the hypothesis offered by Colwell (1996) was sustained in two important studies. The first study (Seas et al. 2000) identified several clinical cases of cholera preceding the onset of the epidemic in several coastal cities. The finding was instrumental because it suggested that the introduction of cholera occurred prior to the arrival of the passing ship, dismissing that explanation and embracing the El Niño hypothesis (Epstein 1992; Colwell 1996; Mourino-Perez 1998). Furthermore, there was laboratory confirmation of *V. cholerae* in water sources, including sewage, rivers, lagoons, and irrigation (Gomez Pando and Pineda 1991; Madico et al. 1996) preceding cholera outbreaks in Lima, Peru (Franco et al. 1997; Speelmon et al. 2000). According to Franco et al. (1997), elevated temperatures precipitated a rise in bacteria, which then amplified once human fecal contamination began to increase. Importantly, a second study demonstrated a quantitative relationship between ENSO and cholera from 1980 to 1998 in Bangladesh. Cholera transmission was explained by ENSO's teleconnected influence on local air–water temperatures and, subsequently, the intrusion of plankton blooms onto the coastal shore (Pascual et al. 2000).

4. Recounting the evidence in Peru

a. Air temperature

Following the initial outbreak in 1991, several studies found temperature-related associations that supported an El Niño and cholera link in Peru. It was shown that diarrheal diseases including cholera correlated with the warmest months (e.g., austral summer, December–March) in the 1990s. Specifically when temperatures increased to greater than 19.5°C there was also an increase in cholera incidence (Madico et al. 1996; Speelmon et al. 2000). Furthermore, it was estimated that an increase of 1°C in mean air temperature led to increased risk ($\sim$8.0%) of diarrhea in children and adults (Checkley et al. 2000; Lama et al. 2004).

El Niño–related risk was not observed until the onset of the 1997/98 El Niño, which began rapidly developing in April 1997 (WMO 1999, 29–38). Subsequently, record air temperatures were observed in Peru from May to December, which meant that Peruvians would not experience winter (Bell and Halpert 1998). The impacts on diarrhea and cholera risk were severe. In north Lima, diarrhea rose by 35.0% in children and 47.5% in adults during the winter of 1997 (Salazar-Lindo et al. 1997; Lama et al. 2004). In terms of cholera, after a decline in

[1] At the Emerging Diseases Workshop in 1993 (Woods Hole, MA), Dr. Rita R. Colwell, while supportive of the potentiality of a climate mechanism, expressed her concerns about assuming causation without further quantitative studies.

1996, cases began to noticeably rise in July 1997. The first outbreaks were reported in southern Peru, where a cholera emergency was declared by September (MINSA 1998a).

According to the Ministerio de Salud (MINSA; the Ministry of Health), the greatest burden of diarrheal disease was observed during the austral summer in 1998 (MINSA 1998d; Huanca 2004). Speelmon et al. (2000) reported that 88.0% of cholera cases in Lima occurred from January to May. It was also estimated that El Niño conditions contributed to an excess of 6225 daily admissions of children with diarrhea (Checkley et al. 2000). Overall in 1998, the number of cholera cases in Peru rose by 112.0% from the previous year (MINSA 2008). MINSA attributed the rise in cholera and diarrheal disease to El Niño (MINSA 1998a,c, 2000).

b. Sea surface conditions

Other studies in Peru focused on the effects of sea surface conditions on *V. cholerae* and cholera incidence along the coast of Peru. In one study, Lipp et al. (2003) found that *V. cholerae* was significantly correlated with air temperature increases every January to March from November 1998 to March 2000. Furthermore, *V. cholerae* was detected in plankton samples at three coastal sites: Trujillo (north), Lima (central), and Arequipa (south). Interestingly, coastal seawater temperature was not a significant factor. In a later study, Gil et al. (2004) revisited the same study areas as Lipp et al. (2003) but included one additional site at Callao (considered part of greater Lima). They also measured environmental variables in relation to cholera incidence and extended the time period to begin in October 1997. In this study, monthly cholera incidence was strongly associated with elevated seawater temperature during the austral summer of 1998. As in the previous study, *V. cholerae* was detected in the seawater; this supported the existence of a coastal environmental reservoir linked to seawater changes (Gil et al. 2004).

More recently, the hypothesis has gained further support in a study that modeled equatorial waves during the 1997/98 El Niño with the occurrence of a proxy vibrio strain called *Vibrio parahaemolyticus* (Martinez-Urtaza et al. 2008). Using sea height anomaly and upper ocean heat content to represent El Niño, the authors showed that the distribution of index cases of cholera followed a pattern along the west coast of South America, similar to the oceanic parameters. Coincidentally, this pattern also resembled locations where cholera was first identified in 1990/91, according to Seas et al. (2000). This was the first study to demonstrate how vibrios and their reservoirs may have traveled via ocean waves and upon arrival (i.e., against the continental boundary) dispersed along the coastal environment of Peru (Martinez-Urtaza et al. 2008).

c. Summary

In sum, descriptive and statistical studies have shown a climate link, both direct and indirect, with cholera, *V. cholerae*, and environmental reservoirs in Peru. The associations were based on inland and coastal water temperatures during the austral summer and climate thresholds which suggested that elevated temperatures led to a greater number of diarrheal diseases in the 1990s. With respect to El Niño, there was an apparent association with the 1997/98 event. While these studies provided some evidence for a climate–cholera connection in Peru, there are still many unanswered questions. In the following section, we explore several factors that we argue are critical to understanding El Niño's link to cholera emergence and transmission in Peru.

5. Reexamining El Niño and cholera in Peru

Here, we use a climate affairs approach to reexamine the association between El Niño and cholera in Peru using a broader conception of ENSO. Our discussion begins by revisiting the temperature-related association followed by an exploratory investigation that highlights the following ENSO characteristics: the definition of an ENSO event, rainfall extremes, the La Niña factor, and social vulnerability.

a. Revisiting the temperature association

While a temperature-related association is plausible because of its potential impact on the reproduction of vibrios, its generalization in Peru is limited by its geographic scope. This is due to the fact that previous findings in Peru were generally based on temporal observations in Lima, which is located on the central coast. Given the country's diverse physical regions, which range from a low-lying coast to highlands and jungle in the east (Fig. 2), it is possible that there may have been regional variations of the temperature association within Peru. For instance, in Loreto, a jungle region located in northeast Peru, seasonal patterns of cholera were indeed different from the Lima time series (Fig. 3a). In Lima, peaks in cholera cases were observed from February to April from 1993 to 1998. In contrast, cholera increased in Loreto from midsummer to early autumn (July to October) in 1993, 1995, and 1998; there were also peaks in May of 1993 and 1998 (Fig. 3b). In general, the temporal pattern of cholera in Loreto was less well defined and lagged in time compared to Lima. These differences may reflect, particularly in the case of 1998, the geography of El Niño–related teleconnections. For example, on the central coast El Niño may contribute to warmer and wetter than average conditions from June to August

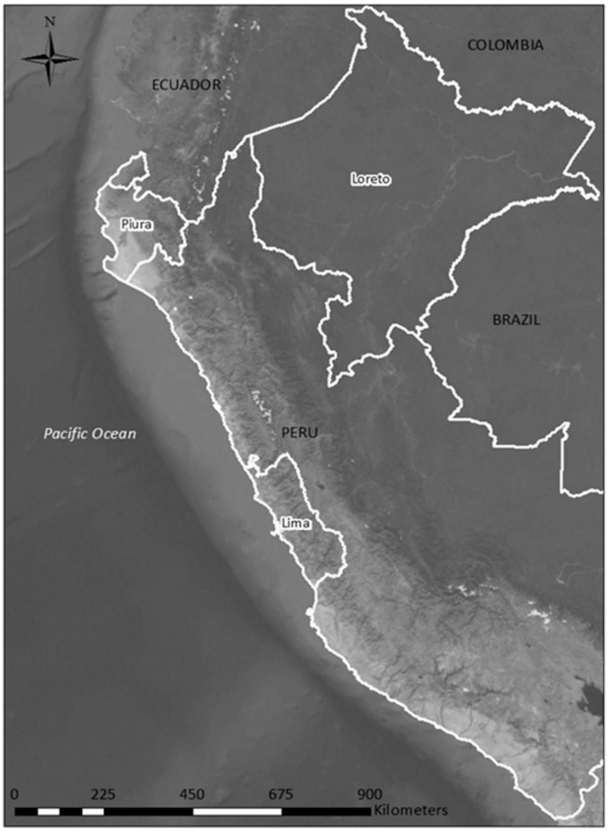

FIG. 2. Physical map of Peru highlighting three departments: Lima (central coast), Loreto (north jungle) and Piura (north coast, discussed in section 5c). Map produced by Iván J. Ramírez. Source: University of Piura (private).

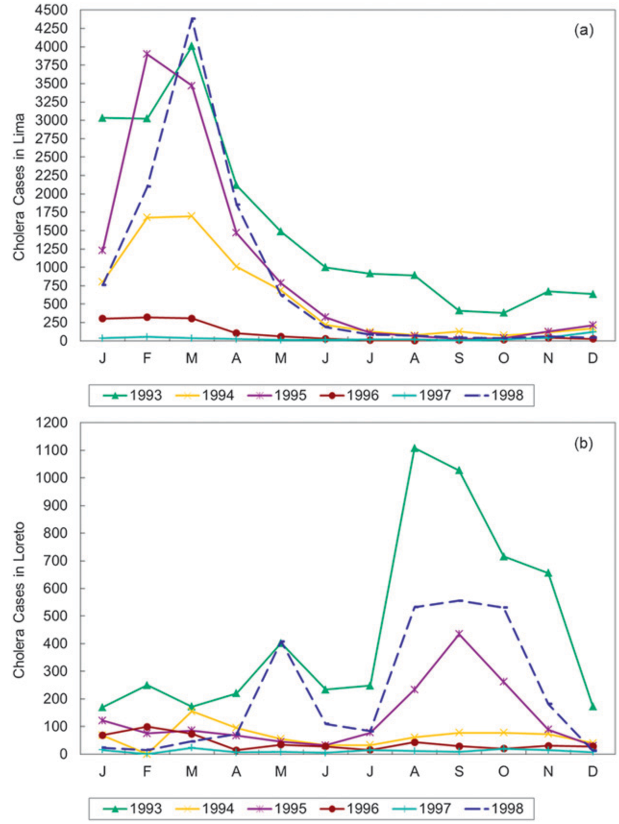

FIG. 3. Cholera cases (suspected and confirmed) by month for each year from 1993 to 1998 in the Departments of (a) Lima and (b) Loreto. Source: General Office of Epidemiology, MINSA, Lima, Peru.

(SENAMHI 2004); in the Amazon, it may contribute to rainfall deficit in November and December (Marengo 1999; Marengo et al. 2008). Importantly, these regional differences suggest not only that different temporal pathways might exist, but also that different climate pathways may have impacted cholera transmission.

The temperature-related association is also limited because El Niño's link to cholera is based on one event in 1997/98. For example, even though Lama et al. (2004) found a strong association between El Niño and diarrheal disease in 1998, it was also shown that prior El Niños were not statistically significant.[2] One reason for this outcome may have been the short length of time series (i.e., 1991–98), which seems to limit studies in Peru because they have generally been cross-sectional and do not include the initial outbreak time segment.

b. *Definition of an ENSO event*

One important assumption of the El Niño–cholera hypothesis is that El Niño conditions were present in order to impact the transport and reproduction of contaminated plankton off the coast of Peru, potentially from October 1990 to January 1991 [i.e., based on Seas et al. (2000)]. Although an El Niño was suspected during that time, there were also varying reports about the timing of El Niño(s) in the early 1990s. For example, for some researchers it was the longest El Niño of the century, lasting five years from 1990 to 1995 (Trenberth and Hoar 1996). For others, it lasted only from September 1991 to July 1993 (McPhaden 1994; Kessler and McPhaden 1995). It was also estimated that three events took place during 1) March 1991 to June 1992, 2) 1993 (February/March to October), and 3) 1994 (June to November) (Goddard and Graham 1997). Importantly, Peruvians questioned the view of an El Niño in 1991

[2] It is important to note that it is unclear how Lama et al. (2004) identified El Niños. According to their study, which cited data from NOAA, the onset dates were 1) September 1991, 2) April 1993, and 3) September 1994.

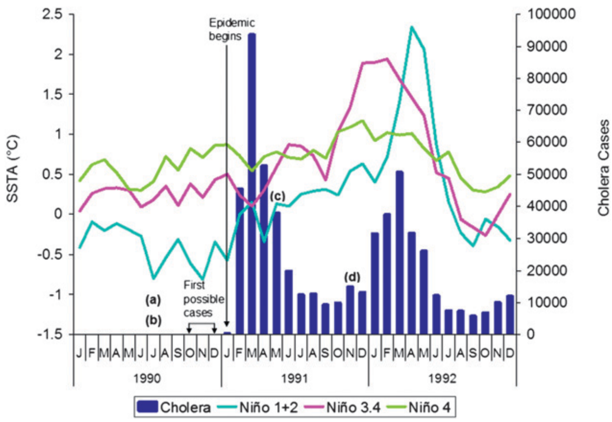

FIG. 4. Monthly SST anomalies (°C) in three Niño regions (1+2, 3.4, and 4) are plotted in relation to monthly cholera cases (suspected and confirmed) in Peru from 1990 to 1992. SST anomalies are based on the period 1971–2000. See Table 1 for ENSO events a–d. Sources: General Office of Epidemiology, MINSA, Lima, Peru, and NOAA (http://www.cpc.noaa.gov/data/indices/).

TABLE 1. Listings of El Niño and La Niña events from 1990 to 1992 as defined by SSTA exceeding ±0.5°C threshold. Each qualifying event is given along with the Niño region, phase, and start and end dates.

Event	Region	Phase	Begin	End	Duration
A	Niño-4	El Niño	Jul 1990	Aug 1992	26
B	Niño-1+2	La Niña	Jul 1990	Dec 1990	6
C	Niño-3.4	El Niño	May 1991	Jul 1992	15
D	Niño-1+2	El Niño	Nov 1991	Jun 1992	8

because they noticed they were catching near-record-setting anchovy landings, highly unlikely during a warm episode (Flores 1998; Glantz 2001a, p. 21). Therefore, two fundamental questions about the hypothesis are raised. The first is whether an El Niño was in development at the time of emergence. The second is whether El Niño's effects were observed in Peru. The latter question is important because it may explain the transport and coastal intrusion component of the hypothesis.

To address the question about El Niño's timing, we explored the definition of an ENSO event, which determines the characteristics of an episode including its beginning and end dates (Trenberth 1997). The definition is important because it may affect how one characterizes the relationship between El Niño and cholera emergence, which is dependent on whether the two events coincided in time. To illustrate this point, we compared sea surface temperature anomalies (SSTAs) in three Niño regions with monthly cholera cases in Peru from 1990 to 1992. As a definition, we chose the operational one used by the National Oceanic and Atmospheric Administration (NOAA), which is commonly employed to identify an event (see NOAA 2012). Figure 4 shows the SSTA and cholera time series and Table 1 lists El Niño and La Niña events, respectively. According to the Niño-4 region, the first possible cases of cholera [identified by Seas et al. (2000)] and the onset of the epidemic occurred in the midst of a prolonged El Niño (26 months) that developed in July 1990 (event a). The timing in the Niño-4 region, which represents the western equatorial Pacific, sustains the El Niño–cholera hypothesis and suggests a time delay of several months between El Niño and cholera emergence. On the other hand, regions Niño-3.4 and Niño-1+2, which represent the central and eastern equatorial Pacific, indicate that El Niño conditions followed the initiation of the epidemic, beginning in May (event c) or November (event d) of 1991. Using Pearson's correlation analysis, we explored these associations further and found the strongest link between cholera and SSTA in the Niño-1+2 region ($r = 0.42$, p value $= 0.010$). This finding is important because the Niño-1+2 region includes coastal and equatorial upwelling near Peru and Ecuador (Glantz 2001a, p. 60), which exhibited non–El Niño conditions preceding and during the initial outbreak in January 1991 (see Fig. 4 and Table 1d). Thus, it suggests that ENSO neutral or La Niña conditions rather than El Niño may have contributed to cholera emergence in Peru.

EL NIÑO'S EFFECTS IN 1991?

To explore the timing of El Niño further, we examined the literature to identify El Niño–related impacts on ecosystems and climate in Peru. One potential effect we looked for is the impact on anchovy, pelagic fish whose habitat and food supply are altered when upwelling weakens during El Niños (Chavez et al. 2008). According to the Peruvian Marine Institute, 1991 was described as a year with cold coastal waters and good for anchovy catch (Pizarro 1999), which implied that average SST conditions were present off the coast of Peru and that upwelling and marine biological productivity was normal too. We also looked for ecosystem impacts in other areas of the eastern Pacific Ocean. For example, biological impacts (associated with El Niño) were reported off the coast of Costa Rica (e.g., coral bleaching and mortality) in March to April 1992 (Jiménez and Cortés 2001), in the Galapagos (e.g., penguin populations declined associated with lower food supply) from 1991 to 1993 (Hernan Vargas et al. 2006), and off the coast of Chile (e.g., changes in planktic fauna species) from November 1991 to March 1992 (Marchant et al. 1998). With regard to impacts on local climate (teleconnections), NOAA reported intense rains on the north coast of Peru during the austral summer of

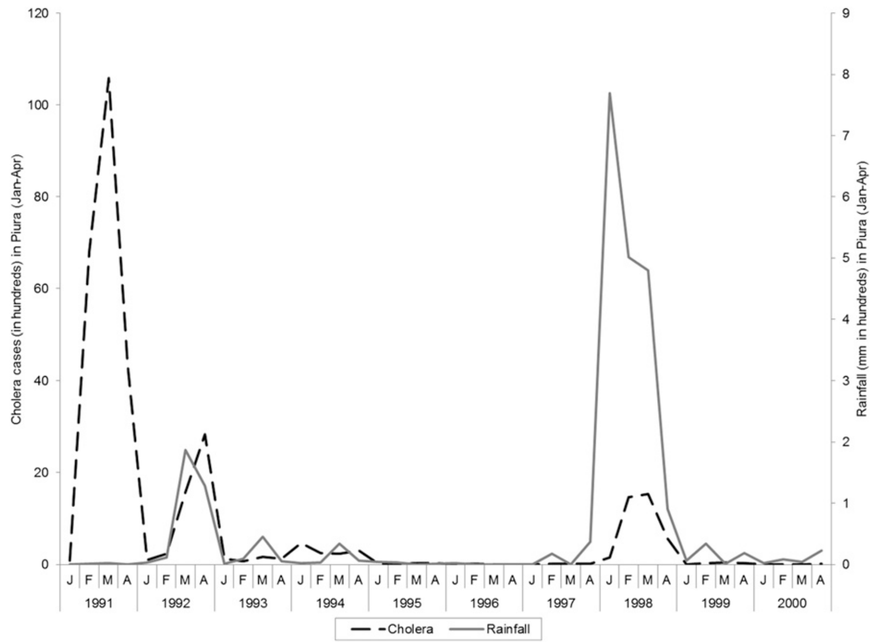

FIG. 5. Cholera cases (in hundreds) and rainfall (mm in hundreds) for January to April for 1991 to 2000 at the Miraflores meteorological station (5.17°S, 80.6°W; 30 m a.s.l.) in the subregion of Piura, Peru. Sources: MINSA, Piura and University of Piura (private), Peru.

1992. Apparently, an El Niño that began developing in mid-1991 was in a mature phase (NOAA 1992, 1993). Supporting the NOAA observations were local newspaper accounts that documented storms, inundations and disasters (El Tiempo 1992a,b). As mentioned previously, the north coast of Peru is strongly associated with El Niño and rains (Lagos et al. 2008) and is therefore a good indicator of a warm event in Peru. Although speculative, biological changes in marine ecosystems along with physical impacts in mid-to-late 1991/early 1992 support the idea that El Niño may have followed rather than led the onset of the cholera epidemic in 1991.

c. *Rainfall extremes*

In addition to temperature, rainfall extremes are important El Niño teleconnections. In Peru they may have been a pathway for cholera transmission. Rainfall extremes can influence the concentration and transport of *V. cholerae* and environmental hosts (Ruiz-Moreno et al. 2007; Hashizume et al. 2008; Mendelsohn and Dawson 2008; Akanda et al. 2009; Reiner et al. 2011; Bertuzzo et al. 2012; Rinaldo et al. 2012). In particular, heavy rains can contribute to nutrient runoff, which in turn may positively affect plankton blooms in coastal areas (Constantin de Magny et al. 2008; Jutla et al. 2011); furthermore, wind-driven rain can drive coastal waters with plankton inland, thereby increasing human interactions with *V. cholerae* (Constantin de Magny et al. 2008). Heavy rains can also increase cholera risk through flooding and overflow of rivers and subsequent contamination of the water supply (Kovats et al. 2003; Ruiz-Moreno et al. 2007; Akanda et al. 2009; Reiner et al. 2011). For example, during the recent cholera epidemic in Haiti, intense rains increased incidence in 2010–11 by enhancing surface runoff and transport of fecal matter from open latrines to inland water sources (Rinaldo et al. 2012). In Peru, cholera may have spread via rains in low-lying areas in the department of Piura (see Fig. 2), which reported index cases in 1991 (Ries et al. 1992). Figure 5 compares cholera cases with rainfall (in mm) in January to April from 1991 to 2000 in a subregion of Piura (located 860 km north of Lima). Cholera and rainfall increases in Piura were clearly evident in 1992 and 1998. Furthermore, during the onset of cholera and the first quarter of 1991 (i.e., October 1990 to April 1991), total rainfall in Piura was 7 mm compared to 329 mm in 1992 and 1842 mm in 1998. Together, these observations along with local reports suggest that flooding, possibly via the breakdown of water and sanitation infrastructures, led to enhanced cholera transmission in Piura (El Tiempo 1992a,b; PAHO 1998b; MINSA 1998b,d). Although elevated temperatures may have contributed to cholera incidence in Lima, heavy rains may have been a more important factor in northern Peru. Alternatively, rainfall deficit may have contributed to cholera transmission as well

(Codeco 2001; Pascual et al. 2002; Hashizume et al. 2008; Akanda et al. 2009). For example, in the jungle region of Loreto, which we discussed earlier, low river levels were associated with cholera risk in places of poor water and sanitation infrastructures (MINSA 1995, 1998b). A dry season along with elevated air temperatures can impact bacteria and increase cholera risk due to limited availability of potable water (Codeco 2001; Huq et al. 2005).

d. The La Niña factor

La Niña, the cold phase of ENSO, is another pathway that may have impacted cholera transmission in Peru. La Niña enhances average SST conditions (e.g., cold) in the eastern equatorial Pacific Ocean. Therefore, via its positive influence on upwelling, which brings nutrients to the surface waters, La Niña may have contributed to cholera emergence (we discuss this pathway within a broader context of the El Niño–cholera hypothesis in the final section). As well, La Niña may have increased cholera risk through its own set of teleconnections (Ropelewski and Halpert 1987; Ordinola 2002; NOAA 2011). During La Niña, climate in Peru is drier than average on the northern coast (Ordinola 2002) and wetter than average in the southern and central Andes, particularly in the Altiplano, which borders Bolivia and Chile (Sperling et al. 2008). Following the 1997/98 El Niño, rain-related disasters were reported in the highlands in February 1999. Subsequently, populations were affected by floods and many were in need of assistance with food, water, and sanitation (MINSA 1999a). La Niña conditions along with those in the tropical Atlantic were blamed (MINSA 1999b).

e. Social vulnerability

When cholera emerged in 1990/91, the Peruvian government was already addressing complex emergencies, which began earlier that year in late summer/early winter. The first was an earthquake in northeast Peru, which affected 70 000 and injured 1500 people in May 1990 (UNDRO 1990a). The second was an agricultural state of emergency declared across highland regions in June 1990. It was reported that over 2 million subsistence farmers were gravely affected by an ongoing drought and cold extremes experienced in 1989, reportedly a La Niña year; consequently, food and water supplies were in decline (UNDRO 1990b). That austral winter was also the beginning of ‘‘Fuji shock,’’ a set of economic reforms implemented by the then-elected President Alberto Fujimori (Brooke 1990). As a result of these policies, public infrastructure and services were reduced including those in the health sector, where resources were already limited (Cueto 2001, 107–137). Furthermore, the country was contending with an energy crisis (El Tiempo 1991; Nash 1991; Youngers 2000). Power outages affecting water and sanitation plants and residents were reported (El Tiempo 1991). Ultimately, these events, which included public policies, may have aggravated the preexisting living conditions of the population, who lacked immunity to cholera and lacked adequate water and sanitation infrastructure.[3] If ENSO and its influence on climate and society had an impact on cholera in Peru, it could not have occurred apart from social vulnerability.

6. Summary and conclusions

Although cholera was eradicated in Peru in 2002 (MINSA 2005), it remains a potential threat to the country and region because of the recent emergence in Haiti, which was also associated with ENSO (Enserink 2011). Therefore, this research is timely, and supports other studies that seek to understand how climate variability affects cholera epidemics (Pascual et al. 2000; Koelle et al. 2005; Constantin de Magny et al. 2007; Reiner et al. 2011; Reyburn et al. 2011). In sum, this study examined the relationship between El Niño and cholera in Peru using a climate affairs approach. From this holistic view, we explained cholera incidence from a broader conception of ENSO, one which includes not only temperature but also geographic variability, how we define ENSO, the impacts of rainfall and La Niña, and underlying social dimensions. In doing so, we linked ENSO to cholera in Peru through multiple pathways.

Specifically, our study showed that a temperature–cholera association may have differed temporally between regions because of geography and the variability of El Niño teleconnections within Peru (e.g., varying cholera peaks between coast and jungle). It was also shown that another pathway may have been rainfall extremes (e.g., torrential rains on the north coast), which impacted infrastructure, river levels, and water supply, leading to increased exposure to cholera. Heavy rains were likely a critical factor in 1992 and 1998 during El Niños. Importantly, when we explored the definition of an ENSO event, our analysis revealed that cholera may have emerged in Peru during ENSO neutral or La Niña conditions rather than El Niño conditions. Our interpretation, which disputes the presence of El Niño in the prior and onset months of the cholera epidemic, is

[3] In 1991 approximately 45.0% of Peru’s population did not have access to clean water and 59.0% were without sanitation services. In rural areas, conditions were much worse; there, less than one-third of the population had access to clean water and other basic services (PAHO 1991).

supported by a significant correlation between cholera and sea surface temperatures in the Niño-1+2 region, an area proximate to the Peruvian coast. Our study also highlights that biological and physical evidence of El Niño was not observed until the end of 1991, which supports further that El Niño did not coincide with the initial epidemic. In light of these findings, our study overall suggests that the cholera epidemic in Peru was likely already underway by the time El Niño developed in 1991. Therefore, the warm phase of ENSO could not have impacted the onset or initiation of the cholera outbreak in Peru.

However, our findings do not preclude an ENSO or climate contribution. Instead, our study suggests that non–El Niño conditions may have played a contributive role during cholera emergence. Non–El Niño conditions are indicative of rich biological productivity due to upwelling processes that may have positively influenced plankton reservoirs in late 1990 and/or early 1991. Concurrently, multiplication of plankton and vibrios in the coastal zone may have occurred because of eutrophication due to human activities (e.g., agriculture runoff and sewage dumping) (Epstein 1992, 1993). Vibrios may have also increased in population due to seasonal warming of SST and air temperatures due to the onset of summer in December. This association is plausible given that cholera and diarrheal disease were correlated with warmer months following the initial outbreak in January 1991 (Salazar-Lindo et al. 1997; Checkley et al. 2000; Speelmon et al. 2000). Human transmission may have taken place by consumption of contaminated seafood (e.g., shellfish), which was an initial vehicle of infection in Peru (MINSA 1994), and suggested originally by Epstein (1993). Once cholera was introduced to the coastal inland, local transmission was amplified by human fecal contamination (Franco et al. 1997), and then propagated under the social vulnerability context, which we described earlier and was reported widely (PAHO 1991; MINSA 1994; Cueto 2001, 107–137).

It was more likely that El Niño's impact arrived much later than the initial outbreak in 1991, contributing to cholera transmission in the austral summer of 1992 via the mechanism proposed by Colwell (1996). More specifically, this multiple pathway occurs as follows: with the arrival of Kelvin waves, which carry warm waters from the western Pacific, SST increased and sea level rose along the coast of Peru. The pool of anomalous warm waters positively affected the reproduction of vibrios living in the coastal zone. With respect to plankton reservoirs, their proliferation was potentially influenced by three factors associated with nutrient enrichment. The first factor is related to human activities (Epstein 1993; Epstein et al. 1993), which we mentioned previously. The second factor is nutrient enrichment by runoff due to high river discharge (Jutla et al. 2011), which may occur during strong El Niños (Lavado Casimiro et al. 2012). Jutla et al. (2011) report that this is the main driver of plankton blooms rather than rising SST in coastal Bangladesh, and therefore this may have been an important pathway in 1992. The third factor is upwelling, which may appear counterintuitive at first. However, while it is likely that equatorial upwelling (near Ecuador) is negatively affected during El Niños (NOAA 2011), it is not always true for coastal upwelling near Peru. There, upwelling diminishes by geographic extent (shifting closer to the coast) but continues to persist and even intensify during warm episodes as winds blowing toward the coast increase (Enfield 1981). It explains why plankton species could remain high under warm anomalous SST conditions during past El Niño events (Barber and Chavez 1983; Chavez 1996). Once vibrios and reservoirs multiplied on the coast, rising sea level heights and storm surges led to coastal intrusion transporting organisms to the inland, where teleconnections (e.g., warmer air and water temperatures and heavy rains) affected their reproduction and distribution. Impacts on cholera, however, varied by geographic region, depending on the hydrology (Akanda et al. 2009; Bertuzzo et al. 2012; Rinaldo et al. 2012), infrastructure, and immunity levels of the population (Koelle et al. 2005; Mari et al. 2012; Rinaldo et al. 2012). Within a poor infrastructure context, rainfall impacts led to collapse of water and sanitation systems along the coast, more likely in the north where El Niño impacts are strongest, resulting in heightened transmission in the summer months of 1992.

Although we cannot explain the initial source of cholera emergence in Peru, we can assume that if El Niño conditions were not present in the eastern equatorial Pacific during the initial outbreak, it is unlikely that distant vibrios would have traveled via El Niño–related ocean waves in 1990/91. The answer to the origins of cholera in Peru is more likely to be found in an explanation that combines information from existing hypotheses (both human importation and climate) with current advancements in the microbiology and genetics of *V. cholerae*. Most recently, the latter has helped confirm the source of cholera emergence in Haiti (Chin et al. 2011), which attributed the introduction to foreign importation by asymptomatic individuals (Piarroux et al. 2011; Cravioto et al. 2011). In Peru, the idea of human importation remains a viable pathway to investigate given a recent genetic study that suggests the Latin American strain of *V. cholerae* came from Africa, possibly by human migration (during some undefined time period) following the arrival of the seventh pandemic in that continent in the 1970s (Lam et al. 2010). It may help explain how *V. cholerae* may have become indigenous to the coast of Peru prior to the emergence in 1990/91 and then ''evolved independently'' through genetic transfer between *V. cholerae* strains (Blokesch and Schoolnik 2007; Nusrin et al. 2009).

In conclusion, it is hoped that this research will stimulate new points of investigations in Peru and in other areas where cholera is emergent or reemerging. To begin, future work should consider each dimension of ENSO because each can serve as a potential pathway that links ENSO to disease incidence. Moreover, quantitative studies are necessary to estimate the relationship between each pathway (described in this study) and cholera. For example, in order to better understand the temporal patterns of cholera by region, it may be useful to utilize time series methods, such as wavelet, to identify changes in the climate–cholera relationship across time–frequency space (Cazelles et al. 2007), including lag effects. Such a task will require researchers to examine the entire record during which cholera was present in Peru (1990/91 to 2002) in order to capture ENSO's quasi-periodicity. Furthermore, the characteristics of various ENSO events should be explored to understand how an event's evolution and magnitude (e.g., degree of impact on SST), which is unique to each event, can impact transmission. It may explain why cholera did not emerge during the second strongest event of the century, which occurred in 1982/83, when health impacts were reported in Peru (Gueri 1984). Equally important is the necessity to consider the modifying effects of nonclimatic factors, which may also address cholera emergence and transmission questions in Peru. These factors include social variables, such as socioeconomic status and infrastructure (Sasaki et al. 2009; Emch et al. 2010; Mari et al. 2012), immunity, which influences biological susceptibility (Koelle et al. 2005; Rinaldo et al. 2012), and human migration, which enables connectivity between infected and susceptible populations (Koelle et al. 2005; Mari et al. 2012).

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