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Review Article

Lethality of First Contact Dysentery Epidemics on Pacific Islands

G. Dennis Shanks[1,2,3]*

[1]*Australian Army Malaria Institute, Enoggera, Queensland, Australia;* [2]*School of Population Health, University of Queensland, Brisbane, Australia;* [3]*Department of Zoology, University of Oxford, Oxford, United Kingdom*

Abstract. Infectious diseases depopulated many isolated Pacific islands when they were first exposed to global pathogen circulation from the 18th century. Although the mortality was great, the lack of medical observers makes determination of what happened during these historical epidemics largely speculative. Bacillary dysentery caused by *Shigella* is the most likely infection causing some of the most lethal island epidemics. The fragmentary historical record is reviewed to gain insight into the possible causes of the extreme lethality that was observed during first-contact epidemics in the Pacific. Immune aspects of the early dysentery epidemics and postmeasles infection resulting in subacute inflammatory enteric disease suggest that epidemiologic isolation was the major lethality risk factor on Pacific islands in the 19th century. Other possible risk factors include human leukocyte antigen homogeneity from a founder effect and pathogen-induced derangement of immune tolerance to gut flora. If this analysis is correct, then Pacific islands are currently at no greater risk of emerging disease epidemics than other developing countries despite their dark history.

"It occasioned a great mortality—so much so, that the natives state, the living were not able to bury the dead." James Jarves, Hawaii.[1]

Extraordinary infectious disease mortality depopulated many of the Pacific islands soon after they were first contacted by western explorers and sailors.[2–4] The reasons behind the tremendous mortality remain largely unexplained, but apparently now no longer exist in the same island populations. Pacific islands were the last geographic points included in the global pathogen pool, and the admittedly fragmentary record of the earliest epidemics may offer insights into the mechanisms of mortality that presumably once existed for all human societies when they transitioned to live in interconnected populations.[5,6] Most of the highly lethal Pacific epidemics preceded the scientific revolution created by Pasteur's discovery of the microbiological nature of infectious diseases, and were described by the few literate observers in archaic terms.[3] Despite these limitations, the historical record offers the only definite information regarding potential explanations for the extraordinary mortality of first-contact epidemics.

Dysentery is a febrile infection leading to frequent defecation with blood, pus, and mucus in the stool. *Shigella* species are the most common cause of lethal (bacterial or bacillary) dysentery epidemics, and have been important factors in both historical and recent military campaigns and other complex public health emergencies.[7–11] Although historical reports are often hard to fit into modern diagnostic categories, the descriptions of obvious blood in stool makes it highly likely that many of the early epidemics were indeed due to bacillary dysentery. Although amoebic dysentery is an alternative possibility, 20th century dysentery epidemics in the tropics were shown to be overwhelming due to shigellosis, once microbiological cultures were obtained and many of the putative amoeba shown to be leukocytes in the stool.[8,12]

Mortality is an unmistakable clinical endpoint, but descriptions of illness before death during the 19th century reflected the then current worldview of miasmic diseases, in addition to more than a tinge of racial superiority by western observers of the Polynesian and Melanesian populations.[2,13,14] Pacific island populations died at high rates from infectious diseases that must have been introduced by single sailing ships that had no symptomatic sailors on board; indeed most ships had not been into another port for weeks to months before landing on a Pacific island.[1,15,16] Few enteric pathogens (apart from *Shigella*) are capable of asymptomatic carriage as well as the great infectivity and extreme lethality described during early Pacific island epidemics. Most of the existing records were narratives from ship's captains or resident missionaries scattered across the Pacific Ocean.[1,13,15] These records are reviewed recognizing that modern concerns for diagnostic and numerical precision were an unfamiliar concept to many of the people involved, Pacific islanders and their observers alike.

HISTORICAL EPIDEMICS

In 1803–1804, an epidemic labeled "*okuu*" by the Hawaiians killed an estimated 5,000 persons on Oahu as King Kamehameha and his army prepared to invade Maui.[16] It was an enteric disease where "thin discharges like sour starch water flowed frequently from the bowels." Jarves, who wrote long afterwards, reports that the enteric epidemic killed so many as to make disposal of the dead problematic, as shown in the quote which leads this paper, and may partially explain why the estimates of the number of deaths vary by a factor of ten.[1] In 1970, the State of Hawaii Director of Health postulated that *okuu* was due to cholera as the enteric disease with the highest acute mortality rate.[16] This seems unlikely as *okuu* of 1803 predated the first known cholera pandemic by over a decade. The local population identified an unnamed American ship as the source of the epidemic. The most common ships arriving in Hawaii in the early 19th century were whaling vessels that often stayed at sea for months between port visits, indicating that any enteric disease would have been spread by asymptomatic sailors into the local Hawaiian population. In 1850, cholera arrived in Honolulu from California, but there were no further *okuu*-type epidemics described.[17,18]

*Address correspondence to G. Dennis Shanks, Australian Army Malaria Institute, Gallipoli Barracks, Enoggera, Queensland 4051, Australia. E-mail: dennis.shanks@defence.gov.au

Other Pacific islands besides Hawaii also experienced epidemic dysentery. Tahiti recorded a lethal dysentery epidemic after Vancouver's arrival in 1792 and then after the whaler *Britania* in 1807. An extensive dysentery epidemic spread through much of French Polynesia in 1843.[19] The missionary John Williams described what may have been dysentery epidemics linked to foreign ship arrivals that killed some hundreds of people on both the Cook Islands (Rarotonga, 1830 and 1843) and Rapa Iti (French Polynesia, 1864).[15] Rotuma is a Polynesian outlier island 500 km from Fiji with a remarkably good epidemiological record for such an isolated place.[20,21] Dysentery epidemics were recorded in 1861, 1871, 1882, and 1901 with mortality ranging from "there weren't enough people to bury the dead" in 1861, to 73 from a population of approximately 2,000 in 1882.[22] In what was then German New Guinea, the first dysentery epidemic was documented in the contract labor ship *Lord of the Islands* in the 1880s while traveling from New Britain to Queensland.[23,24] See Figure 1 showing a representative contract labor ship. A devastating epidemic was said to have involved parts of New Britain and New Ireland in 1899 with death rates up to one in seven. An epidemic of dysentery from a single infected person in 1924 on the isolated Polynesian island of Futuna killed 24 of 350 or 7% of the estimated total population.[2]

Fiji's reputation as an island of cannibals made it a less visited place by ships than Hawaii in the early 19th century, but Fiji still experienced similar devastation by epidemic dysentery. An epidemic caused by "a very acute form of dysentery" was said by Fijian tradition to have occurred in 1802–1803 and was supposed to have killed greater than one-fifth of the population.[14] When Fijian sugar plantations were established in the second half of the 19th century, mortality accounting improved as the British colonial government made such reporting a requirement of the contract labor trade whereby Melanesian workers were brought to Fiji under near slavery-like conditions.[25] Lethal dysentery epidemics were recorded on various contract labor ships such as *Stanley* (1880), *Surprise* (1882), *Lord of the Isles* (1882), and *Meg Merrilies* (1884).[13,26] Mortality in the plantation workers on the sugar fields of Fiji and Queensland was often 10% per year and was dominated by pneumonia and dysentery.[25] Dysentery was specifically noted as the cause of death in a majority of Melanesian workers in Fiji with an annual mortality rate just from dysentery of nearly 7% in 1884.[27] Similar dysentery death rates were seen in laborers from New Guinea and the Solomon Islands decreasing toward the beginning of the 20th century. It was observed that when new island groups were used as a source of labor, the resulting mortality rates were much higher than areas that had previously provided contacted labor.[25] The Chief Medical Officer of Fiji noted meeting the sole survivor of a group of 115 men from New Britain who had been brought to work in the sugar plantations.[28] Interestingly, Indian laborers working in the same Fijian sugar fields as the Melanesians also became infected, but died of dysentery at much lower rates than the Pacific islanders. The local Fijian population and Pacific island sugar plantation workers who were on their second contract only infrequently died of dysentery.[27]

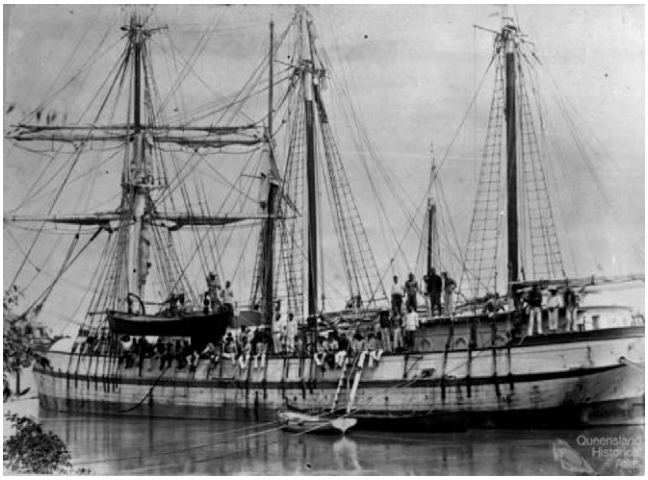

FIGURE 1. Contract labor ship shown bringing Melanesians to the sugar plantations of Queensland, Australia. Dysentery epidemics occurred on several such ships during the 19th century. Collection of John Oxley Library, State Library of Queensland, image 2246.

A particular dysentery epidemic on the contract labor (aka blackbirder) ship *Meg Merrilies* was described in detail by Daniels in 1890.[29] New Hebrides (Vanuatu) and Solomon Islands laborers (N = 120) were being transported to Fiji where they were quarantined once it was known that dysentery had appeared onboard. Thirty-one dysentery cases (attack rate of 26%) were tracked in detail with incubation periods varying from 2–12 days. The feces were described as "frogspawn stools" and as being similar to "large grains of sago pudding" indicating enteric inflammation. Nearly half of the cases (48%) died within 10 days of becoming ill, and in six cases, postmortem exams were conducted. Autopsy findings included acute inflammation marked by a blackish green deposit covering the whole of the large intestine from the rectum and extending into the distal ileum. The small intestine was also noted to be acutely inflamed and contained a red fluid "not unlike anchovy sauce." Only two persons with such grossly bloody stools survived.[29] Stomatitis (many), genital ulceration (N = 2), and reactive arthritis (N = 5) taking months to resolve likely describe Reiter's syndrome making the epidemic's etiological agent of *Shigella* very likely.[29,30]

Dysentery in sugar plantation laborers of Fiji remained such a problem into the 20th century that a special yearlong research project was arranged by the London School of Tropical Medicine for Philip Manson-Bahr in 1910.[26] Manson-Bahr found that, by 1910, the rate of dysentery in Fiji was markedly less than in the 19th century. The case fatality rate for dysentery in hospitalized patients in Fiji during 1903–1909 ranged from 5.2 to 12.8%. Fiji experienced an epidemic of dysentery (estimated 300 cases) after a cyclone on 24 March, 1910; tropical storms being known as an inciting event for epidemic dysentery from previous work in Puerto Rico.[31] Bacterial cultures of dysentery patients showed that both *Shigella dysenteriae* and *Shigella flexneri* were equally common. Only 11 cases of amoebic dysentery were found in Fiji during all of 1910.[12] During Manson-Bahr's study, 7% of the 159 bacillary dysentery patients died. Risk factors were found to be prisoners in gaol or dockworkers, male gender, and illness occurring during the rainy season at year's end.[12] Manson-Bahr subsequently studied much larger numbers of dysentery patients during the Gallipoli and Salonika campaigns of the First World War.[7] Effective treatment in hopes of preventing chronic illness only became

feasible after the introduction of antibiotics during the Second World War.[8,32,33]

DYSENTERY ASSOCIATED WITH MEASLES EPIDEMICS

Measles is a respiratory viral infection that can be reliably diagnosed from historical reports and was also known to kill thousands of Pacific islanders during first-contact epidemics during the 19th and 20th centuries.[5,34] Unlike dysentery, which was usually the first described epidemic disease after first contact, measles was generally the last highly lethal introduced infectious disease as sailing ships could not transit the Pacific within the 2-week incubation period of measles. Once steamships were introduced during the middle of the 19th century, live measles virus could be carried in nonimmune passengers to the Pacific islands within the incubation period.[5] In the best documented instances, measles was brought by naval warships to Hawaii in 1848 from Mexico by the *USS Independence*, and to Fiji in 1875 from Australia by *HMS Dido*.[17,34] Measles is relevant to dysentery not because there was any diagnostic confusion, but because a frequent postmeasles symptom after first-contact measles epidemics in the Pacific was dysentery. This is only rarely seen in other populations, and is reviewed in detail as infection with an immunosuppressive virus such as measles is likely to be informative regarding the immune status of Pacific islanders in the 19th century.[35]

Mortality after first-contact measles epidemics in the Pacific that included all ages was usually not from common respiratory causes such as pneumonia, but from inflammatory gastrointestinal symptoms described as dysentery. During the 1848 Hawaiian epidemic, it was stated that "A diarrhea then succeeded the measles, which affected the great mass of the people."[17] After the 1875 Fijian epidemic, it was observed that "the consequence is that they are almost invariably attacked with dysentery, which very speedily carries them off."[36] During the 1894 Samoan measles epidemic, the local newspaper wrote that measles had caused "at least three hundred deaths although some of these had been caused by a kind of dysentery which attacked persons convalescing from measles."[3,37] Enteritis and dysentery were stated to be the predominant lethal complications during a measles epidemic in Fiji in 1903.[38]

The best descriptions of postmeasles enteric inflammation come from one of the last initial contact measles epidemics that occurred in Rotuma in 1911.[20–22] Nearly a quarter of the population of Rotuma died in 1911, when measles swept the small island. Three-quarters of the measles deaths were attributed to gastrointestinal causes such as ileocolitis. The medical officer at the time, Dr. Hugh MacDonald when writing to the Chief Medical Officer of Fiji stated, "As to the nature of this disease: being a new disease among the people it was of necessity severe; and the appearance of the rash was accompanied by the symptoms of acute indigestion, and later gastroenteric intoxication. Many recovered after showing these symptoms in a marked degree; but most, after such a recovery, passed on to the inflammatory stage and presented the signs of ileocolitis—fever, diarrhea—with blood and mucus in the motions, tenesmus, etc… others when the symptoms of the succeeding ileocolitis had subsided have relapsed again and gone to their graves from a similar cause."[39] It is likely that the subacute gastrointestinal inflammation described was not from direct measles infection of the gut, but was a result of the known immunosuppression of measles disordering the normal host tolerance of enteric microorganisms.[35,40] Systemic immunosuppression by measles was confirmed in Rotuma, as the year 1911 also saw the greatest tuberculosis mortality ever recorded, which was certainly due to reactivation disease and not an epidemic of new infections.[20]

IMMUNE ASPECTS OF FIRST CONTACT EPIDEMICS

Isolated island populations were clearly subject to disastrous outcomes when new infectious diseases were first introduced, but this often did not extend to subsequent epidemics by the same pathogen.[3,5,38] Although some of this amelioration occurred because, by this time, there were immune persons on the island able to care for those becoming ill during secondary epidemics, this does not appear to be the whole explanation.[41] Mathematic models make it clear that whatever the reason the extreme mortality rapidly decreased after the first-contact epidemics on Pacific islands, it was not due to Darwinian selection of disease-resistance genes. The time interval of only 2–3 generations is simply too short to involve such putative disease-resistance genes. The host immune system in small isolated island populations must have been different from those who had been thoroughly integrated into the global pathogen pool from birth.[6,42]

Although there are few living populations from which to draw direct observations, some modern studies of enteric organisms in a very isolated Amazon River Amerindian group have been done.[43] Surprisingly, these uncontacted people were found to have the most complex and varied microbiome ever reported despite isolation for > 10,000 years and no use of antibiotics. This suggests that the natural state of uncontacted human populations is one of great microbiological variety that had accumulated over time and that this variety contributed directly to host tolerance of enteric organisms through immune interaction through the gut. Shigellosis would have massively disrupted such an enteric ecosystem and measles would have disrupted host tolerance of enteric organisms through immunosuppression.[44] Thus, both pathogens lead to a single common pathway of inflammatory enteric disease clinically seen as dysentery.

DISCUSSION

The reconstructed mosaic of first-contact epidemic reports from the Pacific indicates that the initial epidemic was usually the most lethal with anywhere from one-fifth to one-third of the population dying acutely.[3,5,45] Since the first such epidemic usually preceded the arrival of literate observers, retrospective diagnosis is difficult, but most of these initial epidemics involved primary gastrointestinal symptoms such as dysentery. Given the close relationship of the Pacific island epidemics with the arrival of ships from continental ports, it is highly likely that these represented point-source introductions of single, highly infectious pathogens.[13,15] Shigellosis being the enteric disease requiring the least number of organisms to establish an infection, one could logically conclude that the most likely cause of many of the first-contact epidemics was bacillary dysentery due to Shigellosis.

The extraordinary lethality of first-contact epidemics remains poorly understood, but could be hypothesized as the following chain of events. Epidemiological isolation produced a situation that placed Pacific island populations at extreme mortality risk from what are today considered to be "ordinary" pathogens, likely due to failures of host immunity.[20,42] Whatever the gap in host immunity, it apparently corrected itself within a very few generations making genetic causes unlikely. More plausible possibilities include epigenetic modifications of the immune system as seen with early Bacillus Calmette–Guerin immunization.[46] Early exposure to attenuated infections has been shown to cause a remarkably large decrease in all-cause mortality.[47] Additional risk factors could have been due to the very narrow genomic base of such isolated populations which would have produced a high level of human leukocyte antigen homogeneity through a founder effect or a Th2 bias created by exposure to a very limited set of pathogens such as helminths.[48–50] In dysentery, disruption of the stable enteric ecosystem by a single pathogen was magnified by the host response into a dysfunctional inflammatory reaction that presented as dysentery. Death was through a protein-losing enteropathy that further degraded the host's ability to respond to infections not unlike those seen in severely malnourished groups such as prisoners of war.[51–53] Although improvements in sanitation and medical care may have contributed to the fall in mortality, this seems unlikely before the 20th century.

If true, this hypothesis would have both current and historical implications. Although Pacific island populations were extraordinarily vulnerable to infections in the past, this probably does not apply to newly emerging pathogens.[54] Although influenza killed many in the United States and Europe in 1918, extreme mortality during the 1918 influenza pandemic in the Pacific (one-fifth of Samoans died) was directly related to the degree of epidemiological isolation; Pacific islands did not experience unusual mortality during the 2009 influenza pandemic.[42,55] Previously uncontacted populations would likely have been eventually devastated by infectious diseases regardless of the care that might have been taken to avoid exogenous pathogen introduction.[45] If epidemiological isolation was the risk factor that created host vulnerability, this could not have been modified without connection to the global pool of microorganisms.[42] For the few uncontacted groups remaining isolated in distant jungles, they should remain isolated, and severe infectious disease mortality is to be expected if they spontaneously find the outside world.[56,57] The supposed racial superiority of the western explorers was a product of exposure to a wide variety of enteric and respiratory pathogens from birth and not generational selection for disease-resistance genes.[58] As one cannot collect clinical specimens from dysentery patients of previous centuries, the offered hypothesis may be as close as one is able to reconstruct the tragic events of first-contact epidemics on Pacific islands.

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Author's address: G. Dennis Shanks, Australian Army Malaria Institute, Enoggera, Queensland, Australia, E-mail: dennis.shanks@defence.gov.au.

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