Journey after Darkness
A Study of Goma, Zaire in 1994:
Cholera and Clean Water

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"We can all turn into killers, Valcourt had often maintained, even the most peaceful and generous of us. All it takes is a certain circumstance, something that clicks, a failing, a patient conditioning, rage, disappointment. The prehistoric predator and the primitive warrior are still alive beneath the successive varnishings that civilization has applied to mankind. All the Good and Evil of humanity is in our genes. Either one can emerge at any moment, as abruptly as a tornado can appear and destroy everything where minutes before only soft, warm breezes blew."

-from *A Sunday at the Pool in Kigali* by Gil Courtemanche, 2003

"If there is one thing sure in this world, it is certainly this: that it will not happen to us a second time."

-from *Survival in Auschwitz* by Primo Levi, 1958

"It happened, therefore it can happen again: this is the core of what we have to say. It can happen, and it can happen everywhere."

-from *The Drowned and the Saved* by Primo Levi, 1986

Executive Summary

Ten years ago, on April 6, 1994, the majority Hutu of Rwanda, prompted by the shooting down of President Habyarimana's plane, committed the most efficient massacre of men, women, and children since the Holocaust. 800,000 Tutsi and moderate Hutu people were killed in 100 days. Years before, there were warnings of events to come. Hutu and Tutsi factions fought a civil war and committed atrocities against each other. Hundreds of thousands of people were displaced. After the genocide, rebel Tutsi forces swept down from bases in Uganda, quickly occupying most of Rwanda by early July. As a result, approximately 800,000 refugees, most of whom were Hutu *genocidaires*, fled into Goma, Zaire between July 14-18, 1994. The relationships and tensions between the Hutu and Tutsi, that culminated in the genocide and humanitarian crisis in Goma, go back over centuries. Notably, these ethnic differences were promoted and perpetuated by the Belgian, German, and British colonialists.

In the midst of chaos, exhaustion, and dehydration the first case of cholera in Goma, suspected clinically July 18, was proven by culture on July 20 as *Vibrio cholerae* 01 El tor. Within two weeks of onset the effect of the cholera epidemic was devastating to the refugees in Goma town, and in three camps to the north, *Mugunga, Kibumba,* and *Katale*. Between July 21-August 12 there were 62,000 cases of diarrhea seen in health centers, peaking at 6000 cases on July 26. From a camp survey, there were 35,500 estimated cases of cholera, with another 22,400-44,800 cases not able to get to health centers. The case fatality rates anecdotally were reported between 25-50%. A estimated total of 58,000-80,000 cases of cholera occurred during the first month after the refugee influx with an attack rate estimated between 7.3%-16%. The ratio of symptomatic to asymptomatic cholera cases is 1:10. It is likely, therefore, that the entire refugee camp was infected by *V. cholerae* during the epidemic.

Many epidemiological factors influenced the cholera epidemic. Chief among these, outside of the horrible conditions of the refugees and the lack of any adequate sanitation, was the ecological niche of *Vibrio cholerae,* and the water of Lake Kivu, the source of the epidemic. The role of endemic cholera is explored. From previous studies non human reservoirs, eg zooplankton, can serve as potential inter-epidemic dormant sources of non-toxigenic vibrios. The human is the only major host; there are no animal reservoirs. Conversion of non-toxigenic to toxigenic strains is facilitated in the small bowel by several genetically determined markers, one of which binds a crucial bacteriophage that contains and transfers the gene for toxigenicity. Lake Kivu, the only initial source of drinking water for the refugees, was most important. Its alkaline pH and lack of a thermocline, a warm surface cleansing layer of water, was a perfect growth media for toxigenic *V. cholerae*. Therefore, the provision of clean water became the most emergent problem during the epidemic, but even bucket chlorination brigades could not halt the rapidity of the epidemic that peaked within one week of onset, and had virtually run its course in one month. The highest crude mortality rate(CMR) recorded in *Katale* camp, 41.3 per 10,000 per day, exceeded the CMR of the famine of Ethiopia in 1985 and Somalia in 1992.

There were major problems with both contingency planning and preparedness before the influx, and there were problems during the humanitarian response. Despite heroic efforts by as many as 100 NGOs on the ground within 1-2 months of the arrival of

the refugees, not enough clean water could be supplied fast enough to reach the UNHCR guidelines of 15-20 liters of water per person per day. Within one month only 5L/person/day had been provided. Provision of latrine sanitation was impossible due to the hard volcanic rock terrain. There were major deficiencies in the provision of oral rehydration solution(ORS); many relief workers were very inexperienced in administering ORS. Designated cholera treatment tents were inaccessible to many cholera victims.

Media attention was overindulgent of the conditions of camps and the cholera epidemic. Less attention was paid to the important preventative sector of sanitation. Security remained a major problem. Many of the refugees were Hutu militia who had committed the atrocities, and who continued to be a very threatening. The US and French military there did not disarm the militia; their only mandate was to provide humanitarian aid. The US Army offered a stockpile of a two dose oral cholera vaccine, which would have been logistically impractical to administer. Furthermore, the anticipated protective immunity from the vaccine would have developed well after the cholera epidemic had run its course. Cholera vaccine, therefore, cannot be considered a primary intervention in an acute humanitarian crisis.

The tragic events of Goma have roots in ethnic disparities and in political and social turmoil. The failings of the West to recognize the potential for disaster and not intervene, and, in fact, withdraw forces that might have made a difference during the genocide, is a failure of foreign policy. But to understand the roots of this darkness and the journey that followed –and continues today in many other areas of Africa – is to understand that social and ethnic inequities along with failed civil and political systems cause human rights violations that structure violence and exposure to disease, such as cholera in Goma in 1994. A policy that works to correct human rights violations may be just as important as policies that prepare nations and humanitarian relief workers to care for the public health consequences of the violations of these rights.

Prologue

To understand the origins of the Rwandan genocide that began April 6, 1994, the displacement of one million people that followed, and the humanitarian emergency in Goma, Zaire in July, it is important to see these events in the context of the history and geography of this vast heartland of the Great Lakes of Africa.[1]

A circle of deep freshwater lakes, surrounding a table of land 1000-2000 meters high, is the main geographic feature of the African Rift Valley. Looking for the source of the Nile, Stanley explored Lake Victoria in the east, and later discovered Dr. Livingstone at Ujiji, along Lake Tanganyika to the west. In 1876 he discovered that Lake Tanganyika flowed westward through cataracts, later named Stanley Falls, to the great Congo river, and northward into Lake Kivu through the Rusizi River. The Virunga Volcanoes, north of Lake Kivu, interrupt ancient rivers to Lakes Edward and Albert.[2]

Demographically this area is one of the most densely populated in all of Africa. In 1900 there were about 60 people per square kilometer, and today there are nearly 3000.[2] Human settlement over the last 200 years has far outstripped the land's capacity to support the population. Nevertheless, the Belgium, German, and British colonialists, traversing the dry savannas of East Africa and the dense Congo jungle in the mid 1800s, were attracted by green hills and clear lake water. This peaceful land was settled by farmers and by pastoralists herding cattle. The region also seemed protected from diseases that were more prevalent elsewhere in Africa. Later in 1945, Belgians found refuge along Lake Kivu, and in the 1980s Rwanda was a beautiful mountainous retreat where the people appeared friendly and hardworking.[2]

The ancestors of the Hutus of Rwanda were the Bantu peoples that emigrated to the Great Lakes region from the Cameroon between the first millenium B.C. and the sixteenth century. Bantu actually refers to five dialects spoken east to west in present day Uganda, Rwanda, Burundi, and eastern Congo. The Bantu cultural, social, and economic mantle did not assume significance until Europeans arrived in the second half of the 19th century. They attributed certain physical characteristics to the Hutu(Bantu) that were "typical ....of Negroes: flat nose, thick lips, and a low forehead".[3] They planted taro, and banana, imported originally from the Malay peninsula, and sweet potato, cassava, maize, and beans from the Atlantic coast. In contrast, the pastoralist Tutsi ancestors were ancient Hamitic peoples who emigrated from Abbyssinia, bringing their distinctive long-horned cattle. They were taller, and had "straighter noses, high foreheads, and thin lips".[3]

Anthropologists have debated the ethnic differentiation of the Hutus and Tutsis. On one level, the Tutsi cattle were important in social and political contracts. The cattle were a measure of wealth, and land ownership. The Tutsi position in early Rwandan society promoted a form of "pastoral feudalism" that supported inequality. Consequently, powerful Tutsi chiefs at one time, representing a minority of the population, controlled 80% of Rwanda-Burundi. Over several centuries, there was intermingling of ethnic groups through marriage, and the distinction between racial characteristics became less apparent. At the time of the genocide, for example, there were many Hutus killed because they resembled Tutsis.

The arrival of Europeans in 1860 added a new dimension to these people who were peacefully coexisting. The destiny of the Germans in Rwanda and Burundi, the Belgians in the Congo, and the British in Uganda could not be described as enlightened.

There was frank repression, and racism. Stanislas Lefranc, a Belgian prosecutor who had come to the Congo to work as a magistrate, promoted the use of the *chicotte,* a whip made from the dried hide of a hippopotamus, to beat men and children into submission.[4] Stanley spoke of the image of the "aborigines he met" in the same context that an "agriculturist sees his strong-limbed child", a "future recruit to the ranks of soldier-laborer." [4]

The Catholic Church of Rwanda promulgated the distinction between Tutsi and Hutu. The vicar apostolic, Leon Classe, living in Kabgayi, Rwanda until his death in 1945, believed in a medieval-style Rwanda, where the Tutsi aristocracy should rule over the Hutu peasantry.[2] The Church favored Tutsi youth for their desire and ability to learn, and for their perceived desire to imitate the Europeans.[2] Indeed, they may have favored the Tutsi because they looked more 'European' than the Hutus. There are many examples in the Great Lakes history of the colonial powers exploiting their prejudices of local ethnic groups, promoting one over the other, to advance their 'right' to the land and its vast resources.

Furthermore, the Europeans opened this pristine area to disease. Microbes that flourished along the coast of Zanzibar were transported inland by Arab traders, well-intentioned missionaries, and white soldiers. In 1891, a rinderpest epidemic, originating in Ethiopia, decimated cattle. The surviving cattle, controlled by a powerful few, added to the social turmoil. In 1892, smallpox, once restricted to the southern shore of Lake Victoria and Ujiji, along Lake Tanganyika, spread to the mountains of Rwanda and western Congo. 'Chiggers', caused by *Sarcopsylla penetrans,* initially a pathogen found in South America, had made its way across the Congo, penetrating feet and causing

infected wounds for which there was no remedy. Finally, at the turn of the century, an epidemic of sleeping sickness killed more than 200,000 people in Uganda, and overcame half the population of the Rusizi plain of western Burundi.[2]

Geologic calamities were no less important to the history of the region. The drop in Lake Tanganyika's waters by 12 meters between 1879-1896 left swampy lakeside conditions causing more disease and misfortune. Nyiragongo volcano, located just north of Lake Kivu, erupted in 1894 leaving great devastation. These disasters enhanced tensions between populations, so that by the early 1900s existing economic and political frameworks were slowly beginning to unravel.

Genocide

The humanitarian crisis did not begin with the shooting down of the Rwandan presidential plane April 6, 1994, but was preceded by years of ethnic conflict in Rwanda and in neighboring countries. A stronger Hutu elite emerged from the 1950s with a political mandate to rule. In 1988, a contingent of Rwandan soldiers, mostly Tutsi members of the Rwandan Patriotic Front(RPF) encamped along the Ugandan border, conducted operations into Rwanda. By mid 1992, ethnic violence erupted causing the internal displacement of about 300,000 people(IDPs). There were massacres of Tutsi clans, perpetrated by Hutu militia, called *interahamwe*('those who act together'). Reprisals were taken in the north against the Hutu prompting a million people to move toward Kigali. The political situation deterriorated as the world watched.

On April 6, 1994 the Rwandan presidential plane was shot down at the Kigali airport by extremist Hutu, who felt that President Habyarimana had made too many political concessions to the Tutsi. The massacre of about 800,000 people, mostly Tutsi

and moderate Hutus, that followed in the next 100 days was premeditated and efficient. It was carried out with the blessing of Hutu officials, and with the knowledge and consent of religious leaders, academics, doctors, teachers, and journalists. The radio station recited the names of local Tutsi officials and evacuees so the *interahamwe* could go after them. Hutu roadblocks were everywhere. The *interahamwe* were "bloodthirsty and drunk", noted a Canadian physician posted in Kigali.[5] The genocide, however, did not happen in an international political vacuum. The critical role of the United States government and of the United Nations has been described and analysed.[6,7] The omission of an effective foreign policy in this instance and the lack of support for the United Nations Assistance Mission in Rwanda(UNAMIR) under the command of General Dallaire may be among of the gravest lessons of 1994.

On April 8 Major Kagame quickly moved his RPF troops out of the Ugandan border areas towards the south. There was surprisingly little resistance. By April 11 Kigali was taken and the war was over July 18. In this interim period approximately 1.5 million Rwandan people fled, many having committed atrocities, some into neighboring Tanzania, but from July 14-18 most fled across the Rwandan border into Goma, Zaire. Soon, one human catastrophe was replaced by another. The first case of cholera was recorded July 20.

Cholera

Disease

Cholera is one of a few infectious diseases that can cause death within 24 hours of onset. The incubation period varies from 18 hours to 5 days. The painless, voluminous, rice watery diarrheal stools have a fishy odor. The vomitus is clear and watery. Within

hours of onset, victims can develop sunken eyes, tenting skin, restlessness, extreme thirst, a look of apathy, and loss of consciousness, all signs of profound dehydration. They can purge 500-1000cc per hour. Without rapid rehydration, death follows. Without treatment the case-fatality rate in severe outbreaks can be as high as 50%.[8] In well-managed outbreaks the case-fatality rate can be 1-3%.

Prompt effective treatment can prevent death. In an epidemic, experience managing the patients is important. Clinical triage of the sickest requiring IV therapy, and those requiring aggressive oral rehydration solution(ORS) is essential. Clinical assessment of the degree of dehydration determines the choice of therapy. Manpower, supplies of ORS and clean water, and equipment are crucial. Cholera cots, with a hole through which the diarrhea volume can be measured, are invaluable in managing severely purging patients. Severe volume loss demands aggressive fluid replacement.

As 1.2 million refugees, many exhausted and dehydrated, entered Goma after July 14, 1994, the magnitude of the humanitarian crisis became unimaginable. Supplies were scarce to nonexistant. There was no reasonable level of sanitation; no defecation fields were designated. Make shift blanket tents were set up everywhere, but not everyone had shelter. Fuel sources for cooking were quickly exhausted. Many of the children were unaccompanied, wandering hopelessly, the saddest legacy of displacement and civil strife. From a distance Lake Kivu appeared a quiet shade of blue-green, and enticing. It was the only immediate source of fresh water. Yet some of the refugees were so exhausted and dehydrated that they lacked the energy to even get to the waters edge. Because of previous volcanic lava flows the earth was solid rock; latrines could not be dug. Soon, there was sewage mixing with lake water. Many of the refugees were the

interahamve who had just slaughtered 800,000 people. Security was soon to become more of a problem. Chaos, crowding, and cholera became realities.

Epidemiology of Vibrio cholerae

Vibrio cholerae, serogroup 01, biotype El tor, serotype Ogawa is the agent responsible for the seventh cholera pandemic, which began in Indonesia in 1961. In 1994 there were 384,403 cases reported to the World Health Organization(WHO) from 94 countries.[9] There were 58,057 cases reported from Zaire, but more were suspected. The global case fatality rate(CFR) was 2.8%.[9] Other large outbreaks were reported from Guinea-Bissau, Angola, and Somalia. The overall CFR in Africa was 5%.

The role of the environment and potential for genetic reassortment became reality in 1992 when a new strain, *V. Cholerae* serogroup 0139, was first isolated in large outbreaks from India and Bangladesh. Serogroup 0139 replaced 01 from 1992-1993 as a result of genetic reassortment, natural selection involving unidentified ecologic factors, and host immunity.[10] This strain heralded the 8th pandemic. However, since 1996, *V. cholerae* 01 has again caused most of the cholera cases in the world, re-establishing its ecologic dominance. However, the strain differentiation, presumably the result of environment pressures and genetic diversity promoting survival, though not permanent in this instance, could have an impact on the development of future effective cholera vaccines.

There are characteristics of cholera outbreaks, mostly studied in Bangladesh, that *may* have application in Africa. Changes in climate, classically related to warm temperatures and pre and post heavy rains, can directly influence the appearance of

cholera. For example, in 1992 El Nino Southern Oscillation(ENSO), a progressive west to eastward warming of sea surface temperature(SST), was noted to increase the SST in the Bay of Bengal leading months later to cholera outbreaks.[11] The sudden abundance of water without adequate sanitation and disinfection leads to cholera outbreaks in endemic areas. ENSO in Africa is associated with drought, and famine. In 1994 there was no apparent major ENSO, and therefore this weather pattern did not apply. Another example was a dominant ENSO in 1997-1998 that did lead to major drought in Indonesia, Papua-New Guinea, and the Philippines. A scarcity of clean water led to outbreaks of cholera in these endemic areas. Therefore, a better understanding by public health officials of the effect of climate changes would facilitate early warning and planning for cholera epidemics.[11]

There are factors that influence the pathogenicity of *V.cholerae*. Since the rapid onset of voluminous diarrhea is mediated by a toxin attaching to the villous membrane of the small bowel, inoculum size is important. Experimental studies in healthy volunteers indicate that 10^6 organisms are required to cause disease. However, fewer organisms cause illness in malnourished, immunosuppressed children and adults whose normal gastric acid barrier may be severely compromised.[12] Children 2-4 years old, and adult men are also at greatest risk for disease because these groups are the first to be exposed to contaminated water. Cholera stools are highly infectious, containing 10^8 organisms per gram. In an epidemic the ratio of symptomatic to asymptomatic cases may be 1:3 to 1:10 and may be as high as 1:100.[13] There is an interesting association between blood group O and higher risk for cholera, but the pathogenic mechanism is unknown.[14]

Ecology

V. cholerae thrives in brackish, shallow, and alkaline water, and in the human gastrointestinal tract. There are no animal reservoirs. Flourescent-monoclonal antibody colonization studies have demonstrated attachment of non-toxigenic non-culturable vibrio to natural reservoirs like plankton, copepods, and egg masses of chironomids(non-biting midges-a gnatlike fly laying eggs on surface water).[15] The chironomids provide a carbon source to support development of *V. cholerae*.[16] Given changes in climate, water temperature, salinity, availability of nutrients, and genetic adaptation, the organism can pass from a non-toxigenic, non-pathogenic free-living form to the more virulent, toxigenic stage, then posing a risk to its human host.[17] While it is likely that the non-toxigenic vibrio survive in these intermediate reservoirs, it is in the human host that the organism realizes its full pathogenic potential. These mechanisms of vibrio survival play a role in outbreaks of cholera in India and Bangladesh, but there is no evidence that these reservoirs contributed to the outbreak in Goma. Nevertheless, further research to better understand the relationship between the water environment and endemic cholera in the Great Lakes region is indicated.

Virulence Factors

The entire genomic sequence of V. cholerae biotype El tor has been described.[18] Cholera pathogenesis relies on the synergistic effects of cholera toxin(CT), colonization of the microvillous small bowel membrane mediated by pilus colonization factor(TCP), and a regulatory protein, Tox R, which co-regulates both CT and TCP, both required to produce infection.[19] Each of these are encoded by genes. CT has a and b subunits, encoded by the genetic element called CTX. CTX corresponds to the genome of CTX*,

a lysogenic bacteriophage.[20] The CTX can exist as a replicating plasmid or a prophage incorporated into the chromosome.[20] (See Appendix-Bacteriophage[21]) The phage binds to pilus colonization factor(TCP), and thus CTX plasmid or prophage invades the organism, converting a non-toxigenic strain to toxigenic strain.[22] This event takes place in the human intestinal tract.[23] Environmental isolates do not contain CT genes unless found in proximity to infected human hosts.

Factors in the environment that allow strains to withstand nutrient deprivation, and to survive by colonization of non-human hosts, like phytoplankton, are not well understood.[24] Genetic and environmental factors have enabled the *V cholerae* 01 El tor to predominate over the Classic and more contemporary short-lived 0139 strains in S. Asia.[24] It is possible that genes that promote survival in the human gastrointestinal tract are more important than factors promoting survival in non-human reservoirs by mediating bacterial resistance to stomach acid, and allowing prolonged colonization.[24] These same factors also promote increased shedding of vibrios in infected diarrheal stool. Therefore, the human gastrointestinal tract provides the most efficient milieu for vibrio survival, whereas the aquatic environment provides a resting place for non-pathogenic strains between epidemics.

Lake Kivu

This deep natural lake is situated immediately south of Goma, a city of approximately 100,000 population. Its surface area is 2370 km^2, its volume is 650km^3, and its mean depth is 240 meters.[25] Its outflow is minimal(2.45 km^2 per year), hence its estimated replacement time is 265 years.[25] Water flowing in from Lake Tanganyika,

direct rainfall, or watershed runoff promote little recycling because of the prolonged residence time of the lake water.

The hydrologic cycle is the relationship between surface and groundwater and the constant movement of water in the environment.[26] When the precipitation rate exceeds the normal infiltration rate of water into the ground, there is overflow into rivers and lakes. Water quality can be influenced by acute impacts such as a sudden sewage spill or volcanic eruption, and by chronic impacts such as increased human activity within a watershed, accelerating nutrient activity. The term, eutrophication, describes the process that increases the supply of organic matter to an ecosystem. External sources of organic matter are heterogeneous chemical compounds, from watershed runoff, and endogenous sources generated by phytoplankton blooms. Nutrients in the form of organic matter increase microbial activity. Cleansing of this system is dependent on temperature changes in the layers of deep water, the existence of a warm middle layer called the thermocline, and the ability of the body of water to be replaced by fresh water.[27]

Lake Kivu has no real change in temperature from surface to deep water. There is no thermocline. The water is permanently anaerobic below 60 meters; its deep waters are saturated with methane. There is virtually no natural replacement. There is some nutrient exchange between lower and upper layers of water. Peak zooplankton bloom is in July and August.[25] In sum, this body of water has a slow replacement time, and a ph=8 from the still active Nyiragongo volcanic gases. There is no cleansing effect of a thermocline. It is a perfect environment for *V. cholerae* growth.

Journey after Darkness-Goma, 1994

The darkness of Rwandan genocide occurred for 100 days in April. Soon after Major Kagame's RPF victory in July hundreds of thousands of displaced people, mostly fleeing Hutus massed in the northwest Rwanda, crossed the Zaire border into Goma between July 14-18.

Lake Kivu was the only source of water for drinking, washing clothes, and bathing. The first case of cholera was suspected by Medecins Sans Frontieres(MSF) on July 18, proven by culture July 20 as *Vibrio cholerae* serogroup 01, biotype El Tor, serotype Ogawa, resistant to tetracycline, but sensitive to furazolidine and ciprofloxacin. After the onset of cholera a surveillance system was organized to monitor all cases and deaths due to diarrhea. Initial reports from health centers did not differentiate watery diarrhea from bloody diarrhea, or from dehydration. Laboratory facilities were provided by Bioforce(French), by an Israeli field hospital that was set up by July 26[28], and by reference labs in France and the Netherlands. The differentiation of watery diarrhea from bloody diarrhea began on July 31. The peak number of cases of diarrheal disease was 6000 on July 26, and between July 21 and August 12 there were more than 62000 cases of diarrhea reported to health facilities.[29] (See Appendix-graph)

Over the first few weeks an estimate of the number of cases of cholera was difficult to ascertain. Because of the dire situation in Goma, several refugee camps to the north were set up: *Mugunga*, 5 km. from Lake Kivu; *Kibumba* about 27 km. away; and the furthest north, *Katale*. (See Appendix -map) Those who survived the early days of the crisis were ushered through Goma to these awful camps. In early August a more accurate

surveillance system was established to differentiate cases of cholera(watery diarrhea) from dysentery(bloody diarrhea), and cluster surveys of the camps were conducted.

From August 4-14 three surveys were done in *Mugunga*, *Kibumba*, and *Katale* camps. In each camp 30 clusters were surveyed, 20 households per cluster, and 20 children per cluster under age 5 years.[29] Survey questions included number of household deaths since arriving in Zaire, probable cause of death(e.g. watery diarrhea, bloody diarrhea), adequacy of shelter, access to food rations, food reserves, access to health care. In children under 5 years, weight and height were measured; weight-for-height index for each child was compared to a standard reference population. [30]

An estimate of the total number of cholera cases was made so that an estimate of the attack rate for cholera could be calculated. From July 14-August 14, 62,000 cases of diarrhea were reported from health centers. The *Mugunga* survey found that 57% of diarrheal deaths were due to 'watery diarrhea'(cholera). Therefore, roughly 57% of the 62,000 cases of diarrhea(35,500) reported from health centers were due to cholera.[29] During this period there were 47,500 deaths. 41,800(88%) were attributed to diarrhea. 57% of the deaths due to diarrhea or 23,800 were due to cholera. It was more difficult to estimate the cholera case fatality rate in the population not able to get to the health centers. Of those who died from cholera, 47%(11,200) never sought health care. From anecdotal information, the case fatality rate from cholera among those who never sought medical care was 25-50%. Therefore, another 22,400 to 44,800 cases of cholera may have occurred. An estimated *total* of between 58,000 to 80,000 cases of cholera occurred in the first month after the influx of refugees.[29] Given a final conservative estimate of the total refugee population in Goma and the three camps of 500,000-800,000, the attack rate

was between 7.3%(58,000/800,000) to 16%(80,000/500,000).[29] Given the fact that the ratio of symptomatic to asymptomatic cases can be 1:10, it is probable that from July 14-August 14 the entire refugee population was infected with *V. cholerae*.

Morbidity data was collected from health facilities in Goma and from the three camps. Cases of watery diarrhea, bloody diarrhea, measles, meningitis, acute respiratory infections, presumed malaria(unexplained fever) were recorded. Definitions of specific diseases was provided by UNHCR.

Mortality data was based on the estimated population of the camps and refugees in Goma, and the daily body counts from July 14-August 14. Because of the hard volcanic rock, graves could not be dug. Roadside bodies were picked up by trucks and transported to mass graves. Agencies that supplied the trucks supplied data on numbers of deaths. These numbers were exaggerated during the period from July 18-25 because payment to the truckers was made per body transported. Therefore, body counts were adjusted downward during this period by 40%. After July 26, counts were more accurate. Population figures from Goma were most difficult as no census or registration information was available. Total refugee population estimates of 500,000-800,000 were finally determined by food and water distribution figures and mapping exercises by MSF.[29]

Mortality rates are the most specific indicators of the health status of a population.[30] In a humanitarian crisis of this magnitude a crude mortality rate(CMR) of 1/10,000 people/day is serious, and a CMR of 2/10,000/day is an emergency out of control.[31] 48,347 bodies were collected by trucks from July 14 and August 14. The average CMR during this period was between 19.5 and 31.2 per 10,000 per day, based on

population estimates of 800,000 and 500,000 respectively.[29] During the initial two weeks, the estimated CMR was between 28.1 to 44.9 per 10,000 per day.[29] (See Appendix-CMR) The CMR of *Katale* camp, 41.3 per 10000 per day, exceeded the CMR in Ethiopia in 1985 and Somalia in 1992.[32] The CMR of unaccompanied children in some centers was 20-120/10,000/day; for unaccompanied infants it was as high as 800 per 10,000/day.[29] By the end of July bloody diarrhea due to *Shigella dysenteriae* surpassed watery diarrhea as the major cause of death. By the beginning of August the cholera epidemic was waning, but in just one month the toll had been devastating.

Factors contributing to the CMR, other than diarrhea, were noted by relief workers at the time. The Medecins Du Monde rehydration camp in *Mugunga* camp estimated that ~40% of the deaths on July 24, about 10 days after the influx into the Goma area, were due to exhaustion and dehydration.[33] The MSF-Belgium in *Katale* camp estimated this fraction to be about 30%.[33] Entire families, found dead under their makeshift tents in *Mugunga* camp, were unable to get to Lake Kivu 5 km away. In a nutritional survey of 567 children older than 6 months in *Katale* camp(pop 80,000), the global acute nutrition rate(Wt/Ht <-2Z score or edema) was 23.1%, and the severe acute malnutrition rate(<-3Z) was 6.5%.[34] (See Appendix-Malnutrition) This degree of malnutrition since the onset of the crisis further supported exhaustion as a cause of death.[35]

It is interesting to postulate that Lake Kivu harbored dormant non-toxigenic vibrios capable of genetic adaptation to toxigenic vibrios in the human gastrointestinal tract. This endemic process seems more dependent on the confluence of many factors

including climate, existence of vibrios, bacteriophage, and human hosts. However in Goma in 1994, it is more likely that virulent toxigenic *V. cholerae* were transmitted from the stool of an infected person into the surface water of Lake Kivu which then served as a huge infected reservoir for bathing, cleaning, and drinking. The enormity of the acute emergency suggests that events unfolded so quickly and uncontrollably that any efforts to reverse the course, with even the best planning and execution, were destined for failure. Nevertheless, the planning and execution of humanitarian aid in this tragedy is an important part of the analysis. The magnitude of the surveillance data reflect the fact that something should have been done to reverse this course and to prevent death.

Clean Water

Introduction

Studies have shown that quantity of water is more important than quality of water in the transmission of disease.[36] The greater quantity of water allows for hand washing, and appropriate personal hygiene that prevents the transmission of disease. In cholera prevention, however, clean water is more important than quantity. A person's total water need per day is about 30 liters, above which there is significant reduction in diarrheal disease. UNHCR recommends a minimum standard of 15-20 L per person per day be provided.

In 1972, White first demonstrated in his classic, *Drawers of Water*, the connection between coliform counts, disease, and water source. He showed that surface water, in contrast to well water, had the highest coliform counts, often exceeding 500 counts per 100ml. His studies divided water related diseases into four categories: water-washed(e.g.trachoma), water-based(e.g. shistosomiasis), water-related insect

vectors(e.g.malaria), and waterborne(cholera). The classic waterborne diseases have a "low infecting dose of organisms, and may produce dramatic epidemics following relatively light pollution of a large common source...their prevention requires a completely pure supply".[37] Subsequent studies have confirmed the relationship between diarrheal incidence and contaminated water, lack of latrines, and indiscriminate disposal of children's feces into the compound.[38] Finally, Esrey in 1996 found that sanitation improvements conferred a greater impact on diarrheal morbidity than water improvements.[39]

Water becomes contaminated at the source or in the domestic environment. If the source is clean to begin with, then contamination can occur because the water collectors hands, in the process of carrying the open buckets, have infected the water. Narrow necked buckets with handles to facilitate transport reduce the incidence of diarrhea in <5 year olds.[40] These types of buckets prevent contamination at the source and in the domestic environment by limiting hand contact with the water. Similarly, storage of clean water in the domestic environment in a narrow necked pitcher reduces coliform counts by preventing domestic contamination only if the source of water is clean(fewer than 100 coliforms per 100ml.).[41] Adding 2.5mg/L chlorine to contaminated buckets suppresses coliforms, and there is an adequate chlorine residual level for four hours.[40]

Goma

In the initial stages of the cholera epidemic in Goma, the goal was to provide 5 liters per person per day. The desperately needed 4-5 million liters of water a day were not immediately available. The roads were choked by people moving north. Many areas off the road were impassable because of previous lava flows. Refugees entering Goma

were encouraged by UNHCR to keep moving towards *Kibumba* and *Katale* camps. *Katale*, where there was a spring fed water supply, was too far; hundreds of refugees died of dehydration en route. However, many were able to reach *Kibumba* camp, 27 km. from Lake Kivu, but there was no water. On July 29, just after the cholera epidemic had peaked, relief operations were able to supply 1 liter per person per day.[29] Bucket chlorination of water was the only means available to prevent further cases of cholera. By the time this became a logistic reality, the epidemic was slowing and it was too late. Earlier, the only option was to treat the cases of cholera with oral rehydration solution(ORS), and with intravenous feedings.

Treatment – ORS

The volume of fluid produced by the small intestine, about 6500 cc., is reduced to 1500 cc. at the end of ileum by normal small bowel absorption, further reduced to 250cc in the colon. In cholera, normal absorption is blocked by binding of cholera toxin to the enterocytes, initiating a voluminous secretory diarrhea.(See Appendix-Life Cycle) Standard ORS, containing 90mEq/L sodium, 80mEq/L chloride, 30mEq/L bicarbonate, 20mEq potassium, and 2000mg/dl glucose, takes advantage of an intact Na-coupled glucose osmotic reabsorptive mechanism for salt and water. Because of the possible hypernatremic consequences of treatment, a hypo-osmolar ORS has been formulated for use. It is associated with less vomiting, and has been as effective as standard ORS used since 1975 in the treatment of cholera.[42] In 2001, WHO recommended a reduced osmolality solution for global use.[43] On an interesting historical note, in 1943 Harrison developed an oral rehydration solution containing a hypo-osmolar electrolyte-glucose

solution that he successfully used treating numerous cases of diarrhea at Baltimore City Hospitals(now the Bayview Medical Center).[44]

There is current interest in adding Zinc to ORS. Zinc deficiency, a consequence of malnutrition and a dietary lack of animal foods, impairs the function of the gastrointestinal tract, increasing the secretory response to entertoxins. Zinc supplementation randomized to low income Indian children 6-30months old substantially reduced the incidence of severe and prolonged diarrhea.[45] In another randomized control trial, Zinc-standard ORS was given to Indian children with diarrhea not sick enough to be hospitalized. The treatment decreased the total and watery stool frequency compared to controls given just ORS.[46]

The Response

The influx of 800,000 refugees into Goma and the three main camps at *Mugunga*, *Kibumba*, and *Katale* led to a scale of morbidity and mortality that quickly overwhelmed health professionals. There were 3 non-governmental organizations(NGO) in Goma at the start of the influx; MSF had a drug distribution project in the Kivu region in 1992.[47] In the next few weeks 30 agencies and NGOs established cholera treatment centers. Bioforce(France) and a contingent of US military confirmed the *V. cholera* 01 biotype El tor strain; WHO and Unicef provided treatment protocols for ORS and IV therapy. There were over 100 NGOs operating in the Goma area by September.

During the first week of the epidemic a high standard of medical care was not achieved. The epidemic spread rapidly, peaking only one week after the first case was detected. There were 6000 cases of diarrhea reported on a single day.[47] Relief agencies had little time to set up treatment centers. The case load at the rudimentary centers was

extremely high; from July 21 through August 9, 60848 cases were treated.[48] Many people died without reaching the centers because of lack of transport or because they did not know where the centers were located. Outreach programs to identify and treat those who could not access the centers was nonexistent.

The administration of ORS was handicapped for several reasons. At first, scarce water and ORS supplies were distributed to the treatment centers. Some treatment centers had no water storage facilities. Intravenous fluids(Ringers Lactate) were used frequently in situations where ORS only could have been used earlier. When the Ringers Lactate ran out, inappropriate Dextrose solutions were used. There were not enough French speaking relief workers, and morale was low. Security issues dictated that relief workers leave the treatment centers at night, but often when they returned the next day patients had been forcibly replaced by friends and relatives of Hutu militia. Finally, the experience of the relief workers in dealing with disasters of this magnitude and administering ORS to cholera victims was a major issue. Retrospective analyses of the different roles and levels of skill of the NGOs did highlight the need for planning and experience on the ground.[47,49,50] As an example, one of the NGOs delivered 10,000 cases of Gatorade. Another set up IV fluids in shelters and then left them unattended. An antibiotic, to which the organism was resistant, was distributed.

Prevention of an epidemic

The compelling question is whether cholera could have been prevented or the magnitude of the epidemic reduced. Against the possibility of prevention was the large population arriving in Goma over four days. The Lake Kivu region is endemic for cholera, and the lake water was the perfect growth media for *V. cholerae*. Lack of

sanitation and the utilization of the lake for drinking and personal hygiene by hundreds of thousands of people made a cholera outbreak inevitable. Even the hindsight of analysis mitigates against a realistic and effective early intervention. Events just moved to quickly.

The decision to establish bucket chlorination teams at points along the lakefront was not made by the agencies until July 23, three days after the first case was reported. There was a delay, however, before UNHCR, UNICEF, and the CDC actually began injecting chlorine into jerry cans at 12 sites along the shoreline. The effort was too late to halt the epidemic. If every bucket of water removed from Lake Kivu had been chlorinated and the epidemic kept from progressing so quickly- if it had spread over a month rather than a week- efforts by relief agencies to set up rehydration centers would have been more efficient, and the treatment of cholera with existing resources would have been more effective in averting the deaths experienced.

Other strategies to slow the spread of the cholera epidemic would have involved provision of sanitation. Defecation sites closer to, but segregated from, water distribution sites might have helped. Prepositioning water trucks, and stockpiles of medicine, food, and equipment for shelter in Goma before the influx would have assisted, but could never have been adequate for this large refugee population. The municipal water system was deficient; chlorine supplies were low. Contingency planning and preparedness was sorely lacking before July 14.

Contingency and Preparedness:

Before July a number of events that would have a major impact on Goma began to unfold. UNHCR and their Emergency Response Team(ERT), sent to Goma after the

genocide, was responsible for contingency planning there. The International Committee for the Red Cross(ICRC) gathered on the ground information in Rwanda. The ERT's estimate was that 50,000 refugees would be coming over the border.

However, the ICRC and subsequently Oxfam provided estimates that there were between 200,000-500,000 internally displaced people(IDP) moving toward Ruhengeri in the north, fleeing the RPF forces. A meeting held in Nairobi in June, to bring together the major parties for contingency planning including UNHCR and ICRC inexplicably did not share this crucial information. As a result, the UNHCR estimate of 50,000 remained the same, and shortly thereafter, UNHCR withdrew part of the ERT, sending them to Ngara, Tanzania, the site of another large refugee camp.

Nevertheless, there may have been insurmountable obstacles to better planning and preparedness. If the estimate had been more realistic, it is possible that there would not have been political support from the local Zaire authorities for a larger relief effort. Donor financial support may not have been forthcoming and stockpiling of food and contingency supplies would have been risky because of security issues.[48] Hutu were moving into a predominantly Tutsi area and fighting was anticipated. Compounding this was the reduction in ERT force in whose ranks there was little technical expertise in water and sanitation. At the same time, another strategic decision was made that had far reaching consequences on what eventually happened in Goma.

Operation Turquoise, a French military initiative to establish a Safe Zone for fleeing Tutsis in the southern sector of Rwanda, "contributed substantially to the disaster in Goma", according to a report by the Overseas Development Council.[48] Presuming that most refugees fleeing Kigali would proceed south, attention was effectively diverted from

the growing humanitarian crisis in the northwest. Indeed, most relief workers supported the plan of a safe zone in the south because they also thought this was where most IDPs would go. As a result, the RFP were left unopposed in the north. After the fall of Ruhengeri, where the 500,000 displaced Hutus were amassing, crossing the Zaire border into Goma was the only alternative. This movement coincided paradoxically with the completion of the UNHCR contingency plan for just 50,000 refugees.

Provision of Clean Water and Sanitation

Cholera ravaged the population arriving in Goma and in *Mugunga* camp(pop~150,000), 5 km. away. As the daily briefings from CDC epidemiologists came in, documenting the epidemic related attack and mortality rates(10-15% developing moderate to severe cholera, and 50% mortality reported in some centers), the provision of clean water became imperative. Lake Kivu water became quickly infected. Bucket chlorination was not instituted right away so those who had not died immediately of exhaustion and dehydration[31] were able to reach Kivu's edge. The municipal water supply, pumped from the lake, was inadequate.

Preparedness was limited by UNHCR's contingency planning for 50,000 refugees. Oxfam had water pumps and pipes for only this number of refugees. MSF-Belgium had hired 4 water tankers at the beginning of the influx; each tanker had a $20m^3$ capacity(1 m^3=1000 liters). *Kibumba* camp(pop~180,000), into which about 40% of the refugees crossed the border further north of Goma, had no water, and needed a minimum of $600m^3$ daily which is a little over 3 L/ person/ day. To supply this population immediately with water would have required a capacity of 30 tanker trucks per day. This was the extent of the preparation for 800,000 refugees who settled in over July 14-18.

While the UNHCR recommends a minimum of 15-20 L/p/day, the practical goal of relief agencies early in the crisis was to provide 5 L/p/day. This would have been equal to 4-5 million liters of water per day that was just not initially available.

Nevertheless, the international response over the ensuing month was extraordinary. The goal of 4-5 million liters per day was achieved less than one month after the influx, two weeks after the start of the cholera outbreak. A variety of water resources were utilized. At *Katale* camp, furthest from Lake Kivu and Goma, there was a spring from which Oxfam began water production within 5 days of the influx.[48] The 5L/person/day was reached there the first week in August. The municipal water system was refurbished by UNICEF, and the local authority REGIDESO was chlorinating and supplying Goma's residents and the refugees by August. *Kibumba* camp was entirely dependent on tanker water taken from Lake Kivu, an operation initially coordinated by MSF-Belgium. By August 13, there were 60 tankers in operation by the US military, the Dutch military, MSF-Belgium, UNHCR, and UNICEF. The target of 5L/person/day was reached then in *Kibumba*.

Further water requirements were augmented by the US military *Operation Support Hope* which arrived in Goma July 24, almost as the cholera epidemic was peaking. Two Reverse Osmosis Water Purification Units(ROWPU), with a capacity to produce a relatively small amount of clean water, 57600 liters per day, became operational.[*] To finally achieve a large capacity of water distribution, a US company PWSS set up a hydro sub-pump, capable of pumping 3.5 million liters/ day.[48] The ROWPU and sub-pump supplied water to tankers, then injected with chlorine, and

[*] Reverse Osmosis Purification: process by which water is filtered through several filters(carbon) and a fine osmosis filter designed to filter under pressure; filters out dirt and minerals like Chlorine

transported water to *Kibumba*. Goma town was further supplied with water using mobile filtration units from the German agency Technisches Hilfswerk(THW) on August 5.[48] But all these efforts did not slow the epidemic that peaked July 26.

Several observations can be made from the rapidity of the cholera epidemic, and attack rates. The estimate of 30-80,000 cases of cholera in a camp population of 800,000, given a ratio of one active case to ten asymptomatic infected people, suggests that the entire refugee camp was infected. Those without symptoms were probably protected by individual immunity, or the degree of illness was minimized by other host factors or inoculum size. The rapidity of onset suggests a waterborne infection; Lake Kivu was the undoubted source. The short duration of the epidemic again supports the notion of everyone being infected.

There may be other factors that influence transmission of cholera. Proximity of individuals living near an infected water source increases the cholera attack rate. In 1992, cases of cholera requiring hospital treatment in Rumonge, a town located on the Burundi side of Lake Tanganyika, were more frequent in those living closer to the lake than those living further from the lake.[51] Other significant risk factors in this study were drinking lake water, and *time* spent bathing in the lake(swallowing lake water while bathing), suggesting a dose response relationship.

Moreover, cases of endemic cholera have been traced to ports on fishing routes along Lake Tanganyika, and along the Rusizi River, that connects Lakes Tanganyika to Lake Kivu. In July 1994, V cholerae 01 biotype El Tor was simultaneously cultured from refugees in Goma, and in Uvira, the north side of Lake Tanganyika, 160 km apart.

It is possible, therefore, that a source of this epidemic, coincident with a massive influx of refugees, many in the worst state of health, could have been the infected stool of a commercial fisherman traveling this south to north route.

Transmission of diarrheal disease has been linked to contamination of water storage vessels and food by infected hands.[52,53] The CMR of *Katale*, with a spring fed source of water and furthest from the epicenter of the cholera epidemic, was the highest, 41.3 per 10000 per day. *Kibumba* camp, 27 km. from Goma had no water, but had a CMR identical to *Mugunga*, nearest to Lake Kivu. A survey conducted in *Katale*[32] showed that cholera, dysentery, and other diarrheal diseases accounted for 90% of the deaths. *Shigella dysenteriae* emerged as a major problem after August 4. It is possible, therefore, that infected food and water storage vessels, as well as the emergence of Shigella, explained the differences in mortality rates. CMR reflects the quality of treatment provided, which was poor in the camps initially, and the numbers who sought treatment. Those who walked to *Katale* had been in the southern camps first and were exhausted by the time they arrived. The rate of malnutrition was considerable, 30% under 5 years old less than –2Z. Thus, multiple factors converged, and what little water there was became quickly contaminated. The epidemic spread much faster than NGOs could respond. Conditions in the camps were retched and hopeless.

An integral part of the ability to control the rapid spread of cholera is providing for realistic and adequate sanitation. This was a major failure for several reasons. The volcanic rock terrain did not allow pit latrine construction; make shift above ground latrine boxes were eventually constructed. By August 12, according the Overseas

Development Council report, there was one latrine for 1029 individuals in *Mugunga*, one for 500 in *Kibumba*, and one for 184 in *Katale*.[48] Heavy machinery, needed to dig communal latrines, did not arrive in Goma for two months. There was less expertise among relief workers in dealing with this aspect of the overall relief effort. Importantly, media attention was focused on the more newsworthy deaths due to cholera, and consequently there was less attention directed at this vital sector.[48] In such humanitarian crises there is often an "overemphasis on high-profile curative services" and less of a capacity to implement preventative services.[48]

Interventions-Future Policies

Important lessons can be learned from understanding the epidemiology of endemic cholera and the complex humanitarian crisis that progressed quickly in Goma, Zaire 10 years ago. Measles immunization, and vitamin A administration is an important immediate intervention. The outbreaks of meningitis, dysentery, malaria, and contribution of HIV/ AIDS cannot be diminished. The focus of this report, however, has been on what events contributed to the cholera epidemic, and the importance of the provision of clean water. The following recommendations are far from complete.

1. Chlorinate every bucket of water taken from an untreated source. If possible, protect the source from bathing and washing.[51] Narrow necked buckets and storage containers, to prevent hand contamination, should be encouraged and/or supplied by relief agencies.[40,41] The addition of lime juice to buckets, acidifying the water, has been used to control cholera in India.[54]
2. Designate and isolate defecation fields near, but not at, the water source.[29] Latrine construction is as important a preventative measure as provision of ORS. Water and

sanitation technical expertise needs to be represented on the relief team. The rock terrain of Goma was the absolute worse place to dig a latrine, and the camps became isolated for lack of roads. Heavy equipment should not have taken 2 months to mobilize to Goma.

3. A prompt contingency assessment of the deficient municipal water supply should have been a priority; chlorine supplies should have been adequate.
4. Identify existing health systems, eg health posts, and staff capacity. From a long report compiled from the Overseas Development Council there was no specific information about existing health centers in the Goma area, available health manpower, or community health workers. These would have been crucial in any outreach program.
5. Contingency planning and preparedness: pre-position water pumps and piping, supplies of chlorine, more tankers, stockpiles of food and equipment for shelter.[48] This would include tent equipment for emergency treatment centers.
6. Designate cholera treatment centers with adequate storage of clean water to mix ORS, and supplies of Lactated Ringers. Have well-established protocols for case assessment, triaging, and case management. Staff should be experienced in use of ORS.[47,49,50] Many relief workers in the Goma camps had little experience treating cholera.
7. Establish outreach programs to identify sick refugees not able to get to centers, and to initiate treatment with ORS. Community mobilization programs are needed to train community health workers for case finding and referral to the centers.[55] A greater coordinated effort could have been made to assess the severity of morbidity and

mortality outside of the treatment centers, and to have established a means to treat outliers or get them to the center. Transportation off the road was difficult to impossible until the proper vehicles were brought in later in August.

8. Basic education regarding personal hygiene, and soap distribution would be important. Water was so scarce and conditions in the camps so awful that this likely would not have been practical, and better suited for post-emergency phase. Soap has been shown to reduce diarrheal disease in a refugee camp in the absence of a health education program.[56]
9. It is possible that smaller size camps might avert the crowding and other conditions that promote the spread of cholera.[57] Small camps, however, in the Goma area would not have averted the cholera epidemic.
10. A surveillance system and health data needs to guide decisions made. In Goma mortality data indicated that about 40% of the deaths in *Mugunga* camp were due to exhaustion and dehydration. The high death rate the first two weeks after the onset of the outbreak indicates that early handling of the crisis may have greatly impacted the outcome.[31]
11. Support other concurrent relief programs: measles immunization, case identification and treatment of acute respiratory disease, and nutritional support.[58]
12. **Command and coordination**: UNHCR was responsible for overall coordination of relief in Goma in 1994. Technical expertise in water and sanitation was imperative, but initially lacking in the emergency relief team. Control over funding mechanisms is essential. Coordination of many NGOs responding to the emergency through contracting arrangements, and collaboration with other essential international

organizations, like ICRC, could have led to a more coherent strategy. Information sharing, before such emergencies develop, could have enhanced preparations for a mass movement of displaced people that led to tragedy. Critical realistic estimates of the number of refugees was grossly underestimated and the real estimate was not conveyed. The issue of accountability for inaction, lack of adequate information, inappropriate treatments, decisions that have to be made quickly in an emergency, and accessing the views of the beneficiaries of the aid should be mandates of the lead agency. The responsible agency for internally displaced people needs to be determined. Prevention of a tragedy such as Goma should trump sovereignty issues. Finally, competition between humanitarian agencies that jeopardizes objectivity of reporting should have been avoided.

Commentary –Security, Media, and Cholera Vaccine

Significant security issues were present in Goma as Hutu militia entered Goma. Many were armed with guns, grenades, and machete. There were instances of traumatic deaths, and rape. Relief workers were forced to leave the treatment areas at night because there was no safe zone.

Nevertheless, there was military presence. Local Zairian troops were poorly disciplined, and not effectively deployed until 1995. The US and French contingents provided medical assistance, water purification equipment, and management of the airport. Peacekeeping and protecting civilians were not objectives of the military. These reflected the limitations and constraints under which the US commander was operating.[48] Without diminishing the role of the US, French, Australian, British, and Canadian forces

in Zaire, their mandate should have been to disarm the militia, and to keep the peace while providing humanitarian support.

What happened in Goma attracted media attention like moths to a light. An extraordinary gathering of 50,026 journalists and technicians were in Goma and surrounding areas by the end of July.[48] The proximity of the airport to the tragedy enabled them to transport in satellite dishes so that the spectacle of a suffering could be beamed around the world. Media attention was so focused on the tragedy for the world to see that governments like the US flipped a policy of nonintervention to one of humanitarian involvement. Thus, the U.S. government had officially emerged from the shadow of Somalia. However, better control and coordination of the media might have facilitated the delivery of aid.

Cholera vaccines have been proposed as a preventative strategy in refugee crises, but mass vaccination, were it even logistically possible in this setting, would not have halted the cholera epidemic. The only cholera vaccine available in 1996 was the oral whole-cell/ recombinant B-subunit vaccine(WC/bBS). In a study, two doses were administered on day 7 and 14 to 30,000 Sudanese refugees in one month in1997.[59] The administration logistics were daunting: 15 vaccination centers and 114 personnel vaccinating between 250-1700 persons per day. Buffered solution and 200 cc of clean water were given per dose. Immunity was anticipated 2-4 weeks after the second dose, and protection was demonstrated a year later during a cholera outbreak in the camp.[60] It is not surprising that WHO turned down a Walter Read Army Hospital offer to supply 400,000 doses of this vaccine stockpiled since Somalia. A 2 dose oral vaccine, whose administration would monopolize valuable staff resources for protective immunity

peaking at least 2 weeks after the epidemic had run its course, was impractical. Furthermore, what was not revealed on the "60 Minutes" program on which the offer of vaccine was aired in a very favorable media light was the fact that it had expired officially in May 1994. Cholera vaccine, therefore, was considered a pre-emptive and not a reactive strategy.[61] The provision of water, sanitation, ORS, and organization of health facilities along with immediate measles vaccination with Vitamin A administration for children 6 months – 5 years old are still the most important urgent strategies in an acute refugee crisis.[62]

There is a contemporary single oral dose cholera vaccine, CVD 103-HgR, a recombinant live, attenuated vaccine derived from *V. cholerae* Classical Inaba strain in which the gene for CTX subunit A has been deleted, retaining the immunogenic B subunit.[63] Gut derived protective immunity has been detected as early as 8 days after administration. Problems with the vaccine are the short duration of immunity(6 months), and lack of demonstrated cell mediated immunity. Moreover, in cholera endemic areas existing vibriocidal antibodies, thought to be *the* protective antibody, may inhibit colonization of the attenuated oral vaccine in the gut. Thus, more research is needed to find a better vaccine.

Vaccination has proven efficacy in many public health arenas, but the burden of cholera, the lack of sanitation, the contaminated water, the overwhelming masses of people, and absolute chaos and insecurity of the camps around Goma in 1994 mitigated against a single successful 21st century scientific intervention.

The origins of this complex humanitarian emergency emerged from the ethnic profiles of the Hutus and Tutsis, evolving over centuries and influenced by colonial

powers who opened the Great Lakes to the outside. Cholera is an ancient disease. The first Cholera pandemic spread out along trade routes from the Ganges delta in 1817. Since then and John Snow's historic discovery in 1854, much has been learned about this organism and its epidemiology. It thrives in brackish and alkaline water, and in the human intestinal tract. Modern antibiotics and oral rehydration fluids enable us to treat the disease. However, contemporary political and social strife creates the mass movements of people and the tragic and awful conditions of these refugee camps in which cholera in epidemic proportions, as in Goma in 1994, flourishes.

Epilogue

The journey from darkness continued for many refugees who survived the cholera epidemic. By the end of August, 1994 the water provision of 5 liters per person per day was achieved, but by April, 1995 only 10.5 liters per person per day was available, still far below the UNHCR guidelines of 15-20 L/p/day. There were sporadic cases of cholera in 1995. Cases of dysentery, treated with expensive Cipro provided by US Army, were diminishing. Measles outbreaks were common, but a successful vaccination campaign had been conducted by UNICEF, BioForce, and MSF. There was a high incidence of malaria in the camps, but bacterial meningitis was not a major problem after a mass immunization campaign was undertaken in *Kibumba* camp. During the second week of surveillance 34 cases of meningococcal meningitis were detected with a rate of 19/100,000(a rate of 15/100,000 is diagnostic of an 'outbreak').[29] HIV/AIDS was not an apparent problem at that time, but, since a proportion of refugees had come from Kigali where the prevalence was reported 20-30% and rates of violence and rape in the camps was increasing, this disease would likely be a sad legacy of the time after cholera.

There were approximately 10,000 accompanied children that survived the initial months of cholera, and malnutrition. Centers were established to take care of them, but some of the NGOs performed poorly.[48] There was inadequate and inappropriate use of oral rehydration and intravenous fluids. The use of breast milk substitutes was a mistake. There was poor supervision in some of the centers. However, after appropriate education was provided to staff and a pediatrician began to supervise more effectively, mortality rates were reduced by October, 1994.

Security issues prevailed as the most important issue in the post emergency phase of the humanitarian crisis. Roving bands of Hutu militia still exercised the power of intimidation in the camps. Violence was pervasive. The political scene began to unravel in Zaire. There was local ethnic fighting between the Hunde and the Banyarwanda, represented by the rebel forces of Laurent Kabilla. Fighting reached it peak in 1995 resulting finally in the overthrow of Mobutu Sesa Seko.

There was repatriation of approximately 100,000 refugees at the end of July 1994, but this slowed considerably after the killings and persecution by the new RPF regime in Rwanda became known to the refugees. However, at the time of the worst of the civil strife in Zaire from November 15-21, 1996, 500,000 refugees returned to Rwanda from Zaire. The incidence of diarrhea was low. Of the 9407 individuals reporting acute diarrhea, 319 were bloody, with no deaths. There were 23 cases of watery diarrhea. *V. cholerae* 01 El tor was isolated. None of the cases were severe, and none died. There were 38 deaths from diarrhea. The crude mortality rate of 0.1 per 10,000/day compared favorably with the mortality rate of 0.3/10,000/day estimated in *Mugunga* camp from which the refugees had come.[64]

Human Rights

There is no single reason why events such as Goma happened. It is a complex cascade of ethnic differences, colonial influences, ecologic factors, and political and humanitarian responses. If there is one common cause that runs through this history, it is that the resulting health of these displaced people is the result of a violation of human rights. Cholera, other diseases, malnutrition, and dehydration and exhaustion were the causes of death for many, but the mass displacement of people following war and genocide was caused by political turmoil in Rwanda. The social and economic inequities buried in a failing political system in Rwanda, recognized for years before the genocide, as Dr. Paul Farmer would argue, "structured the violence" that ensued.[65,66] It is the pragmatic responsibility of the relief worker to treat the diseases, and provide essential living conditions to victims of humanitarian crises. It is the responsibility of nation states to protect the health and human rights of its citizens. It is also their responsibility, to the degree possible, to protect their citizens social and economic rights. Failing these responsibilities violates the human rights of its citizens.

The major lesson from Goma is that a United Nations should have recognized the origins of the conflict sooner[67], should have predicted what was going to happen without intervention, should have at least been better prepared to act, should have held themselves to a higher standard of protecting human rights in the face of a failing state that jeopardized so many hundreds of thousands of lives, and most importantly should

have **acted** to prevent the horrors of the genocide and the journey from darkness that followed.

Appendix

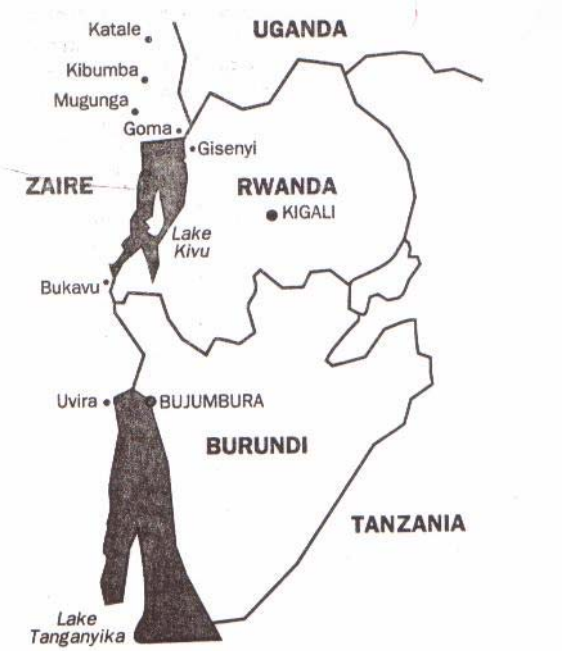
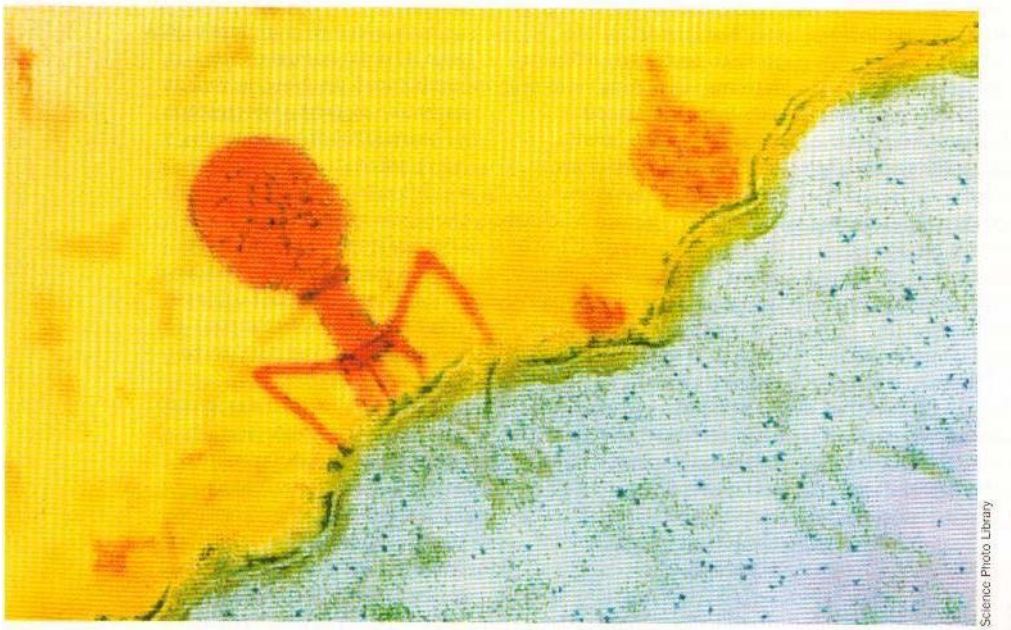


Figure 1: **Map of Rwanda, Zaire, and Burundi**



A bacteriophage uses spidery tail fibres (orange) to secure itself to the surface of the bacterium (blue)

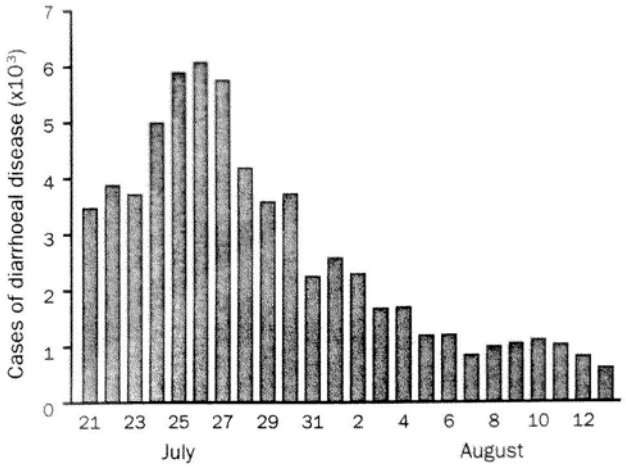


Figure 3: **Reported cases of diarrhoeal disease (cholera, dysentery, and dehydration)**

	Survey period (July 14 to:)	Estimated population ($\times 10^3$)	CMR (per 10 000 per day)	% population dying during period (95% CI)
Katale survey	Aug 4	80	41·3	8·3 (7·1–9·5)
Kibumba survey	Aug 9	180	28·1	7·3 (6·2–8·4)
Mugunga survey	Aug 13	150	29·4	9·1 (7·9–10·3)
Body count (all areas)	Aug 14	500/800	31·2/19·5	9·7/6·0

Table 1: **Comparison of CMR estimates derived from body count and from population surveys**

Camp	Date	Sample size	Percentage of children		Overall percent with malnutrition (95% CI)
			Moderate malnutrition	Severe malnutrition	
Katale	Aug 4	567	16·6	6·5	23·1 (18·3–28·7)
Kibumba	Aug 9	694	17·1	3·0	20·1 (16·1–25·0)
Mugunga	Aug 13	723	14·4	3·3	17·7 (15·0–21·0)

Moderate malnutrition=weight-for-height *z* score less than −2 but more than −3 (between 2 and 3 standard deviations below reference population mean); severe malnutrition=weight-for-height *z* score less than −3 or oedema.

Table 2: **Frequency of acute malnutrition according to population surveys in children 6–59 months**

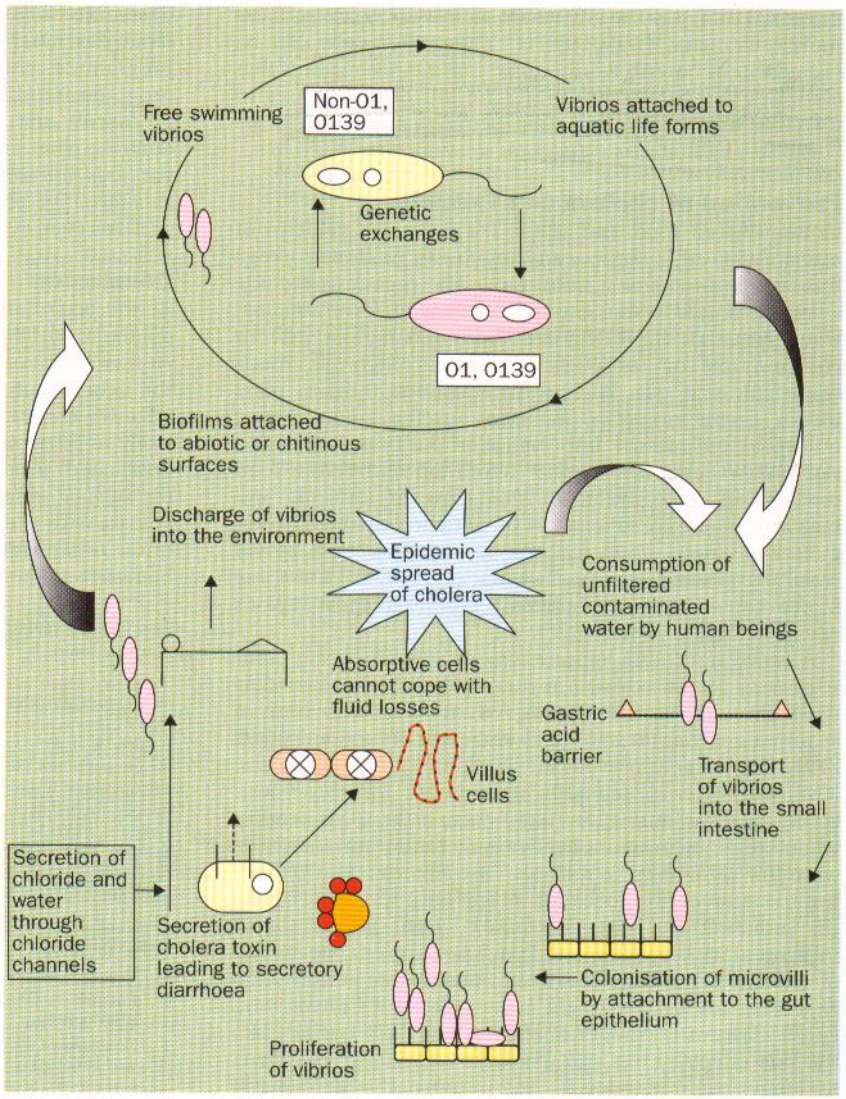


Figure 4: **Life cycle of *V cholerae* involves both environmental and human segments, which sometimes intersect**

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