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# Cholera Outbreaks and Ocean Climate

WHAT I SHALL DO IN THIS BRIEF PAPER, RATHER THAN JUMP INTO THE ring with gloves to join the debate on science and policy, is put a human face on one of the most serious issues we are discussing: the complexity of climate change.* We need to understand very clearly that when one discusses, for example, climate and infectious disease, the problem is complicated and the interactions involved are both multidisciplinary and interdisciplinary. The human perspective must be included if we are to comprehend fully the global effects of climate change.

It would be useful to start not by belaboring points of contention but by indicating where there is agreement. Everyone agrees that global warming is occurring. Over the past few years, the highest average temperatures in history have been recorded. No one argues that. The argument, of course, is whether we are undergoing a natural cycle or anthropogenic-induced change. But, let us look at the fact that the ocean surface annual temperatures have increased. Warren Washington speaks eloquently of his research at the National Center for Atmospheric Research (NCAR) on the dramatic changes such temperature increases will invoke on sea surface levels (Washington, in press).

My focus is on one aspect of global warming: human health and the weather-related effects of climate change on infectious disease. The United States surgeon general's report in 1950 declared the war

on infectious disease over because of the discoveries of many powerful antibiotics. It was a premature declaration; infectious diseases are a moving target and remain very serious threats to the human race. Globally, acute respiratory infections, including pneumonia and influenza (avian influenza is a looming threat), are the number one killer. However, for children under the age of five, diarrheal disease remains a major killer, especially in developing countries.

Cholera, a diarrheal disease, has been with us for a very long time, even being mentioned in ancient Sanskrit writings. A medical textbook published in 1875 reported cholera to be a global pandemic, consistently appearing in India, Bangladesh, Latin America, and Africa. Today, cholera remains a serious problem. Until the nineteenth century, cholera was generally confined to the Indian subcontinent, but it then began to appear in Europe and the Americas as well. Since 1817, Western medical history describes seven global pandemics of cholera that have spread illness and death around the world. The second of these seven pandemics reached the United States in 1832, traveling from New York to Philadelphia in a couple of weeks, and then cases appeared along the Atlantic coast all the way to the Gulf of Mexico. In fact, Washington, D.C. and New York, until 1900, saw frequent epidemics of fevers, including typhoid, malaria, and cholera.

Cholera arose in epidemic form in London in 1849, at a time when the germ theory of disease was being debated. John Snow, in that year, carried out the first published epidemiological study, charting cases of cholera in London. He concluded that the cholera cases clustered around a well in central London when cholera was at its peak during the summer months. The epidemic abated in the September of 1849 but, as I will explain, the decline had to do with natural factors rather than the purported removal of the handle from the pump by John Snow.

In 1977, my coworkers and I reported that *Vibrio cholerae*, the causative agent of cholera, could be cultured from Chesapeake Bay

water samples. It was the first report of the isolation of the cholera vibrio from noncholera-endemic geographical areas; cholera had not been reported in Maryland since the 1900s. It was difficult for us to make our case, namely that the cholera vibrio was a native inhabitant of the Chesapeake Bay, since cholera had not occurred in the region. Nevertheless, the bacterium was there. Subsequently, we were able to apply molecular techniques and show that, indeed, the bacterium is naturally occurring in the aquatic environment, with annual peaks in the spring and fall. Furthermore, we were able to determine that the cholera vibrio is associated with plankton. We now know that river, estuary, and coastal waters are reservoirs of these bacteria globally, but our data showing an environmental source of the cholera bacteria implied a paradigm shift for the medical community. It has taken about 20 years for the paradigm change of cholera being transmitted only by person-to-person contact to the recognition that the cholera vibrio exists in the environment as a natural inhabitant. Furthermore, we discovered that the bacterium undergoes a dormant stage between epidemics and, with molecular techniques—that is, gene probes—we could prove its year-round presence in the environment.

The relationship with zooplankton turned out to be especially important. In the spring, when the water warms, phytoplankton become abundant; using sunlight for energy, the population of phytoplankton increases significantly. That population increase is followed by blooms of zooplankton, the miniature "cattle" of the sea, which graze on the phytoplankton. We were able to show a relationship of sea surface temperature increase with onset of cholera epidemics because of the fact that vibrios comprise the natural microbial flora of zooplankton, the populations of which increase spring and fall in annual cycles. The seasonal pattern of cholera follows the seasonal rise and fall in sea surface temperature and height.

In 1991-1992, a massive cholera epidemic occurred in Peru: approximately 200,000 cases and 5,000 deaths were the result of

the epidemic. This was unprecedented, since cholera had not been reported in South America for nearly 100 years. Furthermore, the epidemic occurred at the time of a powerful El Niño. Climatologists predicted another Niño in 1997-1998. We hypothesized that there was a linkage of the 1991-1992 cholera epidemic with El Niño and predicted additional cholera outbreaks would occur in 1997-1998. With colleagues from Peru, Chile, Ecuador, Brazil, and Mexico, we conducted a training session on molecular techniques for direct detection of the cholera vibrio in water and plankton. As the sea surface temperatures in these Latin American countries increased in 1997 because of El Niño, the team was able to detect the presence of the cholera bacteria associated with plankton, with numbers of the bacteria increasing from spring to summer (September 1977 to March 1998) and cases of cholera occurring in late November through the summer of 1998.

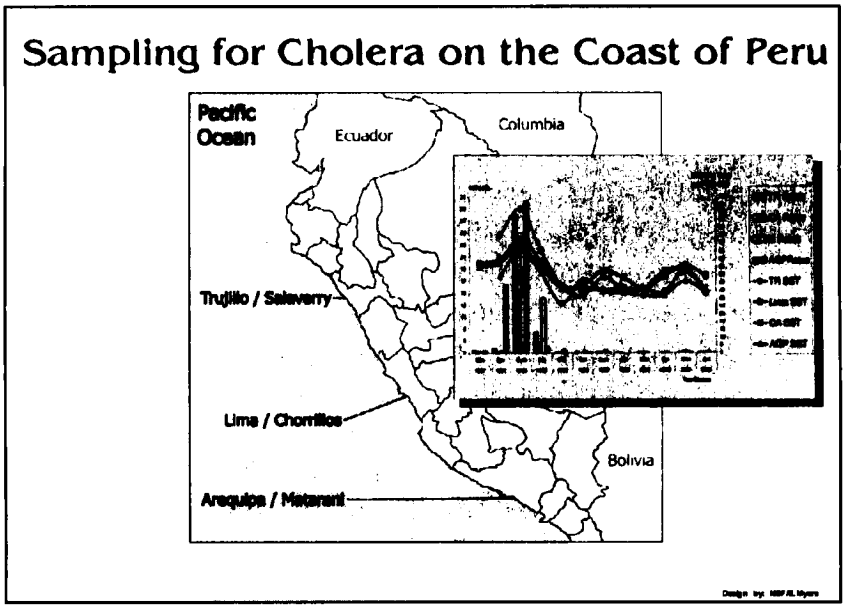


**Fig. 1** Cholera Data, Coast of Peru, Related to El Niño, 1997–1998.

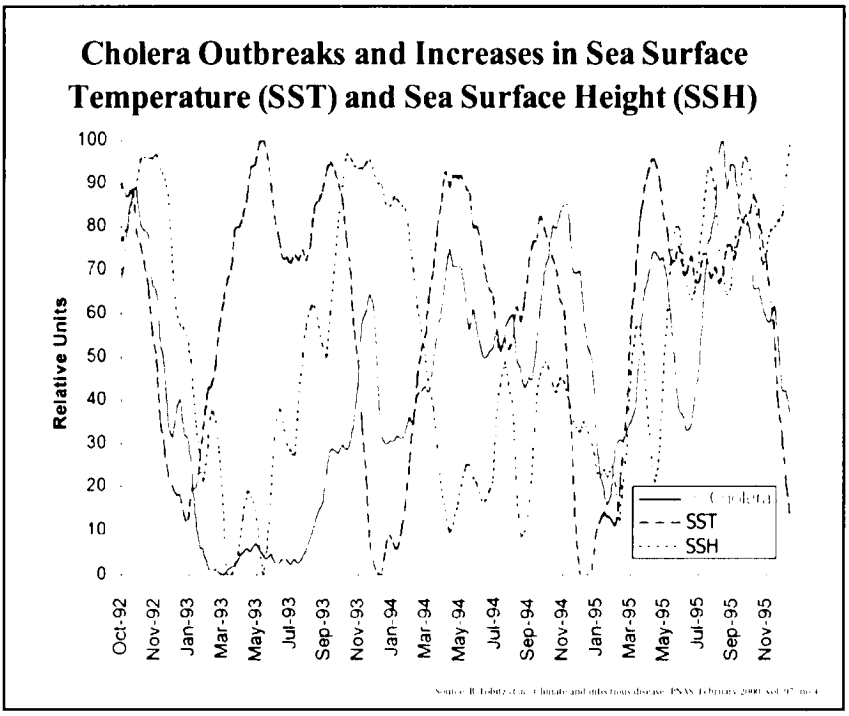


**Fig. 2** Cholera Outbreaks and Increases in Sea Surface Temperature (SST) and Sea Surface Height (SSH).

We were able to conclude that El Niño is another important climate factor related to cholera, notably in cholera-endemic countries. The cases of cholera in Peru in 1997-1998 were directly correlated with sea surface temperature (fig. 1). The relationship of the disease with this climate factor was statistically significant.

In Bangladesh and other countries where severe cholera epidemics occur, such as Peru, Indonesia, and India, the influence of monsoons or severe weather is important. Matlab, near Dhaka, Bangladesh, comprises a "hotspot" of cholera. The villages are constructed around bodies of water, and specific locations of epidemics have been determined. Our research on cholera has been conducted in Matlab, Bangladesh, since 1975.

The influence of the Himalayas on the weather in Bangladesh is significant, because the monsoon rains wash nutrients into the

rivers and ponds. Typically, houses in Bangladesh are located at the edge of a pond, from which villagers draw their water for household use.

A definable relationship between sea surface temperature, sea surface height, and cholera epidemics was established and published in the proceedings of the National Academy of Science (Lobitz et al., 2000; see also fig. 2). Taken together, the complex factors of sea surface temperature, sea surface height, and zooplankton populations provide a predictive capacity for cholera epidemics in developing countries that derives from climate monitoring through satellite sensors.

Finally, with the assistance of sociology researchers working in Bangladesh, we were able to test the hypothesis we constructed: that if we could remove zooplankton from the water the villagers used to meet household needs, the incidence of cholera could be reduced. With a very simple filtration technique that we devised using sari cloth folded in 4 layers, we were able to reduce cholera by approximately 50 percent in villages where families had been instructed in the filtration method. The complex of plankton, people, and climate, together with a simple solution based on science (under the electron microscope, the folded sari cloth could be seen to provide a 20 micron filter—and the zooplankton range in size roughly 50 to 200 microns), provides the interrelationship that allows an understanding of a global infectious disease—an understanding that would not otherwise be possible.

Thus, climate change influences a complexity of biological systems of our planet earth and in our models, and the human factor cannot be omitted from climate models, if we care to constrain global infectious disease and protect human health.

THE QUESTION WE RAISED, BASED ON OUR FINDINGS, WAS WHETHER we could predict, using only environmental data, the location, time,

and intensity of a cholera epidemic. From satellite observations of sea surface temperature, sea surface height, and chlorophyll, we found that we could, indeed, predict these aspects of a cholera epidemic.

Clearly, global warming can have a profound effect on the pattern of infectious diseases, especially a disease like cholera that is so strongly intertwined with the environment. It can have a profound influence on geographic range and intensity of the disease globally. Similarly, both cholera-related waterborne diseases and vector borne diseases, such as malaria and dengue, will pose enhanced threats in some parts of the world.

In summary, the interaction of humans, cholera bacteria, the zooplankton host of the bacterium (the copepod), and the environment in the case of cholera can be employed to make reasonable predictions about this climate-driven disease. The issues are truly international and represent those that comprise a global scientific enterprise and encompass many other infectious diseases. Our research clearly had to be collaborative and international for a fuller, more nuanced understanding of cholera and, I predict, so it will be for human health and survival on this planet. That is the ultimate exercise for understanding the consequences of global warming and, certainly, for policies addressing societal needs related to global climate change.

## NOTES

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