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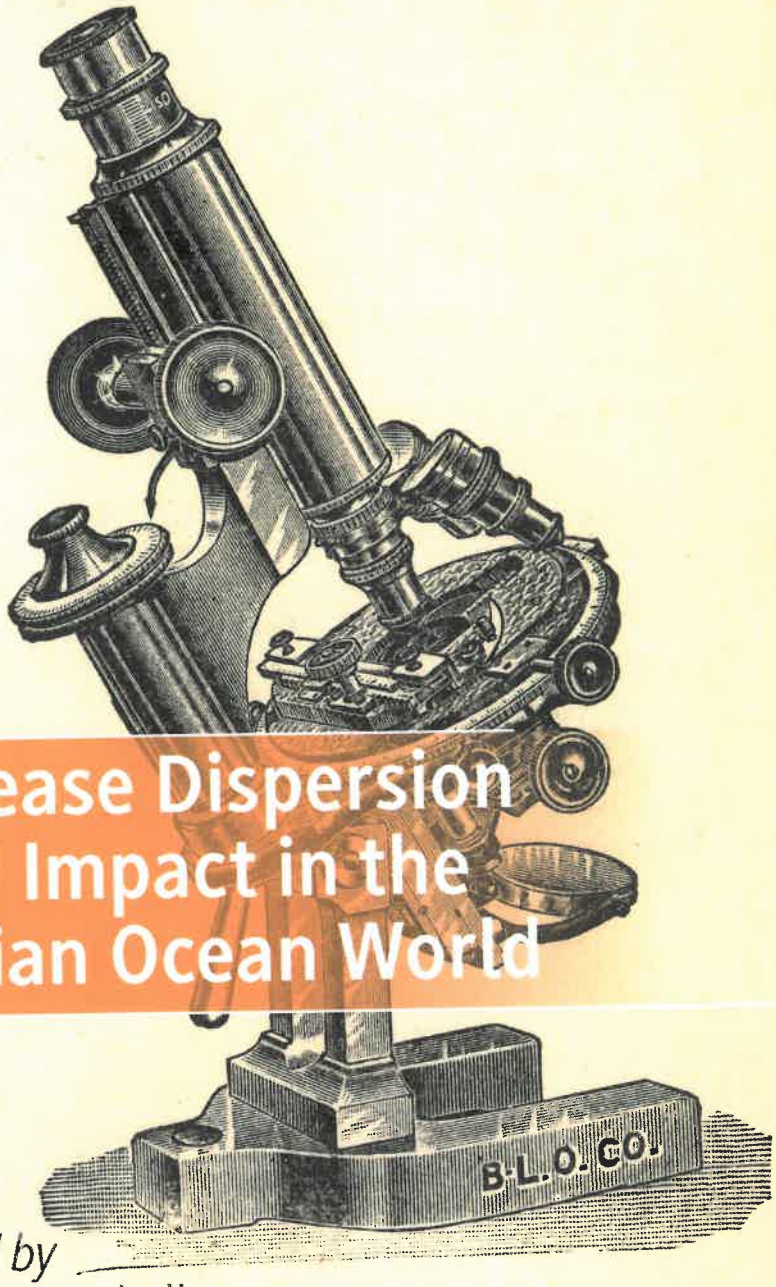

Disease Dispersion and Impact in the Indian Ocean World

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CHAPTER 1

Introduction

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Environment and Movement

Throughout history in the Indian Ocean World (IOW) diseases have, under certain distinctive geographical and climatic conditions, emerged and spread, generating a number of impacts on varying spatial scales. The IOW, a macro-region lying between latitudes 45°S and 45°N running from Eastern Africa through the Middle East, South and Southeast Asia to East Asia, encompasses tropical, sub-tropical, and temperate zones, major oceans, gulfs and rivers, islands, lakes and deserts, and the world's highest mountain range (Map 1.1). It thus experiences major differences in temperature and rainfall, which are further affected by other environmental factors—the most significant of which is the monsoon system of winds and currents that governs the IOW littorals and seas north of about 12°S of the equator. In the northern hemisphere summer, the southwest monsoon dominates, bringing heavy rainfall to the Asian littoral, while in winter the

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system switches direction, creating the northeast monsoon. Most historians have assumed the monsoon system to have been stable, but it could unpredictably fail, triggering drought, crop failure, famine, and disease. A range of other, often associated, environmental factors, such as the El Niño–Southern Oscillation (ENSO), Indian Ocean Dipole (IOD), Intertropical Convergence Zone (ITCZ), volcanism, and cyclones could also significantly impact temperature and rainfall and thus disease. For example, in the aftermath of heavy rain, stagnant pools of water could form, providing breeding grounds for mosquitos and other causal agents of diseases such as malaria, filariasis, dengue, and chikungunya (cf. Meunier 2014). Again, heavy monsoons, cyclones, seismic activities, tsunamis, and storm surges could lead to flooding that might in turn create favourable conditions for pathogenic microorganisms and thus for the spread of water-borne and contagious diseases such as cholera, dysentery, and polio or *Escherichia coli* infections. Furthermore, weather extremes and natural disasters were often followed by famines, conflict, and migration, all of which increase health hazards.

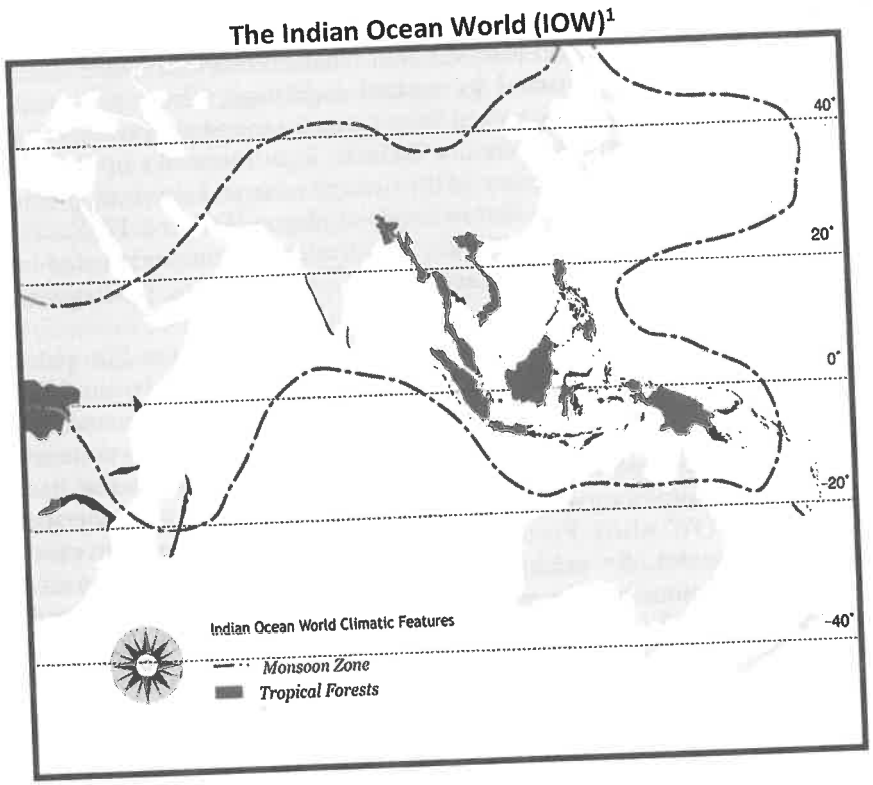
In addition to these environmental specificities—this "deep structure" of the IOW (cf. Pearson 2003)—the macro-region witnessed the rise of the first "global" economy from around 300 BCE. The IOW global economy, linking Eastern Africa and the Middle East to China and all places in between through the creation of a sophisticated network of overland, riverine, and maritime communication, was characterized by an intensifying exchange of plants, animals, and (both voluntary and involuntary) humans—creating the quintessential conditions for disease diffusion. This process, which started with early hominid migration out of Africa, triggered the development of regionally specific immunological responses. With the advent of long-distance trans-IOW seafaring, the entanglement of humans and pathogens gained a novel epidemiological momentum affecting both littoral and hinterland communities (cf. Campbell 2019; Schnepel and Alpers 2017; Pearson 2015; Alpers 2014; Sheriff 2010). The interconnected character of the IOW global economy, and increasing concentration of human and animal populations close to water resources, transformed the IOW into one interconnected disease zone (Issa 2006; Arnold 1991). It formed, for example, a centre of dispersion of a number of diseases such as the plague, smallpox, malaria, and tuberculosis.

However, disease outbreaks and dispersion did not occur in a historically linear fashion. The IOW global economy underwent major cycles of expansion and contraction. The main eras of economic expansion were

from approximately 300 BCE to 300 CE, between the ninth and thirteenth centuries, and again from the mid-nineteenth century—the intervening periods being marked by general stagnation. Times of overall economic prosperity, characterized by enhanced agricultural productivity and demographic growth, were not immune from outbreaks of disease. However, it is notable that some of the most notable and devastating episodes of disease, such as the first and second plague (541 and 1347 CE) and cholera (e.g. 1817 and 1826 CE) pandemics, not only originated in the IOW but erupted during periods of major economic uncertainty (Campbell 2019).

Within this context, there is considerable debate about the European impact on the IOW. For some scholars, such as Arnold, the advent of the European presence from 1500 marked a major epidemiological watershed for the IOW (Arnold 1991). However, Campbell argues that, in contrast to the New World to which Europeans carried Old World diseases that had a catastrophic impact on indigenous populations, the reverse was generally true in the IOW where Europeans proved highly vulnerable to tropical diseases. This was, for example, the case with malaria to which many African populations had, through genetic adaption (sickle cell), acquired resistance. Thus the Portuguese in Mozambique suffered such high mortality from malaria that they often lacked sufficient soldiers to maintain a garrison. For largely the same reason, European attempts to found settlements in Madagascar failed. Only with the widespread adoption of quinine from the late nineteenth century could European soldiers and colonists settle malarial regions of the IOW (Campbell 2019 and contribution to this volume).

The nineteenth century marked a major turning point in the disease history of the IOW for a number of reasons, many of which were related to the rise of a truly international economy that, by the eve of the First World War, had drawn all bar the most peripheral societies into the orbit of modern capitalism. First, with the exception of railways in India, and of late nineteenth-century investment in mining in South Africa, few areas of the IOW benefitted from the enormous outflow of capital to extra-European regions from financial centres, notably London and Paris, based in Western Europe. Consequently, growing demand for tropical and semi-tropical products from a rapidly industrializing West resulted in a commercial boom in the IOW that was largely labour intensive. This was the case with both European and indigenous enterprise in the macro-region. Manpower was required to clear land and cultivate cash crops such as



Map 1.1 The Indian Ocean World (IOW). © IOWC

cloves, sugar cane, coffee, tea, and cocoa; collect forest products such as gum and tropical hardwoods; hunt and extract prized animal products such as ivory, rhino horn, skins, pearls, and whale oil; transport such produce to ports, and carry imported articles into the interior; load and offload commodities at ports; and provide the crews of commercial vessels. However, in the 1800s the IOW failed to experience the same rates of demographic expansion as the West, and, as much labour was already tied up in indigenous forms of bondage, there existed a very limited wage labour force from which to hire workers. As a result, European and indigenous IOW authorities, traders, and entrepreneurs, resorted largely to the use of bonded labour. This was reflected in the continued use of slaves and the increased use of penal and especially of indentured labour. There

developed a large-scale system of bonded labour movements, both intra-IOW and from the IOW to labour markets in other regions of the world, such as the Caribbean, characterized by labour shortages. These large-scale long-distance movements, which continued well into the twentieth century, brought immunologically naïve people into contact with previously unknown diseases and environmental conditions. Such mobile or displaced people, in turn, carried diseases, predispositions for certain conditions, and specific immunological responses to new locations (Campbell 2018, 2014; Sheriff and Ho 2014).

Additionally, the nineteenth and twentieth centuries were marked by major imperialist ventures, both European and indigenous, in which there occurred massive movements of troops and camp followers. Military expeditions often led to significant displacements of populations, both combatant and non-combatant. At the same time, the presence of major concentrations of male soldiers and workers resulted in a rise in the transmission of sexually transmitted diseases (STDs) and of alcoholism. First, such concentrations of males (few European females accompanied white soldiers to the IOW, and indentured labourers were predominantly male—overwhelmingly in the case of the Chinese) established a huge demand for sexual services. This in turn resulted in an often officially encouraged system of recruitment and migration of young women from mostly impoverished backgrounds, who were often forced into the provision of such services. Inevitably, these systems resulted in an explosion in the transmission and diffusion of sexually transmitted diseases. Massive concentrations of male soldiers and workers also led to an unprecedented rise in, and growing public concern about, alcohol consumption. This theme is explored in this volume by Peter Hynd and Manikarnika Dutta who reveal that, in nineteenth-century India, alcohol was a highly lucrative trading commodity that some officials considered to be a panacea for certain deadly diseases such as cholera and the bubonic plague. Official tolerance of the production and sale of alcohol encouraged European seamen and soldiers to consume it, often in adulterated form, on unprecedented levels. However, missionaries in the field, and members of a burgeoning temperance movement in Western Europe and North America, argued with increasing force that alcohol was hazardous to health and morality.

The nineteenth and twentieth centuries were also characterized by an ever accelerating series of technological advances that further propelled disease dispersion. This was particularly the case with transport innovations, notably the expansion in the use of railways from the mid-nineteenth

century, steamships from the 1870s, civil aviation from the 1930s, container and liner shipping from the 1950s, and long-haul flight mass tourism and travel in the modern era (cf. Alpers 2014; Mitchell 2016).

Disease in IOW History

Disease has played a crucial role in shaping the size, movement, and settlement patterns of human populations as well as their peaceful or conflict-driven encounters. Moreover, thinking about and acting upon disease is central to all world views and thus provides a window into how societies perceive events and their progression. Hence, disease serves as an indispensable cornerstone in the reconstruction of human history. Disease is experienced, socially understood, and governed, in the broader understanding of Foucault (2011), as comprising governmental interventions as well as techniques of self-control. This allows for a wide range of investigative approaches that include the addressing of individual experiences of illness, the social organization of care, and institutional interventions.

This volume views the study of disease as essential to an understanding of the key historical developments underpinning the foundation of contemporary IOW societies. In this exercise, it is necessary first to define what we mean by "disease" and second to examine the exchange dynamics of pathogens and healing techniques across both the terrestrial and maritime zones of the IOW. We here follow historian Andrew Cunningham's identification of three fundamental dimensions of disease as "(1) an *experience*—an experience of debilitation, pain, suffering, together with (2) the spontaneous *appearance of non-customary phenomena* with respect to the body, such as spots, vomiting, sweating, aches, and (3) … *outcomes* of recovery, death or disability" (Cunningham 2002, 13). Furthermore, humans "seem to insist on seeking reasons or *causes* for disease: for its incidence, its origin, its course, its outcome" (ibid.).

The origins and routes of disease in the IOW were both terrestrial and maritime. The fourteenth-century pandemic of bubonic plague, commonly called the Black Death, offers a prime example of the terrestrial origins and diffusion of disease. There exist three endemic foci of rodent populations that carry *Yersinia pestis*, the organism that causes bubonic plague: the Eurasian steppe (between Manchuria and the Ukraine), Himalayan foothills, and Great Lakes region of East Africa. The second plague pandemic started probably in China in the 1330s. It erupted because of two occurrences: an epizootic of the plague amongst the rodent population and sustained contact between the affected rodents and

humans. These were probably coterminous and related to major environmental events that upset the habitat and challenged the immune system of the rodents. First, from about 1300, China entered a prolonged period of economic and political turbulence. Second, East and Southeast Asia experienced major climatic and environmental disturbances. In China, the early 1330s were characterized by excessive rain and flooding, while in 1334, Mount Kelud in Eastern Java erupted with a volcanic flux magnitude comparable to the 1815 Tambora eruption ($f \approx 26.3$ kg km^{-2}). Such eruptions resulted in reduced temperatures for two to three years, and harvest shortfalls, often accompanied by famine and disease. During these early fourteenth-century events, an epizootic of plague erupted amongst a population of rodents, forcing them to flee their natural habitat and seek refuge and food in communities of humans, to which the pathogen-carrying fleas transferred from the dying rodents. The disease affected visiting traders, subsequently travelling with some of them along the overland Silk Road to the Near East and Europe: this route ran from China's Mongolian border across Central Asia to Neyshabur (Nishapur), in North-Eastern Iran, from where major trade routes ran to Northern India, Mesopotamia, and on to the Persian Gulf or the Mediterranean, and north via the Caspian Sea to Russia. At the pace of 30 km a day, it would have taken such traders and the plague just over four months to travel from China's Mongolian frontier to Neyshabur. Approximately halfway lies Lake Issyk-Kul, near which are located two mediaeval cemeteries of Nestorian Christian traders. These reveal that at least 106 of the 650 people buried there between 1186 and 1349 died in 1338–1339—and that "pestilence" was marked as the cause of death of at least 10 of them. It is estimated that in Europe, the Black Death killed one third or more of the population (Campbell 2019; Reid 2018; Bos et al. 2016; Sussman 2011; Cohn and Weaver 2006).

Whereas human diseases emanated overwhelmingly from land-based sources, the development of transoceanic voyaging ensured that ships, their cargoes, crews, and passengers also constituted highly significant factors in the long-distance transmission of disease. Most scholarly work on the bio-cultural history of intentional and unintended transoceanic exchange of diseases has focused on the Atlantic in the era of European expansion (e.g. Crosby 1972). Only following David Arnold's seminal 1991 article emphasizing the epidemiological distinctiveness of the Indian Ocean have the maritime spaces of the IOW entered the academic discourse on disease zones of exchange and transformation (Arnold 1991). The early domestication of animals, development of densely populated

trading hubs, and rise of trans-IOW oceanic sail allowed for germ exchange across the maritime spaces of the macro-region from about 300 BCE—well before the European incursion into the IOW from around 1500. Arnold, whose focus was specifically on the Indian Ocean, stressed the development of "epidemic highways" (ibid., 4).

Not least of these were criss-crossing pilgrim routes, notably those of the Muslim hajj to Mecca, which constitutes "one of the greatest, and longest lasting, maritime passenger traffics in the world" (Pearson 2015, 9). Due to the IOW's deep pre-colonial historical interconnectedness, the European impact was not as abrupt and catastrophic as in the New World (Arnold 1991, 5). Nevertheless, as Arnold points out, "the emergence of India as the lynch pin of British power and trade in the East was of great epidemiological significance for the rest of the region and indeed the wider world beyond" (ibid., 7). In the contemporary era, the IOW has witnessed the eruption in epidemic and pandemic forms of a number of diseases such as dengue, chikungunya, SARS, Zika, and subtypes of influenza (Weaver and Lecuit 2015; Zeller 1990; Alpers, Jansen this volume).

The Role of Islands

Of notable significance in disease formation and distribution processes in the IOW were islands (cf. Falola et al. 2019; Pearson 2003; Alpers 2000, 2014)—the focus of five of the chapters in this volume (i.e. Alpers, Campbell, Jansen, Knoll, Warren). Taking advantage of the "laboratory" quality of island settings (cf. Cliff et al. 2000), these contributions reveal in condensed form processes of disease formation, dispersion, and management. As tracts of land surrounded by water, islands are separated, even isolated, as well as bridged and connected by water. Islands thus were both convenient outposts on the peripheries of countries, societies, and empires, and indispensable nautical nodal points in complex maritime networks. As crucial stopovers in trade, stepping stones in migratory movements, and gates to continental hinterland resources and power, islands were also, inadvertently, pivotal centres of endemic disease formation, of virulent epidemic invasion, and of the exchange and expansion of diseases.

Indeed, islands were overexposed to specific health hazards. Smaller islands, often densely populated, were vulnerable to resource scarcity, seasonal storms, drought, and flooding—which often created unique disease environments that challenged the human immune system. Plantation projects on larger islands such as Java and the Philippines could have lasting impacts on human health and wellbeing. Some islands, such as the Maldives and Madagascar, even gave their names to supposedly distinctive fevers. In addition to ecological factors, islands could be precariously dependent on sea traffic and the changing fortunes of the IOW global economy. Some islanders, notably those in pivotally strategic locations along the IOW maritime networks, such as coastal Ceylon, Sumatra, and Java, as well as on smaller islands such as Anjouan and Mahé, were in early and regular contact with voyaging seafarers, traders, and travellers. At the centre of inter-regional and cross-IOW exchange, they were shaped by cosmopolitanism and sexual relations across racial and ethnic boundaries. Islands were distinctive disease dispersion hubs. Genetic founder effects weighed heavily on small populations, and epidemics travelled fast through close-knit island communities. Moreover, the enduring turbulent history of many IOW islands led to the juxtaposition of multi-ethnic and multi-religious populations with corresponding social tensions and conflict dynamics, as is conspicuous, for example, in the case of Sri Lanka.

Their geographical location, and historical circumstances, made some islands epidemiological avenues to hinterlands and staging posts along the epidemic highways in the IOW disease zone. Domination of islands and littorals was thus about controlling not only trading routes but also the pathways and speed of epidemics. Discrete island spaces that existed in abundance in many parts of the Indian Ocean, and were generally manageable due to their small size, served also as liminal spaces, usable, for example, as quarantine islands or leper colonies.

Early Disease Control and Treatment

One of the major human reactions to disease in the IOW was the development and exchange of therapeutic efforts of disease control and treatment (Issa 2006), and theoretical considerations of disease cause and transmission. The IOW is the birthplace of some of the world's oldest medical systems, and a space of intensive exchange of healing techniques, materials, and knowledge. This included the exchange and interlocking dynamics of therapeutics, *Materia medica*, and skilled practitioners of the Ayurveda, Chinese, Tibetan, Yunani (Arab-Persian) medical systems, as well as of Prophetic and Islamic medicine, and non-textual healing rituals and practices (Winterbottom and Tesfaye 2016). Moreover, the IOW was a laboratory for emerging scientific fields such as virology, malariology, parasitology, and tropical medicine. Major biomedical breakthroughs

occurred in the macro-region, including the discovery in India in 1897–1898, by future Nobel laureate Ronald Ross, of malaria transmission by the *Anopheles* mosquito. Quinine, the most widely used prophylaxis for malaria, appears in the IOW to have been first used in Madagascar (Campbell, this volume)—after Jesuits gained knowledge about the traditional medical use of the ground bark of cinchona trees by the Quechua, a people indigenous to Peru, Bolivia, and Ecuador, and transferred this knowledge to Europe in the later sixteenth century. The most recent form of health-related mobility in the IOW is the development of a thriving medical tourism industry with major centres in Singapore and Bangkok, and a correlating increase in medical travel by international patients (e.g. Knoll 2017).

Three decades after David Arnold's seminal article, and three years after the subject was further enhanced by a collection edited by Anna Winterbottom and Facil Tesfaye (2016), this volume sets out to shed further light on the dispersal and impact of disease in the IOW. Disease knows no political or man-imposed frontiers, and some pandemics, such as cholera in the nineteenth century, crossed the entire macro-region which, as noted above, runs from Eastern Africa in the West to China in the East. Indeed, cholera, like plague and some other diseases emanating from the IOW, spread beyond the IOW proper. Time wise, this volume covers the longue durée, from the arrival of early hominids in the IOW up to the twenty-first century. The dynamics of various diseases are here reflected against the backdrop of major transformations in the mobility of humans within the IOW and their interaction with disease. IOW mobility covers travel on foot, mules, camels, and horses and by everything from animal-drawn carts, railways, cars, buses, and sail and steam ships to aeroplanes. Moreover, medical history has, over the centuries, progressed from the study of miasma-based disease and, by the latter part of the nineteenth century, germ-based disease, to a post-Second World War focus on molecular and gene-based disease. The available sources for a reconstruction of IOW medical history have thus expanded, from purely symptomatic patient descriptions, to predominately anatomic definitions of disease (following the initial use of dissections during the Renaissance), to what Michel Foucault has called "the clinical gaze" in the mid-nineteenth century—that is, to ever more detailed test results focusing on disease as a composition of symptoms, rather than a reliance on the subjective descriptions of patients (Foucault 1973), and since the 1990s to DNA analysis and to ancient DNA (aDNA) sequencing.

Origins, Routes, and Impact of Disease

Debates over the origin, dispersion, and impact of disease form a central focus of this volume. In their chapter, Monica H. Green and Lori Jones reconstruct the pre-modern trajectories of malaria, tuberculosis, leprosy, smallpox, and plague in the IOW, although they caution that more ancient DNA needs to be retrieved in order to fully incorporate the macro-region into molecular narratives of global medical history.

Malaria is caused by the protozoan parasite *Plasmodium*, four species of which are responsible for the disease in humans: *P. vivax*, *P. falciparum*, *P. malariae*, and *P. ovale*. In this study, Green and Jones focus on the two most dangerous species, *P. vivax* and *P. falciparum*. The older vivax strain is associated with an Asian and the falciparum with an African centre of distribution. *P. vivax* may have passed, in modified form, from primates to humans. It possibly became most prevalent when the hominid migrants out of Africa became settled rice farming communities. *P. falciparum*, by contrast, appears to have originated as a human disease in Africa 10,000 years ago and to have diffused geographically chiefly through the slave trade: it spread throughout the Roman Empire and travelled via the trans-Atlantic slave trade to the Americas where it found particularly fertile ground on sugar and cotton plantations.

Tuberculosis seems to have an East African origin, although more virulent TB strains were introduced to the IOW with European colonialism. Smallpox was probably another quintessential IOW disease, originating from human-camel-rodent interactions in the Horn of Africa in the second millennium BCE. Those who survived initial attacks developed lifelong immunity to it, so smallpox could only thrive by moving as a "childhood disease" between communities. IOW trade networks provided it with the ideal means of dispersion. Again the slave trade may have played a pivotal role. Prolonged physical intimacy, such as that which existed in concentrated groups of slaves, seems to have been key to the transmission of the slowly replicating leprosy organism. Current academic thinking is that the plague had a Northern Eurasian origin, although there is an intriguing possibility that all three plague pandemics may have originated within the IOW: the first "Justinian" plague pandemic in East Central Africa (Campbell 2019) from where it spread via maritime routes to Ethiopia and Egypt, the second in Central Asia from where it spread via the Silk Road, and the third in Yunnan, China, from where it initially dif-

fused slowly via overland routes and then became global through maritime routes.

In her chapter in this collection, Anna Winterbottom demonstrates commonalities and connectivity across the IOW in how people understood the "Frankish disease" and treatments for it. In Sub-Saharan Africa and Southeast Asia, references to the Frankish disease, considered to have been the same as the "Great Box" disease, or syphilis, in Europe, were rare or absent. The presence of forms of the malady caused by other strains of the bacterium *Treponema pallidum*, such as the non-venereal yaws, seems to have prevented these areas of the IOW from experiencing major epidemics of syphilis. Elsewhere in the IOW, the Frankish disease was generally believed to have been caused by close contact with foreigners. Tracing symptoms and epidemiological blaming across the IOW, Winterbottom demonstrates that in the IOW derivatives of the term *afrang* (from ancient Persian for "Frank"—i.e. the "Frankish disease") were used to refer to syphilis, the suspected transmitters of which were frequently Europeans and the prostitutes they frequented. During the colonial era, the earlier perception of the Frankish disease as originating with Europeans was reversed.

Some of the early treatments for the Frankish disease remained in limited local use, while others spread. A few, such as *Guaiacum* from Central America, *Smilax* or "China root", and mercury, became global remedies. Winterbottom concludes that medical cultures within and beyond the IOW shared some basic understanding of health as rooted in a balance of key bodily substances. The Frankish disease triggered changes in medical thinking in the IOW by associating concepts of contagion with specific groups of people.

Eric A. Strahorn, in his contribution to this volume, reassesses the debate about the early dispersion of leprosy in the IOW from the vantage point of fresh insights from palaeopathology. Bringing literary and archaeological evidence into dialogue with DNA sampling, Strahorn develops a pathology history of the progression of interpretations and transmission theories of "Hansen's disease", as leprosy is also called. The physical manifestations of lepromatous leprosy, the more severe of the two main types of Hansen's disease, which include extensive skin lesions, loss of extremities, and a collapse of the nose, may be identified in disease descriptions found in a number of ancient Chinese, Indian, Egyptian, and circum-Mediterranean texts. Such sources suggest the presence of leprosy in India by the second millennium BCE, and in ancient Egypt, the Roman

Mediterranean, and China by the third century BCE. Archaeological evidence supports this chronology. One influential modern hypothesis proposed that the armies of Alexander the Great carried leprosy from India to the Mediterranean. Critics, however, point to the particularly slow incubation period of *M. leprae* of three to ten years and suggest an alternative hypothesis—that leprosy travelled along age-old trade routes linking India and the Mediterranean. Strahorn considers that, since the diagnosis of leprosy in skeletal remains is complicated, and the majority of skeletons with leprosy-attributed bone damage have been found in Europe, DNA evidence has the potential to fill gaps in the literary and archaeological record. Nevertheless, the lack of aDNA samples from many parts of the IOW, notably India, still leaves many questions unanswered about the role of the IOW in the early dispersion history of leprosy.

In his chapter, James Francis Warren examines the health impacts of climate, weather, and colonialism in the Philippines from the sixteenth century. Consulting sources from the colonial and post-independence periods, Warren draws a picture of an archipelago afflicted by successive adversities. Natural disasters, such as typhoons and droughts, often triggered outbreaks of disease. Arid periods, especially during El Niño events, could lead to the drying up of wells and rivers with, as a result, limited and unsafe supplies of drinking water that often triggered the outbreak of disease. Again, floods, common during the rainy season, often precipitated health problems including coughs, fever and flu, water-borne diseases such as cholera, typhoid fever, amoebic dysentery, diarrhoea, and mosquito-borne diseases.

Cholera, which frequently erupted in the wake of typhoons and floods, was the most feared of water-borne diseases Seven major cholera outbreaks occurred in the Philippines during the nineteenth century, five of them linked to El Niño events. The Spanish colonial government relied heavily on the assistance of various Catholic religious orders and charitable donations for disaster relief and assistance. This proved insufficient, however, and what remained of the Spanish health system broke down under American rule from the late 1890s. In the post-colonial era, the Philippines have struggled with both ever accelerating population growth and the lack of adequate drinking water and sewage disposal systems. In poor rural and low-lying areas, as well as in urban agglomerations, cholera and typhoid remained major health threats. This was manifest, for instance, in the 1970s when the Philippines experienced a devastating series of typhoons and floods. Since then, rapid climate change, increased population density,

and a growth in monocrop cash crops at the expense of food crops, have intensified the incidence and impacts of natural misfortunes and thus the vulnerability of the Filipino people to epidemics.

In his contribution, Gwyn Campbell examines the onset and spread of malaria in nineteenth-century highland Madagascar. "Madagascar Fever" early earned a notorious reputation amongst European visitors to the island and can be identified with reasonable certainty as malaria. Campbell shows that in Madagascar, the distribution and impact of malaria was determined by a mixture of climate, geography, and human activities. The disease probably arrived in the island with the first permanent settlers from East Africa in the eighth or ninth century CE. These pioneers comprised mixed or separate groups of Bantu speakers, who had sickle cell immunity to malaria, and Austronesians, who possessed no natural defences to malaria. This favoured the survival in Madagascar of Bantu speakers or those of mixed Bantu-speaking and Austronesian heritage who were sicklers. Later Austronesian arrivals (the proto-Merina) migrated to the central plateau chiefly to escape the largely endemic fevers of the lowlands. Attempts by Europeans, following their incursion into the IOW, to found settlements in the Malagasy lowlands largely failed due to malaria to which they proved highly vulnerable. The central highlands, at an altitude of 1300 to 1700 metres, were reputedly malaria free, but the evidence is that there, too, malaria became endemic in the course of the nineteenth-century, epidemic outbreaks occurring probably as early as the 1820s. This change in malaria epidemiology was connected to changing climatic conditions, notably warmer weather that facilitated the survival of mosquitos at higher altitudes, and to forced labour (*fanompoana*) policies that induced unprecedented flows of people between the malarial lowlands and the traditionally parasite-free highlands. This resulted in high mortality rates amongst highlanders. Also, people fled the land in order to avoid exploitative forced labour recruitment. In so doing, they abandoned labour-intensive rice fields and the water channels that fed them, clay-brick pits, and alluvial gold diggings, which created numerous reserves of stagnant water that provided ideal breeding ground for the malaria vector *Anopheles funestus* that spread from the eastern lowlands into the highlands.

In their contributions to this volume, Peter Hynd and Manikarnika Dutta examine the relationships between disease in colonial India, the production, regulation, consumption, and taxation strategies of alcohol, and colonial governance. In analysing the impact of disease in the context of alcohol consumption and social behaviour, these authors reveal two

seemingly contradictory attitudes to alcohol which was, on the one hand, considered destructive of health, even a disease in its own right, and on the other was thought of as a remedy.

Focusing on Bombay Presidency, Peter Hynd explores the impact of the IOW disease environment, especially the major disease outbreaks of the late nineteenth century, on colonial governance. He examines official claims about the relationship between disease, alcohol consumption, and excise revenue, and evaluates these claims against mortality statistics. Scrutinizing recurring references to plague and other diseases in the official colonial excise records, Hynd demonstrates that, although disease did not have a serious impact on excise revenues in late nineteenth- and early twentieth-century India, excise department officials often invoked disease, especially in epidemic form, to justify striking increases or decreases in excise revenues. They argued, for example, that alcohol consumption decreased when people fled cities during disease outbreaks, such as the 1896–1897 plague epidemic that ravaged Bombay. The return of disease fugitives to the city after the crisis, and the widespread belief that liquor constituted a prophylactic against plague, probably led to increased alcohol consumption and thus a spike in excise revenues in 1897–1898. However, colonial excise agents were trapped between the imperative to enhance revenue and the increasingly critical voices of missionaries, temperance advocates, and nationalists, who denounced alcohol consumption. In colonial India, tax officials adopted a "maximum revenue from minimum consumption policy" to limit the drinking of alcohol, impose basic hygiene standards on distillers, and repress adulteration. Disease was a most welcome "explanation that worked" for the British Raj tax authorities to justify fluctuating excise revenues.

In her contribution, Manikarnika Dutta examines the impact of alcohol on the health and behaviour of sailors in nineteenth-century Calcutta. British authorities in India were worried about both the alcohol consumption of European sailors in Indian port cities and the low quality of local liquor. They were highly concerned about the drinking binges that sailors engaged in during shore leave and the "crimping system" whereby "crimps"—a particular term for a fraudster—enticed sailors to consume drugged liquor and subsequently tricked, mugged, and robbed their victims. Colonial officials started to investigate the quality of local liquor when reports proliferated of "treacherous" Indians tricking "innocent" European sailors into drinking "poisonous" liquor. Cheap, adulterated liquor was considered a health threat to white sailors and even envisaged

to be a cause of cholera. Regulating the quality of liquor and its production and consumption facilities was therefore of paramount importance to British authorities. The temperance movement also campaigned against liquor consumption and related unruly activities, while the installation and administration of sailors' homes in port cities also helped to counter crimping and excessive drinking. Dutta investigates the extent to which the colonial state's measures to protect the health of European mariners were informed by imperial encounters in the fields of medical intervention, race relations, environmentalism, and legal order.

In his chapter, Edward A. Alpers extrapolates from a specific focus on the recent chikungunya epidemic in the western IOW to comment more generally about diffusion mechanisms of epidemic diseases. The RNA virus that causes *chikungunya* (a word used by the Makonde of Northern Mozambique and Southwest Tanzania meaning "that which bends up"—referring to the sufferer's severe joint pain) originated in two virus lineages in Africa and, in the late nineteenth century, developed a genetically distinct Asian virus genotype. The 2004 outbreak of chikungunya affected 75 percent of the population of Lamu Island, Kenya. It is thought that direct exchange between humans and mosquitos (i.e. independent of animal intermediaries), and intensified air travel, caused a rapid transmission of the disease, both to more distant island groups in the Western Indian Ocean such as Mombasa, the Comoros, the Mascarene Islands, and the Seychelles, and to India where in 2005–2006 over a million people were affected across 13 states. Mutations in the various chikungunya virus strains enabled additional mosquito species, such as the Asian tiger mosquito (*Aedes albopictus*), to become effective chikungunya vectors. This adaptation led to the re-emergence and rapid spread of chikungunya in the IOW some 50 years after the first authoritative identification of the disease in Tanganyika.

Alpers explores the academic debates surrounding the origins and recent expansion of chikungunya, including the virus-vector-environment dynamics of its aptly called Indian Ocean Lineage. The chikungunya pandemic demonstrates that this arthropod-borne virus can adapt and invade new hosts through mutation. With this insight, Alpers also reconsiders the malaria epidemics on the Mascarenes in the 1860s and poses three main questions for future research. First, could the *Anopheles* mosquito vector of the disease have survived a lengthy boat voyage from mainland Africa to these mid-ocean isolates? Second, did a critical mass of human malaria carriers then travel to these islands, enabling already existing populations of

mosquitoes to become vectors and cause a malaria epidemic? And third, did a genetic mutation, similar to that which triggered the 2004–2007 chikungunya epidemic, transform existing mosquito populations into effective vectors for the *Plasmodium* parasite?

In her chapter, Karine Aasgaard Jansen examines the 2005–2007 chikungunya epidemic in Réunion, a French overseas department. As a social anthropologist, Jansen focuses on the social impact of this vector-borne disease in the context of local disease perceptions and the islanders' resistance to public health interventions. The Réunionese had never before experienced chikungunya, and many of them interpreted the disease in relation to the malaria outbreaks of the 1950s. The location of the breeding grounds of the *Aedes* mosquitos in the *jardin creole*—the garden adjoining, and much cherished by, traditional Réunionese households—as well as local people's experiences with previous public health interventions against vector-borne diseases, contributed to the popular stigmatization of chikungunya. Islanders viewed mosquitos as familiar entities in local gardens and French public health agents carrying out mosquito control as trespassers invading the intimacy of the private garden. Moreover, elderly Réunionese associated chikungunya public health interventions with their past experiences of governmental malaria control measures that targeted unsanitary households. This led to the stigmatization of chikungunya sufferers as people whose neglect of domestic hygiene resulted in the creation of mosquito breeding grounds. Stigmatization, in turn, led many Réunionese to reject the idea that chikungunya was a vector-borne disease. At the beginning of 2006, when the chikungunya epidemic peaked—over 25,000 new cases being registered in a single week—there was a widespread belief that the disease was the result of a medical or military experiment, the workings of a chemical plant, or even of a terrorist attack. This heightened public opposition to official health agents. Thus, Jansen argues, public health interventions may have contributed to an increase rather than a decrease in the impact of chikungunya on Réunion during the 2005–2007 epidemic.

In the final contribution to this volume, Eva-Maria Knoll draws an arc from a legendary historical malady to the current impact of malaria-causing parasites in the Republic of Maldives. This small archipelago, lying at the crossroads of the IOW maritime trade routes, has the world's highest rate of a care-intensive inherited blood disorder (beta-thalassaemia). With its current focus on genetic risk, public health discourse in the Maldives is turning the tropical paradise islands into a thalassaemia risk-alert social

world. It does not, however, offer a causative explanation for the archipelago's exceptional thalassaemia burden.

Knoll investigates European and Arab historical reports of the "Maldive fever", a malady that most scholars assume to have been malaria. These reports date back about six centuries, starting with Muslim traveller Ibn Battuta in the fourteenth century, followed by castaway François Pyrard de Laval, East India Company surgeon David Campbell, Archaeological Commissioner of Ceylon H.C.P. Bell, and, in the late nineteenth century, collector and explorer Carl Wilhelm Rosset. By the mid-twentieth century, the small Maldives island habitats were recognized as mixed infection zones where the local population had to struggle with three different malaria parasites, the health impacts of which reinforced historical eyewitness reports of inescapable and deadly fever. WHO malaria control activities started comparatively late, in the mid-1960s, but were highly successful. By the mid-1970s, the deadly malaria parasites had been eradicated from the islands and soon thereafter were also eliminated from the collective memory of the Maldivian population.

Disease Dispersion: Tracing Dynamic Biosocial Phenomena

This volume comprises contributions on disease dispersion and impact from a number of academic and scientific angles, including history, social and medical anthropology, archaeology, epidemiology, and palaeopathology. These show that disease-causing agents often took advantage of Anthropocene environmental transformations such as deforestation, monoculture, and irrigation systems, and that they adapted to new environmental circumstances.

Diseases are highly *dynamic biosocial phenomena*, which often makes it difficult to identify them retrospectively and trace them in the historical record (Cooter and Stein 2013; Cunningham 2002; Arrizabalaga 2002; Wilson 2000). DNA analysis demonstrates that some pathogens in the IOW have relatively stable genomes. This is the case, for example, with *Mycobacterium leprae* (which causes leprosy), which has changed little over at least the last 1000 years. Other pathogens, however, may alter considerably over time. Their biological dynamic allows diseases to emerge unexpectedly; to strike, retreat, and re-emerge; and to affect a specific locality, where they might become endemic, or expand in the form of epidemics and even pandemics. Evolutionary mutations enable diseases to change genetically, offering them the potential to transmute, move, and adapt to new environments and circumstances (Alpers, this volume).

Here, we consider diseases to be both "real entities" and "thought entities" (Cunningham 2002, 15)—both biomedical realities and the results of historically produced knowledge, analysed according to either naturalist-realist or historical-conceptualist perspectives (Wilson 2000). The naturalist-realist approach uses modern concepts of disease and biomedical diagnosis to examine what historian Piers Mitchell (2011) calls "social diagnosis" made by people in the past. Thus some scholars identify malaria with certain historical descriptions of "fever". By contrast, the historical-conceptualist approach acknowledges that a general understanding of the nature of a specific disease is embedded in the "thought collective" of a particular time and society (Fleck 1979). Aetiological and epistemological theorizing, by consequence, may change over time. Such changes in the descriptive and diagnostic categories of disease are sometimes difficult to discern (Strahorn, this volume).

Most contemporary medical historians adopt a mixed approach, combining the methodologies used in the humanities with those employed in biomedicine, molecular biology, and genetics, in order to shed new light on historic categories and descriptions. The chikungunya virus, for example, was identified scientifically in 1952 and thus distinguished from dengue and the broader fever category (Alpers, this volume). Since "fever" is a comprehensive disease category used extensively in historic descriptions, some aetiological confusion between dengue, chikungunya, malaria, and other variants of tropical fever characterizes the medical history of the IOW (Kuno 2015, Knoll, this volume). Genomic studies of tuberculosis (TB), by contrast, have expanded the TB category to the *Mycobacterium tuberculosis* complex (MTBC), comprising several species of the causative organism. Furthermore, they have shown that tuberculosis is much younger than hitherto assumed. Rather than representing a prehistoric disease originating from "out-of-Africa" migrations, tuberculosis dates back only 4000 to 4500 years (Green and Jones, this volume).

Sequencing ancient DNA (aDNA), retrieved from bones and dental remains, is the most recent development in the methodological toolkit for the reconstruction of human ailments and disease in history (Hagelberg et al. 2015; Strahorn, this volume). Though technically challenging, and of limited utility in the reconstruction of infectious and genetic disease history, aDNA results provide new kind of data. This invites, if not obliges,

a reassessment of previous historical data and resulting insights and arguments that were largely based on European text sources.

This volume invites readers to consider disease in history from both a life science and social science/humanities perspective. Such an approach takes pathogen and gene pools as well as disease perception and experience into account. However, the poor preservation of human skeletons, especially in the warm and humid climates of the IOW, imposes a serious limitation on the aDNA revolution. Thus Green and Jones (this volume) argue that no pathogen aDNA samples of any of the five major infectious diseases of the IOW—malaria, tuberculosis, leprosy, smallpox, and plague—have yet been retrieved from this macro-region. Finally, the disease history of the IOW also reveals a wide range of perceptions about disease aetiologies and epidemiologies. For example, some IOW communities believed that disease was linked to supernatural forces or blamed it on social "others"—such as Indian "coolies", immigrants, slaves, the crews of visiting ships, pilgrims, or colonizers.

In sum, this collection focuses on disease dispersion across time, space, and various cultural settings rather than on, for example, the large field of culture-specific diseases captured as culture-bound syndromes or folk illnesses. It will hopefully stimulate scholars to engage in other, interdisciplinary studies, of the biosocial dispersion of disease in the IOW, past and present.

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CHAPTER 8

European Sailors, Alcohol, and Cholera in Nineteenth-Century India

Manikarnika Dutta

Introduction

In early modern Europe, British sailors had the reputation of being rootless, often violent, promiscuous, and dipsomaniacs—an idiom for social evil (Conley 2009, 2). However, in the late eighteenth century, this image started to change as naval authorities, Christian missionaries, authors, and playwrights sought to project sailors as valiant founders of Britain's maritime empire. Subsequently, in Victorian and Edwardian Britain, sailors were increasingly portrayed as defenders of the nation and devoted family persons. American sailors were likewise presented sympathetically in accounts such as the 1852 annual report of the American Seamen's Friend Society, which stated that more than 70,000 sailors had pledged themselves

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to the temperance movement and nearly 50 Sailors' Homes had been established in port cities (Haughton 1855, 111). At the same time, the merchant and war navies of several countries supplied sailors with premium quality coffee and tea in an attempt to discourage them from drinking liquor. Naval authorities around the world floated the idea that abstinence improved the chance of surviving the ordeals of maritime voyages (Gustafson 1884, 97). However, in stark contrast to this generous characterization of the sailor in Europe, the British Indian government expressed great anxiety about the worsening situation of European sailors in Indian port cities. Its efforts to regulate the health and behaviour of sailors drew a fault line between imperial and colonial contexts of governance.

Alcohol was widely considered central to seamanship, to the extent of being a determinant of the collective behaviour of sailors. Drinking together was a means to befriend strangers, allay fears of perilous journeys, and generate a sense of bonhomie among crew members. The captain exercised authority over his crew through regulating their access to alcohol (Gray 2016). Consumption of liquor by discharged sailors at port cities at the end of a ship's journey was more problematic. In early nineteenth-century India, members of the British ruling elite were particularly embarrassed by their failure to adequately discipline drunken sailors (Tambe 2009). They were also concerned that local liquor, often found to be adulterated and of low quality, was pernicious enough to cause Europeans to become debased. Harald Fischer-Tiné has argued that colonial narratives blamed crime among sailors on the consumption of drugged liquor rather than pure European alcohol. He observed that the behaviour of these drunken 'white subalterns' embarrassed the colonial government, which considered sailors to be a potential threat to its policy of racial supremacy. The state was concerned that if the reckless behaviour of European sailors brought them closer to the 'uncivilized natives', its lofty ideals of the civilizational purity of race and class might implode (Fischer-Tiné 2012). This anxiety was visible in colonial policies regarding the health and welfare of seamen.

The works of Fischer-Tiné and David Arnold (1979) have explored the disreputable section of European settlers in India that the colonial state was unwilling to acknowledge as its own. Douglas Peers and Erica Wald have analysed the colonial state's efforts to maintain the health of European troops by regulating their intake of liquor. Peers (1998) contends that British military administrators were concerned at the high morbidly rate

among European troops of both diseases such as cholera, malaria, and plague—almost twice that of Indian sepoys—and of venereal diseases, which they associated with alcohol consumption. The authorities were initially reluctant to prohibit soldiers from venturing out of the barrack for sexual gratification, lest it led to an increase in alcoholism and homosexuality in the barracks. Later, Act XVIII of 1853 enabled commanding officers to prosecute unregistered liquor dealers who were previously untouched by military law (Peers 1999). Wald's (2012) analysis of military records reveals that, unable to control the alcohol consumption of soldiers, garrison authorities tried to restrict alcohol suppliers. They criticized the wives of soldiers who practised prostitution and procured alcohol illegally, and the arrack vendors in the vicinity of barracks who presumably sold adulterated liquor. The works of both Peers and Wald, in emphasizing the colonial state's need to preserve order and health among its soldiers, focused on the administrative strategies of limiting alcohol consumption within the military establishment.

Building on previous scholarship, this chapter explores the impact that adulteration of liquor and the 'crimping system', in a colonial setting, had on the health of white sailors who British authorities in India believed to be vulnerable to local influences such as cholera and cheap liquor. Additionally, liquor was envisaged as a cause of cholera at a time when aetiologists were uncertain about the exact nature of bacterial contamination leading to the disease. In the nineteenth century, port cities were known as disease entrepôts where disease pathogens could be spread both towards the port's hinterland by road and rail links and outwards through oceanic voyages. As Arnold (1991, 10) has argued, as an 'incubator of infectious disease' like cholera, Calcutta was at the focus of health hazard monitoring. Soldiers, sailors, pilgrims, and migrant labourers passing through the city were potential agents of disease dispersion. Although whether drinking or drunkenness constituted a disease was largely a matter of opinion rather than medical fact, a number of surgeons suspected a connection between liquor and cholera. The latter was still considered a disease that originated in a specific locality, that is, the lowlands of Bengal. The nature of cholera was so little known until at least the mid-nineteenth century that physicians could not reconcile various theories of cholera occurrence and naval surgeons were often unable to distinguish it from severe diarrhoea and dysentery (Preston 1895). Thus, consumption of liquor was a potential threat to public health that could escalate into a massively fatal but little understood epidemic.

Thus, this chapter argues that in British India sanitary regulation was inseparably linked to an anti-vice attitude in administrative policies. It begins with an analysis of the Government of Bengal's attempts to regulate liquor consumption among European sailors. Thereafter it examines the connected history of the fight of British colonial officials against adulteration of liquor, notably in Calcutta. Finally, it traces the adverse social effects of the medical problems of adulteration on the behaviour of sailors in port cities. The chapter aims to investigate the extent to which the colonial state's measures to protect the health of sailors were informed by imperial encounters in the fields of medical intervention, race relations, environmentalism, and legal order. Specifically, this study of adulteration and quality control of drinks takes into account David Arnold's (1993) emphasis on the linkages between colonial and British medical developments. Shula Marks (1997) has pertinently pointed out the necessity of considering what precisely constituted 'colonial' in the overlap between the colony and the metropole. She specifically points to the desire of British colonizers to uphold certain ideologies of governance and the differences they considered to separate the colonizer from the colonized. This forms the context here for an examination of the agency of colonial power in controlling the discourses of medicine, in which emphasis is placed upon the extent to which medicine in the colonial situation differed from the metropolitan context.

Prevention of Mortality and Regulation of Profligacy Among European Sailors

Colesworthy Grant (1813–1880), a British resident of Calcutta, wrote in 1850 that white sailors brought immense disrepute to the city. Their favourite haunt was Flag Street in Lalbazar, which was full of taverns run by Indians and Europeans from Italy, Spain, and Portugal. They would 'disappear for days in the nests and fastness of riot and profligacy which are numerous in that vicinity' (Barrett 2004, 384). In order to retrieve their sailors in time for the outward journey, captains of ships often had to visit gambling and drinking dens, sometimes accompanied by police constables. As part of a global campaign, the temperance movement in Britain inspired voluntary organizations in India, especially Christian associations, to discourage sailors from 'temptations'. When stories of drunk European and American sailors in Indian port cities circulated internationally, the British colonial state became active in disciplining them. The police were always on the lookout for 'houses of ill-repute' that sponged off visiting sailors. Since they did not expect abstinence from the sailors, they monitored the sale and consumption of liquor, particularly adulterated liquor, in an attempt to restrain and protect their health.

In November 1832, the magistrate fined Charles Neville, Richard Barrett, and Thomas Owen for running unlicensed public houses. On the same day, a police sergeant named Crawford broke into an illegal tavern maintained by a person named Lazarus. He had been spying on a particular den for months, but somehow Lazarus had been evading his search. Finally, when Lazarus was apprehended and brought before a court, he denied the accusation, saying that he was ill and had little idea when and how the sailors occupied his living room as if it were a tavern. He added that the sailors ordered food and drink without him noticing, and had no inkling about the bottles of alcohol that the sergeant had seen on the table. He was found guilty and fined 15 rupees (*John Bull*, 4 November 1832). Around the same time, about a dozen drunken sailors assaulted two Indian police constables opposite the Writers' Building. They wanted to take the rattan stick wielded by one of the constables and started beating the constables when they were refused. A third policeperson who came to his associates' rescue was beaten as well and later taken to the Native Hospital. The main perpetrator stumbled against a wall and fell in a drain as the sailors gambolled towards the river. He was detained until a European sergeant apprehended him. The other sailors were summoned to appear in court (*John Bull*, 25 November 1832). According to an observer (Sykes 1992, 47–48) writing in the 1850s:

> There is a great outcry in Calcutta, and for once a reasonable one, against the grog shops, and the danger of them to the British soldiers and sailors. The spirit is bad, and very cheap indeed, and they have indulged terribly in it. Some got drunk that their medals were robbed from them, and few have died of drink... A good many days ago, the Lieutenant-Governor [of Bengal] was told to enforce the act withdrawing licenses from those shops where people came out drunk, but now a better thing is being done by establishing a Government canteen on the Maidan in tents, where good spirits and tea and coffee and beer can be had, and skittles and games, and newspapers and books for amusement.

Dr. Hugh Macpherson (1861), Inspector General of Army Hospitals, noted that between 1856 and 1860, 716 European Protestants died in Calcutta from cholera. Of these, some 76 per cent were part of the city's floating population, mainly sailors. Macpherson remarked that ship captains and crews had identified certain anchor sites along the Hooghly River as more perilous than others. Colvin's Ghat, close to the mouth of a long sewer, was particularly notorious, as were Thompson's Ghat, Cooly Bazar, Fort Point, and Armenian Ghat. He concluded that, though none of these sites were perennial breeding grounds of diseases, disembarking sailors were in danger of catching cholera. A report in the *Saturday Review of Politics, Literature, Science and Art* (29 April 1865, 507) noted that 11 cholera victims were admitted to hospital from a house in Bow Bazar within a few weeks. It criticized the Calcutta Municipal Corporation as a body of 'wrong-headed people who muddle each other and do no earthly good for public'. The lack of progress in combating cholera is evident from a report in *The Lancet* (5 November 1887, 931–932) which blamed the high mortality among European sailors, around 11.1 per 1000 persons, on 'breathing [the city's] foul air, and partaking of drinks diluted not always with hydrant water'. It noted that the Jack Tar was expected to adapt to this unparalleled atrocious environment, portraying them as helpless victims of circumstances unique to the colony.

The Government of India was arguably more concerned with the health of European soldiers than sailors. The *First Annual Report of the Sanitary Commission for Bengal, 1864–1865* contained 55 pages on the need for improving the diet and barrack accommodation of troops, and the necessity of new hospitals for them, compared to only three pages on sailors. A *Friend of India* correspondent (1863) wrote that sailors were neglected in comparison with soldiers, whose achievements were celebrated vigorously, especially after 1857, in government blue books, newspapers, and pamphlets. Death and disease in the army were given more importance. Questioning the colonial government's policy, the newspaper correspondent asked:

> What does India do for the sailors who carry to and fro the wealth which enriches her? Nothing that can be appreciated. Calcutta, Bombay and Madras are all bad alike and all complain equally… choleraic drains, a life-destroying sun, drugged brandy, brothels exceeding in beastliness the pictures of juvenal, robbery under the name of discount and charge on bills and notes.

It was the combined effort of actors in Britain and India that finally made the colonial state ponder the need for greater medical attention for sailors. An address by Dr. Norman Chevers (1818–1886), Principal of Calcutta Medical College, to seamen at the Floating Library, Calcutta, on 5 January 1864, provided the initial impetus for the state to act. Lamenting the poor physical state of sailors who should be healthy and convivial, Chevers (1864) wrote, 'The British Seamen ought to be—and, when placed under favourable circumstances, is,—one of the healthiest of mankind'. While the state was aware of the high mortality rate among European sailors, it was only after the publication of Chevers' essay that it began taking measures to improve the living conditions of seafarers in Calcutta. State officials started maintaining registers for sailors and enacted new sanitary regulations on the basis of accumulated data. As part of its welfare programme, the Sanitary Commission recommended providing sailors with comfortable accommodation and amusement (Cave-Browne 1865, 466). Sanitation and hygiene in the old Sailors' Home, constructed in 1838 in Bow Bazar, had by then deteriorated.

A second impetus for state action was the cyclone of 5 October 1864 that destroyed many ships at the port of Calcutta, leaving 547 European sailors without occupation. This meant that, as 458 discharged sailors were already on shore, the port authorities had 1005 sailors to rehabilitate. Many of them landed, destitute and sick, and ended up in prisons and hospitals. By 23 January 1865, the authorities had provided for 563 sailors, sending home 163 at the expense of the Board of Trade and the Relief Fund and 187 on nominal wages, recruiting 30 for the Royal Navy in Bombay, and employing 183 others on full wages, chiefly in maritime activities (Annual Report on the Administration of the Bengal Presidency, 1864–1865, 102). Further, the Lt. Governor of Bengal asked the Sanitary Commission to investigate the state of sailors. In May 1866, Major G.B. Malleson (1866, 9) responded with a report titled 'The State of Sailors in Calcutta', raising concerns about their living conditions, health, and conduct. He quoted the comment by the Superintendent of the Reserve Force of Police that Calcutta port was overpopulated with sailors. Another report from Captain Alexander Caw, Shipping Master, showed that between 1 May 1864 and 30 April 1865, 629 ships with a total of approximately 17,298 sailors visited the port of Calcutta. Of this number, 3655 were discharged, 129 deserted, 214 were sent to prison and 232 to hospital, 231 died, and the rest were left without employment (Malleson 1866, 4).

Malleson (1866, 5) indicated that Caw wrote a letter to the Board of Trade on 30 June 1865 expressing concern about the Calcutta port turning into 'a sort of a depot' for seamen from other ports of British India such as Bombay and Rangoon; from Shanghai, Sydney, Melbourne, and Port Louis (Mauritius); and from English towns such as Shields and Tyne. This influx strained the ability of Bombay port authorities to employ and accommodate sailors. Consequently, they stipulated, in the 206th section of the Merchant Shipping Act, that captains should contact the Shipping Master before discharging any inbound sailor and that they could be prosecuted for mistreatment of employees should they ignore this ruling. Caw further stressed the need for the Board of Directors to prevent ships arriving from colonial ports such as Melbourne and Sydney, or ports in England, from discharging sailors in Calcutta unless those sailors possessed a contract guaranteeing their return passage. In support of his argument, he pointed out to the Master Attendant, J.G. Reddie, that on 12 July 1865 the number of jobless seamen in Bombay was 692, whereas the maximum the port could sustain was 500.

The surplus population of seamen further compounded their health and legal problems. Disease, suffering, and mortality among sailors were principally associated with poor eating habits, stale and contaminated air in tiny ship cabins, exposure to various unhealthy climatic conditions (Harrison 1999), and above all exposure to drunkenness and venereal diseases contracted from local prostitutes (Chevers 1864, 37). Chevers (46) stated that the Sailors' Home was 'surrounded with drinking shops of vilest description' and situated in the 'centre of about the worst atmosphere discoverable in this unsavoury city'. He probably meant a combination of insanitary living conditions and availability of 'vices' as the worst atmosphere. Chevers suggested the construction of a larger building in a 'healthier' and 'reputable' part of the city. Montague Massey (1918, 89), a civil servant, wrote that the Sailors' Home in the 1860s was a 'crying scandal', because it was situated in an area abounding with 'native grog-shops in which [shopkeepers] sold to the sailors most villainous, poisonous decoctions under various designations' and 'boarding houses run by a thieving set of low-caste American crimps'. Moreover, Lalbazar, the hub of watering holes and brothels, did not have a working sewage system. The drains were mostly open and full of black putrid slime that had accumulated over the years. British travellers usually disdained the marketplaces for their 'disgusting' appearance (Chakrabarty 1991).

Newspapers such as the *Saturday Review of Politics, Literature, Science and Art* and *The Friend of India* criticized the irregularity of sanitary supervision and failure to enforce sale of hygienic food in the port area. However, the threat of fatal diseases failed to deter sailors from spending time there, mainly for want of better options. Malleson (1866) indicated that conditions in the Flag Street neighbourhood would encourage the rapid circulation of epidemics and demanded constant care and vigilance by both the police and the municipality. In a letter dated 25 February 1864 (British Library IOR/P/433/52:1866), S.H. Robinson, Secretary of the Sailors' Home, requested Lt. Colonel H.C. James, Private Secretary to the Lieutenant-Governor of Bengal, to provide a new establishment in a better locality. He also expressed the need for a recreation ground for seamen, an area enclosed with a bamboo fence, resembling a cricket ground. In response, the government enclosed a part of the ***maidan*** (a vast field between the fort and the esplanade) for sailors to play cricket.

The existing Sailors' Home was later sold, and the proceeds were used to build a new house at 13 Strand Road under the 'special care' of Lord Henry Lawrence (1811–1879) (Firminger 1906, 163). It was situated in a better locality but accommodated fewer than the 200 men housed in the former building. Henceforth, avoidance of disease and other social evils often determined urban restructuring. Chevers (1864, 51) recommended the implementation of better drainage along Flag Street to improve sanitation as a necessary measure to protect sailors from diseases such as cholera and dysentery. He further advised that, at the start of each cholera season (which usually lasted from July to early October), the captain of every vessel should be given a set of regulations to prevent the occurrence of the disease and to cure those crew members it affected. The authorities also thought about accommodating homeless sailors in other boarding houses, which were to be carefully inspected for cleanliness in order to ensure proper living standards. Landlords were warned that they might have their licences revoked unless they resolved problems reported by residents. However, the infamous liquor addiction of sailors proved to be a greater concern for the colonial state, particularly as the quality of liquor was below standard and sometimes proved fatal.

Drunkenness and the Abuse of Adulterated Liquor

As the number of deaths from cholera among European sailors increased, the quality of drinking water came under scrutiny (Macpherson 1861). The authorities believed that, in the absence of clean water, the intake of unfiltered river water was responsible for the high cholera mortality rate (Chevers 1864, 41). However, European doctors considered that there existed aggravating factors other than insanitary living conditions or poor quality water—namely, environmental factors like the 1864 cyclone (Gastrell and Blandford 1865, 126). They also commonly believed that consumption of adulterated alcohol might have been responsible for the prevalence of cholera in certain localities, particularly around the port. The Medical College Hospital, situated near Flag Street in the centre of the city, admitted more than twice as many sailors as the Presidency General Hospital, located at the city's southern fringe. Chevers (1864) estimated that as many as 10 per cent of the sailors entering the Calcutta port every year died of cholera.

It could be argued that the colonial state's concern about adulteration was influenced by the metropolitan British attitude. The chemist Fredrick Accum (1769–1838) discussed in 1820 how some dealers adulterated food and drink with harmful substances. 'There is death in the pot', he wrote, as a preamble to the detailed exploration into how the poisonous extract of *cocculus indicus* (popularly known as the black extract) was mixed with malt liquors as a cheap way to increase the level of intoxication (Accum 1820). Sometimes a substance called multum that was comprised of gentian root, liquorice juice, and black extract was used (Accum 1820, 6). More dangerous was the practice of adulterating wine with lead to stop the process of acescence and maintain the transparency of white wine when it became turbid. Even a small amount of lead acted as slow poison, prompting Accum to castigate those responsible as murderers (Accum 1820, 102–103). The book aroused considerable public attention, and in 1851 Thomas Wakley (1795–1862), surgeon and editor of the medical weekly, *The Lancet*, and his colleagues started a campaign about the dangers of adulteration. They observed under a microscope foodstuff bought from different markets. This was followed by the establishment of the Analytical Sanitary Commission under the supervision of British physicians Arthur Hill Hassall (1817–1894) and Henry Letheby (1816–1876). Hassall examined about 25,000 food and drink samples between January 1851 and December 1854. The ensuing report, published in *The Lancet*,

emphasized many instances of death, poisoning, paralysis, or any illness caused by intake of adulterated drinks and attracted much attention (Morton 2005).

A Select Committee established in 1855 to enquire into the adulteration of food and drink advised that adulteration must be stopped in order to protect public health, honest merchants, consumers, and especially public morality—so as to prevent depreciation of 'the high commercial character of this country... both at home and in the eyes of foreign countries...' (Hassall 1861, 37–39). However, many producers and retailers claimed that adulteration was harmless and served consumers by keeping prices low (Otter 2006, 520). Nevertheless, in 1872 an amendment of the Adulteration of Food and Drink Act (1860) was passed, incorporating Hassall's proposal to appoint a public analyst, which resulted in the establishment two years later of the Society of Public Analysts. The 1875 Sale of Food and Drugs Act stipulated that manufacturers print a guarantee of purity on wrappers and packets alongside certificates obtained from public analysts (Morton 2005, 170–171). This measure was not adopted in British India until the twentieth century when the technology of ascertaining dilution levels first became available across the territory. As an essential aspect of sailor welfare, a pattern emerged in major administrative centres such as Calcutta, Bombay, and Madras of investigating allegations of adulteration and implementing crackdowns on public houses. In addition to concern for public health, checking the loss of revenue was a compelling reason for the government to control locally produced liquor. The East India Company monopolized the liquor trade in 1773 and subsequently generated huge revenue from the steep excise tax on alcohol. However, this chapter concentrates on the medical aspect of the state's intervention in liquor trade.

The threat of drunken sailors was not exclusive to Calcutta. In a letter to *The Mariners' Church Gospel Temperance Soldiers' and Sailors' Magazine* (December 1845, 542), G. Drago, Aqueduct Sergeant in Poona, reported preparing a petition for the government to order hotels and taverns to stop their entertainment programmes on Sundays. Sailors frequented these establishments and surrendered to the 'most vicious kinds to intemperance', creating 'disgusting and demoralizing scenes' that disgraced Britain. Michael Kirwan Joyce (1854, 1) of the Bombay Police noted that certain areas of the city, such as Dobee Tank and Duncan Road, had a high number of liquor shops, although these were dispersed rather than concentrated in one location, making it difficult for the police to raid. Many

of these shops employed discharged European soldiers as unwaged crimps or 'catchpoles'. These people earned their livelihood from the 'plunder of the unfortunate wretches', such as sailors. The liquor shops also employed abandoned women, nominally as domestic servants, but in reality as prostitutes who lured sailors into their establishments, called 'Tereerams', and there drugged them with adulterated liquor, and looted their belongings.

Joyce (1854, 3) argued that adulterated liquor was responsible for numerous deaths. Many licensed shops sold concoctions exclusively for sailors. Named 'Sailor Jack' or 'Tom's Brandy', such drinks were prepared from strong arrack mixed with the 'poisonous juice' of *datura*, extracted from tobacco and chillies and opium. The beer, priced at 50 paisa per bottle, was a mixture of beer, water, and a concoction of vinegar, soap nuts, sugar, and soda. The so-called wine, which cost a rupee a bottle, was a combination of vinegar, sugar, Parsee Brandy, and a decoction of logwood. The abundance of liquor shops where such products were sold compounded the problem. Of the 422 liquor shops, 3 described themselves as hotels and 13 as taverns. The rest comprised 172 retail shops and 234 toddy shops (Joyce, 4).

The debates and correspondence about adulterated liquor engendered a discourse about the corrupting influence of the Orient on western people. Some colonial officials reported a conspiracy by the 'deceitful', 'cruel', and 'dangerous' natives to induce innocent white sailors to get drunk so that they could steal their possession. This narrative recast white sailors as victims of colonized subjects whose crimes, including theft and murder, were often attributed less to a lack of morality than to the intake of pernicious Indian narcotics mixed in drinks. Some reports excused sailors as semi-educated men whose only escape from hard physical labour was to indulge in liquor.

Official concern over the quality of liquor sold at local grog shops grew with the increase in reports of 'treacherous' Indians tricking 'innocent' European sailors into drinking 'poisonous' liquor. Colonial officials increasingly felt that they had to contend not only with the traditional ill-discipline and disreputable behaviour of mariners but also their consumption of adulterated liquor. This led the government to investigate the quality of liquor sold in local markets. In general, it was made 'fiery hot' with red pepper and other 'tongue-rasping' and 'bowel-scorching abominations', and in some shops was found to have traces of several strong narcotic drugs such as *datura*, *cocculus indicus*, and *gunjah* (Chevers 1864, 41). Investigations into the sale of adulterated liquor to poor

Europeans, especially sailors and soldiers, in Lalbazar, Bow Bazaar, Rada Bazaar, and College Street, revealed that many shopkeepers used 'native' liquors such as *Mudut* and *Doasta*, described by Chevers as 'bazaar sharab', and falsely sold them in English bottles with labels such as 'Old Tom' and 'Exshaw's Brandy' (Chevers 1864, 65). Better quality liquors such as 'Exshaw's first class Brandy' were sold to Europeans at a higher than market rate. Chemical analysis of liquor in Calcutta in 1857–1858 found that it was diluted rather than drugged, and it was less its quality than the quantity of consumption that resulted in drunkenness and associated misdeeds.

According to Chevers (1864, 37), unadulterated liquor was so difficult to obtain in Calcutta that a 'sober man' (ostensibly a British sailor) could hardly ever find proper beer. In many cases, after drinking liquor purchased from local grog shops, sailors contracted cholera from which they suffered a higher mortality rate than others affected by the disease due to their overindulgence in poisonous liquor (Chevers 1886). Chevers argued that the mortality rate could be reduced by a third if alcohol consumption was properly regulated. He speculated that equal duty on rum and *doasta* would increase the sale of rum and advocated that all *doasta* in Calcutta should be distilled under strict surveillance. However, despite the bad publicity given to it, low-quality 'poisonous' liquor continued to be sold unabated. In his report, Malleson (1866, 2) mentioned a conversation with Dr. C. Fabre-Tonnerre, the municipal health officer in Calcutta, who told him that Magistrate Macleod Wylie had reduced the sale of 'noxious liquor' in the late 1850s by raiding public houses and withdrawing licences from those selling tainted liquor. Disease, destitution, and crime were thus intermingled in medical reports.

A number of prominent people, organizations, and publications supported the campaign against adulterated liquor. The *Indian Year-Book for 1862* (Murdoch 1863, 117) applauded the newspaper *The Friend of India* for doing 'good service by directing attention to the alarming increase in the consumption of spirits'. It quoted the following from the newspaper:

> As if it were not tough that drunkenness should be the national crime of the English at home, and should only too unmistakeably characterise her sailors and lower classes abroad, it would seem as though the Government of India were determined to make their heathen subjects and their own soldiers as bad as the people of the mother country [...]

Since it proved difficult to dissuade European sailors from drinking, alternative steps were taken to ensure 'none but pure alcohol would be sold'. Additionally, the civic authorities tried to reduce the number of shops that sold the cheapest liquor on the grounds that 'sober men' should be provided the opportunity to buy 'well-made coffee', 'good soda water', 'ginger beer', and 'lemonade' at proper rates (Chevers 1864, 51). In a letter dated 13 June 1864, Chevers recommended appointing a competent official to inspect every ship arriving at the port and install special taps to sell good quality spirits, wine, beer, and other drinks to sailors.

Borradaile, Schiller, and Co., a major partner in the Port Canning Land Investment, Reclamation, and Dock Company in Calcutta, suggested sellers of 'that most intoxicating drink, the Indian Rum and Doasta' be banned and pay heavy penalties. They supported the reduction of duty on European drinks to make their price competitive in the local market. The municipality adopted Tonnerre's suggestion of registering all seamen on arrival in the Calcutta port and obtaining from the captain of each vessel a list of the number and cause of casualties on board (Malleson 1866, 3). The government closely monitored inquiries made by Chevers and other officers about 'unwholesome food and drink'. Chevers' report in particular drew much attention and led to appropriate measures for the first time. Following its publication in 1864, a special committee was appointed to assist health officers to systematically inspect food and drink sold in the public markets and confiscate 'unwholesome articles' (Chevers 1864, 63). However, such measures were not uniformly successful. Authorities in Bombay were more effective in regulating the production and sale of liquor, as Peter Hynd's chapter in this volume shows. He argues that in the late nineteenth century, the Bombay excise officials were not driven by the perceived public health benefits of regulation of liquor; rather, they were keener on imposing basic hygiene upon distillers and reducing instances of liquor-induced public disorder. Therefore, the emphasis on health and sanitation seems to be Bengal specific, probably due to the prevalent notion that cholera was a disease of locality and could not have emerged in full virulence other than in the Bengal delta.

Crime and Crimps in Sailor Dens

Surgeons in the Indian Medical Service suggested that consumers of intoxicants containing certain narcotics were more likely than others to commit a crime. It was commonly believed that respite from long and

difficult sea voyages caused sailors to abandon every pretence of discipline, drink with abandon, and act insubordinately. The police frequently received complaints about sailors' drunkenness, assault, theft, refusal to work, absence without leave, inability to pay fines, suspicious loitering, rioting, and indecency (Cave-Browne 1865, 462–463). Several decades of court records indicate that many of the crimes committed by sailors were perpetrated under influence of adulterated liquor sold in the markets. There were also complaints that the government had not done enough to stop such illegal activities. Juries in Calcutta regularly pleaded to the court to prohibit this 'evil' on the rise in the streets and markets of the city. Nevertheless, despite the power invested in it, the first Sanitary Commission accomplished less than was anticipated (*The Friend of India*, 2 June 1864).

Some Europeans in Calcutta also emphasized that it was wrong to criticize all mariners as a 'drunken, reckless, mutinous lot', since it was the difficult circumstances of seafaring that drove them into disreputable activity (Cave-Browne 1865, 453). It is hard to determine the actual number of 'criminal' sailors, as police records are sparse, and numbers were often inflated. Malleson (1866) referred to a lock-up register that contained 365 instances of sailor drunkenness and confinement. Of these, 186 comprised mariners living ashore—nearly 3 per cent of the off-duty sailors. Many among them were charged with assault. However, only 35 were committed to the Sailors' Home for corrective measures. Repeated complaints about sailor behaviour maligned them in the eyes of some, but also sparked attempts by others to understand and redress their problems (Cave-Browne 1865, 462–464).

Additionally, Flag Street, the residential neighbourhood of sailors, facilitated contact with local street women and prostitutes for whom Chevers (1864, 65) recommended the construction of licensed and regulated lock hospitals—establishments that specialized in treating sexually transmitted diseases. Many argued that sailors could hardly avert the temptations offered in Flag Street, the Sailor's Home, or any boarding house in disreputable neighbourhoods. They needed some amusement to keep themselves busy. For example, Seamen's Chaplain A.L. Mitchell advocated the creation of an institute where sailors might socialize; entertain themselves with a large bowling alley, chess, and draughts; and drink tea, coffee, 'good' soda water, ginger beer, and lemonade at proper rate (Malleson 1866, x–xiii). His dream was realized when the Seamen's Reading and Coffee Rooms were opened under the auspices of the Methodist Church at 19 Lall Bazar in 1874. The institution conducted a religious service

every night for seamen visiting Calcutta and maintained a coffee room that offered excellent refreshment at low prices and a reading hall with newspapers, magazines, and about 500 books gifted by 'friends of the institution'. The main agents of the institution were a group of women, including Mrs. Meik, Mrs. Conklin, and Mrs. Henderson, who visited grog shops and invited the seamen to the services (*Annual Report of the Missionary Society of the Methodist Episcopal Church* [henceforth ARMSMEC] 1892, 222). Rev. Frank W. Warne quoted George Henderson, the person responsible for the coffee rooms and seamen's work, saying in 1891 that sailors regularly attended and greatly appreciated the refreshment rooms at the mission (ARMSMEC 1891, 202).

The impetus behind opening the Sailors' Home in 1837, which fell into disrepute soon afterwards, was to 'suppress crimping and all the evils arising from it to which owners, commanders, officers and crews are subject in the port of Calcutta' (*Madras Missionary Register*, January 1838, 122–123). Sailors' Home in Bombay and Madras came up later in the same year. Mitchell gave a vivid description of how crimps operated in Calcutta in his narration of destitute white sailors. The 'crimping system' was practised widely on board ships, in Flag street, and in the vicinity of the shipping office. The members of the gang of crimps called themselves 'runners'. He accused them of being 'harpies' who enticed sailors to consume drugged liquor. The sources of Mitchell's information were convalescent sailors, who presented themselves as helpless, 'unfortunate victims' of crimping, as 'dupes of conspiracy' (Malleson 1866, xiii). These despondent sailors wandered around the city, often ending up in prisons. The problem persisted throughout the nineteenth century and, according to an observer, resulted in the sailors' debilitation 'from the effects of methylated spirits administered under the name of gin or whiskey, etc., by opium, or cocculus indicus in the name of beer, and in addition, by some loathsome complaint that may and probably will incapacitate him after a day or two at sea' (*The Nautical Magazine*, June 1871, 385).

Crimps were reported to be ubiquitous in the sailors' quarters of London—boarding houses, tap rooms of public houses, long rooms of gin palaces, and brothels—and to have ruined many a maritime career. An anonymous writer to the *Sailors' Magazine* (A Captain and His Mate 1845, 139–141) blamed sailors for landing themselves in traps they were well aware of and ship owners and captains for insensitively driving sailors towards destitution. The propensity to relax in the port city after too much hard work and lack of recreation aboard vessels led to drinking binges. An

inebriated sailor could be easily persuaded to turn to crime and a 'libertine' life. The writer refers to 'foes' that drugged the drinks of seamen who risked becoming addicted and 'enslaved' to the 'virile' poison. By the time the sailor had recovered from his drug-induced reverie, these crimps would have disappeared with his belongings—clothes and money. Subsequently, unable to afford meals and accommodation, the sailor would have no choice but to depend on the same or another crimp for credit.

The crimps, masquerading as employment agents, carried placards reading 'able seamen wanted'. They advanced sailors credit for exorbitantly priced accommodation and clothes to be paid from any future earnings. Again, unscrupulous ship owners gave commissions to crimps who could provide mariner labour cheaply at short notice. A 'drugged', 'stupefied', distressed sailor was in no position to bargain for a proper wage. As one ship captain reported to the *Sailors' Magazine* (October 1844, 116–119), 'thus is the most noble and most generous of Britain's sons duped, before he sets his foot ashore'. The effectiveness of the Sailors' Home and the judiciary in protecting seamen from crimps was continually questioned. *The Calcutta Christian Observer* (September 1841, 590–591) wrote that the Calcutta Seamen's Friend Society should strive harder to uproot the crimping system. It argued that a comparison between the numbers of sailors provided jobs by the Sailors' Home and crimps, or sailors finding their way to the Home and to crimps, would illustrate the success of sailors' welfare measures. It suggested to the managers of the Home to seek police cooperation to dispose of crimps, ask shipping lines to give jobs only to seamen sent by them, and employ a number of agents for visiting ships on their arrival and shepherding sailors to the Home. Evidently, some people, Christian missionaries in particular, considered crimping a threat to the physical and moral wellbeing of sailors.

Conclusion

This chapter examines the significance of the social and moral concern for seamen visiting Calcutta to colonial medical and sanitary policy. It discusses the issue of the propensity for drunkenness among sailors as manifested in its impact on their health and demeanour. The efforts to alleviate cholera and adulteration of liquor provide important insights into the ambivalence of the early colonial administration in India. The colonial state devised policies in response to the threat Indian society was considered to pose to white sailors, who were variously labelled as disruptive and

mostly vulnerable, and thus destabilized the carefully constructed idea of imperial British identity consisting in righteousness and discipline. The transfer of medical knowledge from Britain to India, and its translation into public policy, formed a salient feature of imperial formation, designed to protect the racial superiority and honour of colonizers from the threats posed by both white subalterns and Indians.

Despite similarities in the concern for seamen's health in British and Indian ports, the methods of addressing them were not uniform. Europeans tended to describe their sailors outside Europe sympathetically, usually as innocent and sober men struggling with a challenging environment. Thus Calcutta was described as an unhygienic port city whose 'natives' were responsible for many of the problems experienced by visiting sailors. The Europeans and Americans who made up the bulk of the city's crimp population and tavern owners were not criticized as vigorously as were Indians who sold poor quality liquor. Indian crimps were portrayed as conspirators who tricked 'innocent' European sailors into consuming low-quality liquor, sold often in bottles bearing labels of English brands that caught them unawares. Despite crimps across continents operating in similar fashions, British commentaries on Indian crimps suggest a racial differentiation. In sum, this chapter explores the living conditions of European sailors visiting India and the changing image of such sailors in the public and official mind.

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CHAPTER 9

Chikungunya and Epidemic Disease in the Indian Ocean World

Edward A. Alpers

Introduction

Twenty-five years ago, David Arnold (1991) published a pioneering article on the medical history of the Indian Ocean. In his wide-ranging essay, he emphasized the impact of European intrusion into the Indian Ocean world (IOW) after 1500, noting especially that "the emergence of India as the lynch pin of British power and trade in the East was of great epidemiological significance for the rest of the region and indeed the wider world beyond" (ibid., 7). Arnold particularly acknowledged the importance of maritime movement, whether by traders, soldiers, pilgrims or migrant labourers, as a central element in disease dispersion—what he called "epidemiological routes and conjunctures"—during the post-contact period (ibid., 9). More recently, Amina Issa (2006) has built on Arnold's essay by arguing for the significance of indigenous sailing ships and the diffusion of epidemic disease in Indian Ocean ports in the nineteenth century before the advent of steamships. In this chapter, I propose to jump forward in

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time to the contemporary period by examining the chikungunya epidemic that swept across the IOW in the first decade of the twenty-first century, and to ask whether it is possible to apply what we have learned from studying the origins and spread of this disease to improving our understanding of earlier epidemic diseases in the IOW.

Although my background is not in the history of medicine, I have long been familiar with the remarkable work of Dr James Christie, who served as physician to the Sultan of Zanzibar for a decade from 1865 (Anon. 1892). My primary interest in his account of cholera epidemics in East Africa (Christie 1876) was how vividly they revealed caravan trade routes linking the Swahili coast and the Nile Valley to the interior. In his very first contribution on cholera in East Africa, Christie observed: "In all the turnings and windings of the cholera epidemic, there seems to have been one unvarying principle directing its course. It has invariably accompanied trade wherever its direction may have been" (Christie 1871, 115).[1] Here, however, I look more carefully at Christie's medical work as an epidemiologist. This re-examination prompts me to use it as a springboard for thinking about disease diffusion in general and the difficulty of identifying diseases from the historical record.

Chikungunya as a Way to Think About Epidemic Disease

So where does chikungunya enter this picture, and how can an analysis of this latest example of epidemic disease help historians to think about earlier examples of disease dispersion in the Indian Ocean world? As I hope to demonstrate, it is the genetic ability of biological organisms to adapt to changing circumstances—whether natural or anthropogenic—through mutation that is the key lesson from the history of chikungunya. The chikungunya virus was not firmly identified scientifically until an outbreak occurred in 1952, in the southeast of present-day mainland Tanzania, when strains of a new virus were identified from the sera of afflicted patients (Robinson 1955; Ross 1956). This newly identified virus was given the local Kimakonde name "chikungunya", meaning "that which bends up", because of the clinical symptoms of "the severe arthralgia

[1] For modern citations of Christie's work, see Hartwig (1975, 63), Koponen (1988, 159–162, 173–176), Kjekshus (1996, 23–24) and Rockel (2006, 132), and the critical commentary by Echenberg (2011, 53).

[joint pain] that is a hallmark of chikungunya fever, the disease caused by the virus" (Weaver and Lecuit 2015, 1231). The chikungunya virus (CHIKV) is an RNA virus that belongs to the family *Togaviridae*, genus *Alphavirus*, and is part of the Semliki Forest virus antigenic complex (Sergon et al. 2007, 1189). There are three major genotype lineages of CHIKV: East/Central/South African (ECSA), the original Tanzanian strain; West African and Asian. Identification of the new virus represented a major breakthrough in the analysis of mosquito-borne tropical fevers since it was initially described as a form of "dengue" fever, a designation that for many years had served as a broad lumping category for a wide variety of clinically similar diseases. When Donald Carey began to look into the fever's history in the early 1970s, he discovered that what had in many instances been called "dengue" may in fact have been chikungunya. Most interestingly, exploring the history of chikungunya leads us back to James Christie, who, as Carey comments, penned "an important and fascinating epidemiologic account of dengue" in 1881 (Carey 1971, 255).[2]

After he left Zanzibar in 1874 and returned to Glasgow to practice medicine and publish his *magnum opus* on cholera, Christie continued to draw upon his East African experiences to indulge his interest in tropical epidemic diseases. His paper "On Epidemics of Dengue Fever: Their Diffusion and Etiology" (Christie 1881), cited by Carey, was a product of that continuing curiosity, but Carey overlooked a paper that Christie wrote a decade earlier while still based in Zanzibar, which, like his writings on cholera, was based on his exceptional powers of observation. Christie reported that in July 1870, "after the complete disappearance of cholera from the island of Zanzibar, a new form of fever, quite unknown to the bulk of the population, was epidemic in the island of Zanzibar, and more especially in the town, where it attacked almost the entire population" (Christie 1872, 21). In 1871, Christie recognized its similarity to descriptions of dengue by other physicians in other locations, but he also saw that this fever had certain different characteristics. When he returned to this subject a decade later, he explained that "the older inhabitants recognized the disease as one which had been epidemic about forty-eight or forty-nine years before, and they gave to it its former designation—Ki-dinga Pepo" (Christie 1881, 165; also quoted by Carey 1971, 255), meaning "cramp-like pains, caused by an evil spirit" (Christie 1872, 22). Christie

[2] Carey had studied endemic dengue in south India in the 1960s; when he published this article, he had a position at the Rockefeller Foundation of the University of Ibadan, Nigeria.

made several astute observations that have been confirmed by methods unknown in the nineteenth century. First, he observed: "Regarding the epidemic of 1870, I may state that it was not introduced to Zanzibar from without. The disease appeared at the height of the south-west monsoon, and for at least three months before, dhow communication from the north was impossible" (Christie 1881, 172). In view of his understanding of the spread of cholera, it should come as no surprise that he also noted: "With regard to the diffusion of the epidemic from Zanzibar to the mainland, I can only state generally that it took place along the lines of human intercourse, and that the coast towns were infected" (ibid., 166). What I find most remarkable, however, is Christie's hypothesis concerning the aetiology of *kidinga pepo*, which he speculated might possibly have been linked to a transformed version of cholera. "If chemical changes, of such a nature [human decomposition], take place in the *cadaver* [*sic*], may not physiological or pathological changes also take place? The hypothesis is not unscientific". Inspired by the work of Charles Darwin, he wondered further: "If the germ theory of diseases be admitted, the possibility of hybridization must be admitted also" (ibid., 174–175). Since Christie's conditional statements were penned before the great discoveries in virology, at a time when the miasma theory of contagious diseases was still dominant, this is all the more remarkable (Karamanou et al. 2012). The history of this century's chikungunya pandemic appears to bear him out.

What led me to take chikungunya as a starting point for thinking about the keynote presentation on which this chapter is based was the combination of the terrible impact it had on La Réunion in 2005–2006, where more than a third of the total population was affected; the disease's incredibly rapid and extensive diffusion across the IOW in the last decade and the ability of medical science, armed with the tools of genetic analysis, to track its aetiology definitively (see Her et al. 2009, 1165–1166). At the outbreak of the chikungunya epidemic, when in 2006 an editorial in a major French medical journal admitted that the chikungunya epidemic was completely "unexpected", virtually no scientific attention was being paid to this arbovirus. "Up to then unknown to both the public and to most physicians, a virus with an unpronounceable name, Chikungunya (CHIK), has invaded French news for several months" (Simon et al. 2006, 437).[3]

[3] See also early reports in Paganin et al. (2006); Pierre et al. (2006); Josseran et al. (2006).

Even at this early stage in the medical alert, it was recognized that the origin of the virus was in East Africa, that it had spread to the Comoro Islands before reaching Réunion and that "the diffusion among the islands had been promoted by the intensity of their human exchanges and their proximity (from several hours to a few days by boat)" (Simon et al. 2006, 439). What makes the history of the chikungunya epidemic so remarkable, however, are the genetic changes in the virus itself that enabled chikungunya to wreak such devastation on Réunion and eventually in India, where at least 1.4 million cases were recorded during the same 2005–2006 epidemic.[4]

It is now firmly established that CHIKV originated in Africa and that there exist two lineages of the virus, one West African and the other East/Central/South African [ECSA] (Burt et al. 2012, 662). There is also a genetically distinct Asian genotype that derived from ECSA in the late nineteenth or first half of the twentieth century (Weaver and Forrester 2015, 35). According to Felicity Burt et al., "In Africa, the virus is maintained in a sylvatic transmission cycle" between *Aedes* mosquitoes, which are "the main vectors", and small primates, mostly monkeys, although birds, cattle and rodents are also hosts (as distinct from dengue [DENV], where only primates act as hosts). Among mosquitoes, the most widespread vector is *A. aegypti*. An important distinction, however, is that CHIKV may be exchanged directly between humans and mosquitoes without animal intermediaries during epidemics (Burt et al. 2012, 662). Kamran Khan et al. (2014, 3, 15) state that humans infected by the virus soon become viremic and can transmit the virus directly to these insect vectors. In May 2004, a virulent outbreak occurred on Lamu Island, Kenya, where it had never previously been recognized. A serosurvey revealed an attack rate of 75 per cent in a total population of about 18,000 (Sergon et al. 2008, 335). The epidemic also struck Mombasa and by January 2005 had reached Ngazidja (Grande Comore), where, through 5 May 2005, 202 cases were reported. The same research team that investigated the Lamu outbreak was asked by the Comorian authorities to conduct a seroprevalence study on Ngazidja, where they identified a 63 per cent infection or seropositivity rate (Sergon et al. 2007, 1191). Taken together, these surveys suggest that many thousands of individuals on both Lamu and Ngazidja were infected by chikungunya.

[4] For an early report on the Indian epidemic, see Lahariya and Pradhan (2006).

Although it is not surprising that CHIKV spread from coastal Kenya to the Comoros in view of the long and intimate history connecting Ngazidja to Unguja, the main island of Zanzibar, it is noteworthy that the epidemic did not register significantly on Zanzibar. This may reflect limited research on Zanzibar, as in all of Tanzania, or the clinical confusion of CHIKV with malaria (Kajeguka et al. 2016),[5] but a small serosurvey of dengue (DENV) at Chake Chake Hospital on Pemba Island indicated that, while 15.4 per cent of patients tested revealed seroprevalence for dengue, none evidenced chikungunya (Vairo et al. 2012, e45). Without yet understanding the island-hopping nature of the CHIKV from coastal Kenya directly to Ngazidja, and having no direct evidence, it seems probable that the virus was transmitted by human carriers flying on the regular Kenya Airways flight from Mombasa to Moroni, the capital of Ngazidja. From Ngazidja, the virus spread to Mayotte, Mauritius, the Seychelles and Réunion, where, as we shall see, it had a major impact, precipitating a flurry of scientific research both to determine the cause of the outbreak and to recommend preventive measures, a topic addressed in this volume in the chapter by Karine Aasgaard Jansen and elsewhere (Jansen 2016). Indeed, increased air travel is one important factor generally recognized in the literature in promoting the rapid and widespread distribution of CHIKV (Renault et al. 2012; Morrison et al. 2016).[6]

The fundamental question is what precipitated the CHIKV to re-emerge and cause such damage after decades in retreat. For although there were outbreaks of the Asian strain of chikungunya in Southeast Asia at the end of the twentieth and beginning of the twenty-first centuries, the virus has been reported only occasionally in Africa since the 1960s (Lam et al. 2001; Laras et al. 2005; Burt et al. 2012, 662–663). Here the answer lies in a single genetic mutation in CHIKV. Genetic analysis was based on viral isolates taken from 127 patients from all the affected southwest Indian Ocean islands, plus Madagascar. The authors of this important study (Schuffenecker et al. 2006) traced the cause of the epidemic to evolution in the East African strain. Specifically, they discovered that "the emergence of genotype E1-226V, which was observed from the beginning of September 2005 and experienced a spectacular rise in frequency…preceded the explosive epidemic peak of mid-December 2005". This suggested to them "that such a mutation provides a selective advantage to the virus in mosquitoes". When they published their report in July 2006, they considered that the epidemic began in the Comoros, since the first verified cases reported in March 2005 on Réunion were linked to travellers from those islands, undoubtedly from Mayotte, which, like Réunion, is an overseas French territory (ibid., 1059). However, a subsequent study suggested that the ultimate source of the virus could be located in coastal Kenya, since the genetic footprint was the same as in Réunion (Njenga et al. 2008, 2757). Its authors also surmised that further mutations may have occurred and exacerbated the epidemic, a scenario at which Schuffenecker and her colleagues also hinted.

[5] Kajeguka et al. (2016, 7/9) conclude: "Chikungunya virus appears to be actively circulating in the population", which is not surprising given its endemic character in Africa.

[6] More generally, Siddhartha Mukherjee (2016, 33) notes that "viruses prefer to travel these days—on transcontinental airplanes".

Yet, despite its important contribution, the authors did not fully recognize what it was, specifically, about this mutation that rendered it so effective in spreading CHIKV on Réunion. This discovery was made by a research team from the University of Texas Medical Branch at Galveston, which discovered

> that a single nucleotide change, which arose during the epidemic, significantly increases fitness of the virus for *Ae. albopictus* mosquitoes and was associated with CHIKV dependence on cholesterol in the mosquito cell membrane. This change likely enhanced CHIKV transmission by an atypical vector and contributed to the maintenance and scale of the epidemic. (Tsetsarkin et al. 2007, 1896)

The key here is that, whereas the vector for CHIKV in East Africa and the Comoros was the familiar *A. aegypti*, which was absent on Réunion, "an E1-226V mutation in CHIKV results in increased fitness of CHIKV in *Ae. albopictus* mosquitoes", the much hardier Asian tiger mosquito (so-called because of the stripes on its middle body section), which was endemic on that island (ibid., 1900). By itself, it appears that "a single mutation is sufficient to modify viral infectivity for a specific vector species and as a consequence, can fuel an epidemic in a region that lacks the typical vector" (ibid., 1901). Furthermore, the team argue that, prior to this mutation, the level of CHIKV viruses in the blood was sufficient to indicate *A. albopictus* mosquitoes as the source (ibid.). In other words, when the initial Comorian travellers entered Réunion carrying CHIKV, they served as a human reservoir that, once bitten, was able to transform the hitherto unaffected *A. albopictus* into a vector for CHIKV. In addition, the team

contends, this mutation gave the Asian tiger mosquito a selective advantage over the African mosquito that was the fever's primary vector in Africa. Accordingly, once this rapid adaptation occurred in *A. albopictus*, it accelerated CHIKV so that it rapidly infected what was an entirely unprotected human population (ibid., 2903).[7] One particular advantage of the *A. albopictus* vector is its great adaptability in an urban environment, where it had evolved in Asia (Pialoux et al. 2006, 255).[8] In the end, the attack rate on Réunion was somewhere between 34.3 per cent and 38.2 per cent, depending on the mode of analysis, in a population of more than 700,000, yielding estimates ranging from 244,000 to 266,000 to as high as 300,000 cases of chikungunya. Moreover, for the first time ever, some 203 deaths were reported that were attributed to the virus (Renault et al. 2007; Pialoux et al. 2007; Gérardin et al. 2008).

Nevertheless, a few years after the chikungunya outbreak, Njenga et al. (2008, 2758) lamented that there was still no satisfactory explanation for the cause of such a pandemic. Notwithstanding the identification of the E1-226V mutation and its ability to transform *A. albopictus* into a vector for CHIKV, it was evident that the major changes in CHIKV had occurred before its earliest known appearance on Lamu in 2004. Moreover, while prior outbreaks in Africa were caused by strains within the same ECSA genotype, none were apparently ancestors of these new strains, so that their origins might have been any of a number of existing strains in either Africa or Asia (ibid., 2579). A year later, however, impelled by the extension of the pandemic to India, where it affected 13 states and at a minimum more than a million people, by applying several different modelling systems, researchers from the National Institute of Virology in Pune, Maharashtra, had succeeded in identifying a Ugandan strain that they could date to about 2000 (Cherian et al. 2009). Thus, within a few years of this completely unanticipated CHIKV epidemic, which had spread across the entire IOW, the Singapore-based authors of a modelled network for this historical process concluded that "current knowledge underscores the complexity of the vector-virus-environment interactions, and clearly demonstrates their role in changing the infectious disease epidemiology" (Ng and Hapuarachchige 2010, 882). Even though in 2013 much was yet to be discovered about how the E1-226V variant emerged,

[7] For a simplified synopsis, see Anon. (2007).

[8] For a recent example of how this mosquito and this disease have invaded a new urban territory, see Kampango and Abílio (2016), Gudo et al. (2015).

the authors of a validating follow-up report emphasize the broader point that arboviruses have the ability to invade a new host as a result of the process of genome replication (Arias-Goeta et al. 2013, 1). I should note, as well, that the structure of the Asian strain prevents it from acquiring the E1-226V variant unless another substitution, E1-T98A, is also present (Weaver and Forrester 2015, 35, Box 1). It is appropriate that the genomic strain created by the E1-226V mutation identified by this global research into the CHIKV pandemic has been named the Indian Ocean Lineage (IOL).

A further complication to the history of CHIKV is that its main vector in the south Indian outbreak of 2005–2006 was not the Asian tiger mosquito, but *A. aegypti*; nor was the strain of the virus the older Asian genotype, but the ECSA strain (isolated at Yawat, Maharashtra) that we now know carried the E1-226V genotype associated with the radical transformation of the virus first observed at Lamu and Mombasa (Yergolkar et al. 2006). Aggravating the situation in India, however, was a "second-step mutation" that appears to have made *A. albopictus* an even more efficient vector for CHIKV in other parts of India, where between 2006 and 2011, CHIKV infection spread to 19 states and affected as many as several million individuals (Tsetsarkin and Weaver 2011, 1). According to the Galveston group of medical scientists, they suggest that a "novel substitution, E2-L210Q, identified in Kerala, India in 2009, caused a significant increase in the ability of CHIKV to infect and develop a disseminated infection in *A. albopictus*" (ibid., 2, Author Summary). In fact, subsequent research by this same team indicates that this substitution is only one of four such mutations that cause even greater increases in infectivity (Tsetsarkin et al. 2016, Table 1). Stated in more technical terms, they find that

> adaptation of CHIKV to a new mosquito vector can be a multistep process that, since 2005, has involved at least 2 amino acid substitutions in the envelope glycoproteins. The substitution that provides the strongest selective advantage, E1-226V, was followed by second adaptive mutation (E2-L210Q) that has resulted in a strain circulating in India with the fittest phenotype detected yet for transmission by *A. albopictus*. (ibid., 11)

Further evidence of the remarkable ability of CHIKV to adapt rapidly to new circumstances may be found in the identification of an entirely novel CHIKV outbreak strain among 19 mutations in India that did not

contain the E1-A226V mutation (Kumar et al. 2014). The authors of a subsequent study into this phenomenon write that

> after CHIKV reached the first-step E1-226V *A. albopictus*-adaptive peak, its evolution was no longer constrained to a monolithic peak and multiple adaptive peaks of relatively equal fitness became available for Darwinian evolution. (Tsetsarkin et al. 2014, 10)

They continue, in language that recalls Christie's allusion to Darwinian evolution:

> Overall, our findings somewhat mirror traditional Darwinian models of macroevolution, where major adaptations, such as development of wings by ancestors of birds, or the E1-226V substitution in the case of CHIKV, can result in the rapid radiation/diversification of new lineages/species. (ibid.)

These findings imply that the combination of a bundle of environmental factors, including climate change and population growth, together with increased urbanization, may give rise to future arbovirus outbreaks in the Indian Ocean world (Gaüzère et al. 2012; Abstract).[9]

So was Carey (1971, 261) correct when he concluded of diseases clinically reported in Java in 1779, Zanzibar in 1823 and 1870 (by Christie), and India in 1824, 1871, 1902, 1923 and 1963–1964 that all appear to have been chikungunya or something akin to it? Perhaps. But in the absence of the kind of genetic evidence that we possess for the 2005–2006 outbreak that riveted the attention of modern medical researchers, I tend to concur with the more cautious conclusion of Goro Kuno (2015), who recently reviewed the same confusion between dengue and chikungunya that Carey observed 35 years ago. Kuno cautions that the absence of available details about past diseases, changing diagnostic methods and the descriptive language of observed symptoms render after-the-fact historical identification of diseases extremely difficult. Accordingly, it remains an open question as to whether what in the past has been identified as dengue may have been chikungunya.

[9] For a discussion of "the current geographic range and the relevant biological traits of *A. albopictus* in order to explain its rapid spread", see Paupy et al. (2009); also Delatte et al. (2011).

Comparing Chikungunya to Malaria in the Mascarenes

Notwithstanding Kuno's cautionary words, I propose to take these lessons from the very recent history of chikungunya detection and apply them to our thinking about the longer-term history of disease dispersion in the IOW. I am under no illusions about the limitations of such an exercise, but I would like to assess what may be possible in the following section of my chapter. To do this, I will focus on the disease history of Mauritius and Réunion. These two remote islands share a deep history of mid-ocean isolation and the absence of indigenous human populations before European colonization, as well as closely linked colonial and settlement histories. They also shared many of the same epidemic diseases that reached them across the water during the first centuries of French and, later, British colonial rule.

Historically, perhaps the most interesting comparison with chikungunya is malignant malaria, which struck Mauritius in 1867 as an endemic crowd disease and then Réunion in 1869.[10] Quite apart from early (and some later) depictions of the Mascarenes as Edenic in their healthfulness (compare Ève 2009), there is no evidence that this most deadly form of malaria existed endemically prior to its introduction in the 1860s. However idealized this image may have been, especially in light of the history of both smallpox and cholera epidemics in the decades preceding the malaria outbreak (Boodhoo 2010, 57–62, 134–137, 143–145, 149, 159), it is quite different from the negative reputation of the notoriously unhealthy Maldive Islands (Knoll, this volume and 2018). In his 1908 study of malaria prevention in Mauritius, Nobel Laureate Sir Ronald Ross, who discovered the link between the malaria parasite and its mosquito-borne vector, argued that, although there were undoubtedly some, probably many, captive Africans and Malagasies, as well as Indian indentured labourers, who came to the island carrying malaria, it was not present there as an endemic disease before the 1860s (Ross 1908, 44). In his opinion, but begging the question of what is meant specifically by malaria, had malaria been present even minimally, people in Mauritius would have recognized it and commented on its presence (ibid., 45). The fact that the island was

[10] From a contemporary public health perspective, both dengue (DENV) and the zika virus are clearly important comparisons, especially as they relate to tropical islands like the Mascarenes. See Cao-Lormeau (2016).

used by the British as a hospital for ailing military personnel from its Indian Ocean empire holdings apparently convinced Ross that malaria was not a significant presence in Mauritius. To demonstrate this conclusion, he cites an analysis of medical statistics for British troop mortality rates in Mauritius, which show that upticks in malaria cases among British troops who had arrived on the island from India and China in the decade before 1867 were a result of relapse, rather than of the presence of endemic malaria (ibid.).

Like the challenge of identifying chikungunya noted above, Ross points to the presence of other fevers in Mauritius before the malaria epidemic of 1867 that complicated matters of disease identification (ibid., 46). Most significant among these was a form of epidemic relapsing fever that mainly affected the Indian population and was known locally as "Bombay fever" (ibid., 47). However, when the malaria epidemic of 1867 hit Mauritius, for Ross there was no mistaking the fact that it represented an entirely different disease. As Ross noted, "Accounts of eye-witnesses of the fever at Port Louis recall descriptions of plague and cholera" (ibid., 48). More than one-fifth of the population of Port Louis perished from the fever in 1867, while almost 9 per cent of the total island population died, and in the words of the Fever Enquiry Commissioner's Report of 1868, "the survivors 'were so prostrated by disease that the living were scarcely able to bury the dead'" (ibid.).[11] What Ross clearly described as malaria was unquestionably *Plasmodium falciparum*; what he dismissed as not being malaria may, however, have been *Plasmodium vivax*, *P. ovale* or *P. malariae*, the three other most common species of this protozoan parasite that infect human beings.

"This astonishing occurrence", Ross wrote, "caused much perplexity at the time among the more thoughtful students of malaria. It showed that the disease is at all events not due to any inherent poisonous property of soil, but rather that it might be caused by some living organism capable of invading a country from without". Indian coolies were the favourite target for such suspicions, as were visiting ships, as well as various natural phenomena, such as cyclones. But as we know, and as Ross argued, a solution

[11] Here I must note the very great significance of the inauguration of the combined archival and historical archaeological research at the Bois Marchand Cemetery in Mauritius, a burial place specifically opened to deal with the exceptional number of deaths caused by the malaria epidemic of 1866–1867, being led by Krish Seetah of Stanford University and recounted to us at the 2016 conference (see British Library 2016).

lay in the not yet discovered identification of the mosquito as the disease vector (Ross 1908, 49). Ross considered two competing hypotheses, one that the appropriate mosquito had only recently been introduced to Mauritius, the other that it was already present, but needed a critical mass of infected carriers to become endemic. He preferred the first hypothesis, especially because of the almost simultaneous introduction of malaria to Réunion, and therefore attributed the 1867 outbreak to the recent arrival of a mosquito that he named *Pyretophorous costalis*, a subspecies of *Anopheles gambiae*, as the vector for the malaria parasite (ibid., 49–52).

Indeed, Ross's preference for the first hypothesis has become the accepted interpretation; but if the second were preferred, might it not be possible that, in light of the mutations that I have described above for CHIKV and its mosquito vectors, the same possibility exists for the history of malaria in Mauritius? What I am suggesting here is that, since we know that non-endemic malaria did in fact exist in Mauritius before 1867 in the bodies of enslaved Africans and Malagasies, recuperating British soldiers and Indian indentured labourers, is it not feasible that a similar genetic mutation might have occurred that transformed existing mosquito populations on the island so that they were in a position to become effective vectors for malaria? Certainly, Raj Boodhoo's careful presentation of contemporary efforts to understand "the malaria scourge", as he calls it, provides plenty of evidence for questioning Ross's conviction that earlier forms of fever in Mauritius were not malaria (Boodhoo 2010, 173–191). The tendency to blame its introduction on Indian immigrants, who were massively transforming the demography of the island colony, undoubtedly obscured the possibility that other varieties of malaria might have been introduced previously in non-endemic, non-malignant forms by both enslaved Malagasies and Africans. I find it interesting, for example, that quinine, the most widely used prophylaxis for malaria, was first used to treat fever patients in Mauritius as early as 1828 and that one local physician observed in 1868 that "intermittent fever had always existed" (ibid., 181, 184). It is worth adding here what William Twining noted of Bengal in 1832: "malaria has been generally acknowledged the efficient cause of intermittent fevers" (quoted in Mukherjee 2008, 55). Similarly, yet another medical doctor wrote of Mauritius in the late nineteenth century that "the island had never been a healthy country" (Boodhoo 2010, 186).

Absent from discussions of the origin of endemic malaria in Mauritius is any consideration of contemporary changes in the virulence of malaria in India, especially in Bengal, where "a savage new malaria was devastating

regions previously healthy or lightly afflicted by the malady" (Klein 2001, 147). Specifically, the middle of the nineteenth century witnessed the advance of what was described as "malignant malaria" caused by *Plasmodium falciparum*, the deadliest form of the four species of the Plasmodium parasite that cause malaria in humans. Recalling Mauritian references to "Bombay fever", this newly virulent form of malaria was known by contemporary observers in India as "Burdwan fever" after the area of West Bengal from which it spread across the wider Bengal region; in other areas of Bengal, it was called "Jessor fever", "Nadia fever" or "Hughly fever" (ibid., 161). David Arnold (1999, 136) describes this disease as "the Bengali black death", while Ira Klein (2001, 159) argues that this deadly transformation in malaria was a consequence of the extensive ecological interventions that resulted from British development policies of railway, canal and road construction that transformed colonial India during these decades through deforestation and the increased prevalence of standing water. Indeed, these changes stimulated the proliferation of several different *Anopheles* mosquito vectors that carried *Plasmodium falciparum*. It seems almost certain that it was this "Burdwan fever" that was carried from Calcutta to Mauritius in 1865, a year in which more than 16,000 Indian emigrants embarked from that port aboard 43 ships (Deerpalsingh and Carter 1996, 312). Indeed, Arabinda Samanta (2002, 58) specifically comments on the fact that malaria moved to Mauritius in the mid-1860s during a period of heightened epidemic malaria in Bengal in her important monograph on epidemic malaria in Bengal. We have seen that Ross put forward only two hypotheses for the origin of malaria in Mauritius, either that the mosquito vector had only recently been introduced into Mauritius, or that it was present but required a sufficient number of infected carriers to become endemic. Might not this vivid example of Indian Ocean disease dispersion admit a possible third hypothesis, one that allows for the pre-existing presence of both mosquitoes and non-malignant forms of malaria in Mauritius *and* for the introduction of a newly virulent form of malaria plasmodium from Bengal? According to this hypothesis, these two forms would have combined in ways unknown to precipitate the epidemic of malignant malaria that devastated the island in the mid-1860s.

In the late nineteenth century, however, the question remained unresolved. When Dr Daniel Anderson visited Mauritius in 1889, he was struck by the fact that no one could yet agree on the origin of malaria on the

island (Anderson 1918, 173; compare Floate 1969).[12] Yet he joined Ross in dismissing the earlier school of thought regarding unhealthy soils and claimed that it was not until the arrival of an immigrant ship named the *Spunky* from India in 1865 that malaria was introduced (ibid., 173, 175).[13] On the face of it, this assertion was quite inaccurate, as the British medical records examined by Ross reveal. In any case, at the time of Anderson's visit, the Anopheline mosquito had not yet been identified as the malarial parasite vector. However, in light of what was then known about the disease in West Africa, Anderson asked: "Are there any Anophelines in Mauritius and did they always exist there?" He confirmed that *Pyretophorous costalis* were indeed numerous, mainly in the low-lying coastal zone, but that no one had any idea of how long they had been established on the island (ibid.). Returning to the arrival of the *Spunky* and its malarial passengers, he explained that "Ross's mosquito-carrier discovery had not yet astonished the world". Thus, while his initial impression about how the parasite was somehow a consequence of the unhealthy air and water of the marshes around Port Louis proved to be wrong, the link between mosquitoes and that aquatic environment was correct (ibid., 176). Accordingly, addressing the London Hygienic Congress in 1890, Anderson could declare:

> Now we can answer our question in full. The two or three hundred malarial coolies landed at marshy Petite Rivière infected Anophelines, which at that time of the year and under the special circumstances that had favoured their extensive propagation were ready to bite the newly arrived infected immigrants, and to carry the parasite from village to village and estate to estate. (ibid.)

Staying with Anderson, he next shifted his emphasis from how and when malaria came to Mauritius to the process whereby malaria subsequently became endemic on the island. He noted "its multitudinous small marshes, its grass-ridden streams, and stagnant pools which form on either side of the river-beds after the heavy rains are over". It was in this aquatic

[12] Anderson here cites his 1890 lecture on these events, to which I refer below.

[13] I have found no other reference to this ship, to which Anderson attributes so much significance. Boodhoo (2010, 185) mentions an unnamed "fever stricken ship that had been quarantined off the coast of Albion four months earlier" than the November 1865 outbreak of malaria at Albion estate, located a short way south of Port Louis near Petite Rivière. Perhaps, this ship was the iconic *Spunky*.

environment that the *Anopheles* mosquito flourished. Furthermore, he associated continued Indian immigration and trade with Madagascar, two regions of the Indian Ocean world with long histories of endemic malaria, with maintaining a ready supply of malarial infection (ibid., 178).[14]

To my way of thinking, Anderson's one-to-one conclusion begs the question by taking the Anopheline mosquito as an unproblematic environmental given just waiting to be infected by the Indian carriers of the malaria parasite. But if, as in the case of chikungunya—and as I have just suggested—this insular mosquito population had already undergone certain genetic micro-transformations that rendered them more receptive to the *Plasmodium falciparum* parasite carried in the blood of Indian immigrants from Calcutta, regardless of whether or not they were among the *Spunky*'s passengers, then the sudden eruption of this devastating epidemic makes perfect sense and significantly complicates received interpretations. In addition, since the *Spunky* reached Mauritius in 1865, why did it take a full year for the epidemic to take hold on the island?

An important corollary of the 1866–1867 malaria epidemic in Mauritius was its spread to La Réunion in 1868–1869. Like Mauritius, this French island colony had experienced earlier varieties of vaguely identified relapsing fever, as well as other epidemic diseases such as smallpox and cholera, but never anything identified as malaria. Similar to the situation on its sister island, it initially spread along the low coastal plain, then into the foothills of the island and within three years had become endemic. Not surprisingly, once the mosquito vector had been identified, the main focus of malaria research concentrated on eradication and entered the larger realm of public health policy (see Parahoo 1986; Tchen et al. 2006). More recently, French and Mauritian researchers have sought to understand the specific mechanisms of how malaria came to the Mascarenes. They recognized that they first needed to understand how the mosquito vector established itself on the islands, which they hypothesized occurred in two stages: first the arrival, and second the indigenization of the *Anopheles* (Julvez, Mouchet, Ragavoodoo 1990, 254). Because of the length of time it took to reach the Mascarenes by sail, the short life span (ranging from 5 to 14 days for all four life stages) of the female *Anopheles* mosquitoes and the need for a stable reservoir of fresh water for their eggs to develop, the

[14]This link between the study of disease history and labour migration in Mauritius is a topic that Yoshina Hurgobin (2016) has discussed in a pioneering collection of essays on medicine in the Indian Ocean World (Winterbottom and Tesfaye eds. 2016).

authors ruled out sailing ships—even from Madagascar, where malaria had long been endemic—as a viable means of transportation. What enabled migration across the open waters of the southwestern Indian Ocean and thus facilitated the transportation of the mosquito vectors was, the Franco-Mauritian group deduced, the opening of a regular steamship service by Messageries Maritimes in 1864 that linked the islands to both Madagascar and Africa, without the delays involved in sailing (Julvez et al. 1990, 255; Julvez 1995, 357).[15] For Réunion, Julvez and Mouchet propose in addition that "the theory of wind transport during the trade wind season, since Réunion is located 'leeward' of Mauritius…is supported by the fact that the malaria epidemic started in the North-West of the island facing Mauritius and not near the anchorages" (Julvez and Mouchet 1996, 164).

As research on mosquitoes advanced, investigators sought to identify more accurately than previously the specific variety of *Anopheles gambiae*, which is today the principal vector for malignant malaria globally, that was the original vector. But the existence of four freshwater and two saltwater species of this mosquito, combined with the impact of the mosquito eradication programme on both islands, has complicated matters. Thus, their findings proved elusive except to conclude that "the exclusively African origin of the vectors cannot be doubted" (Julvez et al. 1990, 255). Perhaps of equal interest is their argument that human environmental transformation was an essential factor in preparing the islands for the implantation of malaria. In the case of the two major Mascarene islands (but not Rodrigues or the Seychelles), "the implantation of *Anopheles gambiae s.l.* was consolidated by the deforestation that followed the development of the monoculture of sugar cane, culminating towards 1860" (ibid., 256). In a word, the agricultural transformation of Mauritius for sugarcane plantations, which included road and railway construction, as well as field clearance and the digging of irrigation channels, created an extensive watery environment that was more conducive to the reproduction of the *Anopheles* mosquito (see Julvez et al. 1990, 256; Julvez and Mouchet 1996; Julvez et al. 1998; Boodhoo 2010, 53–54).[16] This is an important general point made by Randall Packard (2007, 12–15 and passim) in his global history of malaria and by Benjamin Reilly (2015) in his case study of malaria in Arabia, as well

[15]For a parallel case of faster steamship travel being the source for the introduction of malignant malaria to northeast Brazil, see Oliveira-Ferreira et al. (2010).

[16]For the Seychelles, see Robert et al. (2011). For the parallel extinction of indigenous birds on the Mascarenes as a consequence of "anthropogenic activities", see Hume (2013).

as by Klein and Samanta for Bengal. Nevertheless, these findings still do not answer unambiguously either how the *Anopheles* mosquito reached the Mascarene islands or, contrarily, whether and how endemic mosquito populations mutated to become vectors for the malarial parasite. For example, in a study of genetic differentiation among geographically distant populations of mosquito vectors, one team of researchers argues that mosquito migration is quite possible across great distances, including oceans (Simard et al. 1999, 1006). In a word, steamships were not necessary for the introduction of *Anopheles* mosquitoes to the Mascarenes. To push these ideas further, closer inspection of the incomplete pieces of information regarding how malaria became an endemic crowd disease in Mauritius in particular raises unanswered questions about chronologies, disease descriptions and the assumptions underlying received interpretations.

Other Possibilities?

There are numerous additional instances of how various epidemic diseases spread around the Indian Ocean, including the Mascarenes. Following up on Myron Echenberg's call for historians to pay more attention to Christie's study of cholera (see footnote 1), a more in-depth exploration of the impact of that disease on the Mascarenes might be essayed. Anderson (1918, 111–151) includes a long presentation on cholera in Mauritius, while medical historians B.A. Gaüzère and P. Aubry (2012) have written about its course on Réunion in the nineteenth century. Although the chronologies of some of these outbreaks in the Mascarenes differ from that described by Christie for East Africa, in other cases there is a direct link, as in the introduction of cholera to Réunion by a boatload of so-called *engagés* (indentured labourers) arriving from Kilwa in 1859 (Echenberg 2011, 55–56; Christie 1876, 113–116; Role 1974).

Another intriguing example is a disease called "Le Barbiers" that repeatedly struck Réunion and other regions of the Indian Ocean world in the first half of the nineteenth century. According to Gaüzère and Aubry, neither the name nor the origins of the disease are known. They note further that it is more than possible that this disease may describe a number of different syndromes (Gaüzère and Aubry 2014, quoted from Abstract). Here again we can see the problem posed by the confusion between dengue and chikungunya: as historians we depend critically on eyewitness and contemporary reported descriptions of whatever it is that we study, in this case epidemic disease, yet words alone are not sufficient for historical analysis.

The literature on chikungunya and malaria suggests to me that, even when we have the full apparatus of modern medical and biogenetic science at our disposal, the identification of epidemic diseases and their dispersion is not always easy. Locating a historically grounded pairing of a disease, whether a virus or a parasite, and its vector(s) is equally challenging. Circumstances matter greatly. In the cases of chikungunya and malaria, even when the terrain is familiar, the mosquitoes that serve as disease vectors are mutable. Moreover, where viruses and parasites are concerned, they too are regularly and constantly adapting to new environmental circumstances, as we can observe with the emergence of resistance to antimalarial drugs, especially by the most lethal form of the malaria parasite, *P. falciparum* (White 2004; Cui et al. 2015). For me, at least, it is this astounding adaptability of both disease and vector that emerges as the most challenging aspect of reconstructing the medical history of disease dispersion in the Indian Ocean World.

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CHAPTER 10

Challenging Chikungunya: Resistance to Public Health Measures and Aetiology During the 2005–2007 Epidemic in Réunion

Karine Aasgaard Jansen

Cyclical monsoon winds have long carried ships across the Indian Ocean. In their turn, vessels have facilitated the diffusion of both human and nonhuman cargoes such as vectors and viruses.[1] Indeed, disease—the focus of this volume—has constituted an integral part of the extensive network of maritime exchange and migration of the Indian Ocean World (IOW). Scholarly studies of the IOW exchange of people and pathogens include David Arnold's seminal article 'The Indian Ocean as a disease zone, 1500–1950' (1991) and the anthology 'Histories of Medicine and Healing in the Indian Ocean World' (Winterbottom and Tesfaye 2016). Travelling disease is, thus, neither a new empirical phenomenon nor only a current interest within IOW studies. This regional disease diffusion was demonstrated in the

[1] Vectors are living organisms that can transmit infectious diseases between humans or from animals to humans. Many of these vectors are bloodsucking insects, such as mosquitoes.

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2004–2007 epidemic of the vector-borne disease of chikungunya, the first ever to hit the IOW. The epidemic spread from Kenya's Swahili coast to the Western Indian Ocean islands of Lamu, the Seychelles, Madagascar, the Comoros, Mayotte, Réunion, and Mauritius, followed by India. In addition, imported cases from exposed travellers returning from the affected areas were later identified in France, Italy, Hong Kong, USA, and Canada (Alpers in this volume; Taglioni and Dehecq 2009; Njenga et al. 2008).

In this study, which is based on a total of eight months of ethnographic fieldwork in the island of Réunion from 2009 to 2010, I will discuss how human-environment interaction in a traditional Réunionese household (*kaz creole*) and its adjacent garden (*jardin creole*) played a central role in forming local perceptions of chikungunya and resistance to public health interventions—and aetiology.[2] My main argument is that many Réunionese interpreted chikungunya, a new disease to the island, within preexisting narrative frameworks of vector-borne disease diffusion and prevention that had developed in response to the malaria outbreaks of the 1950s. I will investigate how the *Aedes* mosquito's breeding grounds in a *jardin creole*, as well as previous public health interventions against vector-borne diseases, contributed to the stigmatisation of chikungunya. Moreover, despite widespread and easy access to public health information regarding chikungunya's mode of transmission, I will argue that stigmatisation may have led many Réunionese to reject the idea that chikungunya was a vector-borne disease.

Chikungunya: An Outline of the Epidemic in Réunion

Chikungunya was first identified in 1953 when the virus was isolated from the blood of a febrile Makonde speaker on the border between Tanzania and Mozambique (Pialoux et al. 2007, 319; see also Alpers in this volume). Chikungunya attacks the joints, leading to painful swelling and considerably reduced motor function that may last for a few days up to several years. The virus is carried by the female *Aedes aegypti* and *albopictus* mosquito, which is also responsible for spreading dengue, yellow fever,

[2] In Réunion, *creole* refers to anyone or anything of local Réunionese origin. This is in contrast to, for example, Mauritius where *Creole* rather functions as an ethnic identity marker for Mauritians who are primarily descendants of African and Malagasy slaves (Eriksen 1998).

and zika. As of now, there is no effective vaccine against the virus, but infection results in lifelong immunity.

According to the French Institute for Public Health Surveillance (*Institut de veille sanitaire*, INVS), the chikungunya epidemic began in Kenya in June 2004 and reached Réunion in March 2005. One month later, its presence was communicated to the general public by the Regional Department of Health and Social Affairs (*Direction régionale des affaires sanitaires et sociales* (DRASS); Watin 2008, 2010; Weinstein and Ravi 2009), the governmental agency responsible for vector control and eradication.[3] DRASS assured the Réunionese population that chikungunya was nonfatal and that the epidemic, which authorities were closely monitoring, would be over by the end of July 2005 (Watin 2008, 243, 2010; Weinstein and Ravi 2009). None of these statements proved correct. The epidemic peaked in the first two months of 2006, with over 25,000 new cases being registered in the last week of January and an astounding 45,000 in the first week of February (Taglioni and Dehecq 2009, 15). In several cases, severe health complications were reported, and in January 2006 chikungunya claimed its first fatal victim worldwide—a ten-year-old boy (Leyral 2006, 14).

At the peak of the chikungunya epidemic, many Réunionese claimed that the air had been polluted by a medical or military experiment, a chemical outlet, or even through biological terrorism inflicted by Bin Laden and his alleged Comoro Muslim accomplices (Jansen 2016, 163). The reaction from France was different. Following the boy's death, the French government acknowledged the potential gravity of the epidemic and responded with economic, medical, and preventive assistance. In total, 91 million Euros were contributed towards research, sanitary improvements, and financial relief. The government also mobilised 720 locally stationed metropolitan—that is, French mainland—military personnel to conduct so-called demosquitofications (P. D. 2006, 6; Payrard 2006, 14), the local term for prophylactic measures such as the spraying of insecticides. These measures occurred, however, fully a year after the virus's presence had been documented in Réunion and almost two years

[3] In April 2010, DRASS underwent an organisational transition and is now known as the *Agence de santé de l'Océan Indien* (The Health Agency of the Indian Ocean, ARS). ARS is non-governmental, whereas DRASS responded directly to the French prefect. The prefect represents the French national government at a local level. As DRASS was still in operation during both the chikungunya epidemic and my main fieldwork in 2009, I will still refer to DRASS and not ARS throughout this chapter.

after the epidemic first broke out in Kenya. Intentional or unintentional misinformation regarding the severity of the epidemic, and late and inadequate official intervention, depending on one's point of view, prompted significant local criticism of the French authorities, and of France's neglect of its overseas citizens (Audifax 2006). Official response to the epidemic also aroused general public mistrust in the public health administration, DRASS included. This politicisation of the epidemic resulted in a deep division, in which the metropolitan French government found itself opposed by all interests in the island, including the Réunionese regional government, local media and civil institutions, and the general population. By April 2007, when Philippe Bas and Hervé Mariton, respectively French ministers of Health and Overseas Departments, declared the epidemic to be over (Payrard 2007), 266,000 local Réunionese, nearly 30 per cent of the island's total estimated population of 802,000 (Rallu 2009), had been infected by chikungunya and 250 had died (Renault et al. 2008).

Methods and Research Background

With some exceptions (e.g., Jansen 2012, 2013; Watin 2010; Weinstein and Ravi 2008, 2009), most studies of the chikungunya epidemic in Réunion have been by epidemiologists and entomologists who used quantitative methodologies (e.g., Pialoux et al. 2007; Renault et al. 2008; Taglioni and Dehecq 2009). However, as a medical anthropologist, I am interested in how disease and illness are experienced, explained, and treated at a local level—and in order to study this, I have employed qualitative methods, notably the gathering of ethnographic data based upon researcher participation, which provides a good indication of every-day social life as it unfolds in particular contexts. A large part of my fieldwork in Réunion was, for example, spent outdoors in the gardens of 16 informants who had suffered from chikungunya during the epidemic. Due to the island's temperate weather, people spend as much time outdoors as indoors. A *jardin creole* functions as a de facto extension of a *kaz creole*, and people's activities and interactions in them played a central part in this study's data collection and analysis. Conversations with informants were often carried out while performing various household and gardening chores, such as doing laundry, watering plants, and picking and shelling beans, or enjoying a cup of coffee on the *varang* (overbuilt terrace) in front of a *kaz creole*. During an intense week of fieldwork that followed the discovery of three cases of chikungunya in Réunion in August 2009, I also

joined a team of DRASS field agents to conduct daily garden inspections for mosquito breeding grounds. This enabled me to learn more about vector-borne diseases in general and chikungunya in particular, DRASS's preventive methods, and how DRASS and the public health policies they implemented were received by the public. From the peak of the epidemic in January 2006, DRASS officials regularly conducted garden inspections as part of their preventive public health measures, and these were still being conducted during my last period of fieldwork in late 2010.

A crucial part of fieldwork is the writing of field notes in order to gather ethnographic knowledge (Emerson et al. 1995). Observations and conversations are meticulously jotted down from notes taken on site, or, as in this case, primarily at home in the evening. I also conducted semi-structured interviews with informants, public health and sanitation staff, entomologists, and epidemiologists, in Creole or French according to their preferences. Furthermore, I analysed various written sources on chikungunya, such as local and national newspapers, public health reports, and preventive campaign material. In addition, I reviewed governmental documents and newspaper reports of the massive anti-malaria campaigns that took place on the island during the 1950s at the Departmental Archives in Réunion's capital, St Denis. This review allowed me to compare the public health discourses on chikungunya with those of a previous vector-borne disease, specifically the discourses surrounding their spread and the preventive measures that were adopted in response to them.

Placing Chikungunya: Disease Transmission, Vector Adaption, and Living Conditions

The location for my fieldwork was the town of St Pierre on the west coast of Réunion. St Pierre has a population of approximately 26,000 and was one of the island's urban centres most affected by the epidemic. Indeed, the first case of chikungunya in Réunion occurred in a St Pierre neighbourhood inhabited by several of my informants, close to a public semi-open-air *lavoir*, or wash house. A *lavoir* consists of a long row of large double washbasins set in stone and in this case was covered by a slightly dilapidated and rusted tin roof. The risk of contracting chikungunya increases with exposure to stagnant water, in both artificial and natural reservoirs, that acts as a breeding ground for the disease vector. Examples of artificial reservoirs of stagnant water include wash basins, such as the

ones at the *lavoir*, gutters, flower pots, bottles, bottle caps, plastic food containers, and discarded car tyres, while rain ponds and plant axils typically comprise natural reservoirs (Bonn 2006).

As the *Aedes* mosquito thrives in close-knit spaces provided by artificial reservoirs in densely populated and urban environments (ibid.; Nading 2014), chikungunya affected large sectors of the Réunionese population, above all in areas where people live in close proximity to each other. Moreover, according to Raude and Setbon (2009, 690), there is a well-established link between a person's socioeconomic position and his or her risk of contracting chikungunya. In St Pierre, the neighbourhood surrounding the *lavoir* called *quartier lavoir* is known as a *quartier chaud* (hot area)—a term denoting high local unemployment rates and accompanying social problems.[4] In addition to being used for its original purpose of doing laundry, the particular *lavoir* in question also served a social function as a neighbourhood meeting place. This contributed towards the accumulation of waste near the *lavoir* and, in turn, gave the *Aedes* mosquitoes an additional breeding ground. While the women who lived in the nearby houses, the majority of whom were Mahoran, were busy washing clothes and chatting with each other, men sat in small groups on the grassy slope next to the *lavoir*, drinking rum and eating take-away food, playing cards, dominos, or the guitar, and watching the women work. As much as the *lavoir* itself contributed towards the accumulation of stagnant water in ponds, so did some of the men's leftover rubbish such as bottles, bottle caps, and food containers.

That the first case of chikungunya was identified in the *quartier lavoir* was brought up by several informants living in the area's vicinity. Instead of looking for mosquito breeding grounds in their own gardens, many informants looked 'over the fence' to poorer neighbourhoods for possible mosquito breeding grounds, especially in *quartiers* primarily inhabited by Mahoran immigrants. This contributed to the stigmatisation of St Pierre's large population of Mahorans as responsible for spreading the disease. However, although poor sanitary conditions in economically and socially challenged areas may have facilitated the spread of vector-borne diseases (Castro et al. 2010; Nading 2014; Raude and Setbon 2009), the *Aedes* does not differentiate between different sources of stagnant water. The mosquito can equally breed in the flower pots that characterise well-kept

[4] As of 2010, Réunion had the highest level of unemployment in all of the French departments (over 30 per cent) (Roinsard 2010, 21).

gardens of better-off neighbourhoods, and are the distinguishing feature of a traditional *jardin creole*, the pride and joy of many of my Réunionese informants who lived near the *quartier lavoir*.

Staging the Context of the *Kaz* and *Jardin Creole*

While the concept of a garden may intuitively bring to mind the manicured lawns and meticulous flower beds of suburban America (Jenkins 1994), the purpose of a *jardin creole* in Réunion is not merely decoration or leisure. Instead, a *jardin creole* mirrors the spatial layout of a *kaz creole* and acts as a stage for the unfolding of every-day domestic life in both rural and urban, and privileged and impoverished neighbourhoods. A *kaz creole* is composed of two compartments, and each has a distinct purpose in terms of practicality and comfort. These compartments consist of the *kour devant* (the front garden facing the street) and the *kour derriere* (the garden at the back of the house) (Wolff 1991). The *kour devant* functions as the public area of a *kaz creole* and can be compared to Erving Goffman's (1992 [1959]) dramaturgical understanding of a front stage for social behaviour. This is, for example, where the household commonly receives guests on the *varang* for a cup of sweet tea or coffee, usually served among potted plants, and if space allows it, frangipani, mango, or lychee trees, sometimes with orchids growing off their trunks. In a *jardin creole*, plants are typically potted rather than grown in flower beds. The quantity of potted plants varies from a few to several hundred according to the household's available space and income. With the potted plants as stage props, the performance, in Goffmanian terms, thus concerns both the aesthetic staging of the *kour devant* and the social interactions and activities taking place there. Here, it may be useful to compare the function of Réunionese gardens with those of urban Swedish allotments where, Michel Conan (1999, 200) claims, gardeners function as actors who play out the myth of what they imagine to have been a good rural life in 'the olden times'. This re-enactment is done through the performance of various social activities, such as collective gatherings and sharing traditional meals, and it bears a resemblance to the sipping of coffee on the *varang* in the *kour devant* of a *jardin creole*.

However, a *jardin creole* also consists of a *kour derriere*, which can be seen as the very opposite of the staged *kour devant* in both a literal and an allegorical sense. In the *kour derriere*, public performance is replaced by private undertakings that are considered 'behind the scenes' activities,

such as cleaning and drying clothes, food preparation, and other mundane household tasks. Moreover, the stage props found in the *kour devant*, such as pineapple flowers, anthuriums, hibiscus plants, or bougainvillea, are replaced in the *kour derriere* with weeds growing among various discarded items, such as car tyres, bicycles, toys, bottles, gardening tools, and furniture or electrical appliances both in and out of use. The *kour derriere* may also house the household's poultry, dog or bird cages, and storage sheds (Wolff 1991). It thus functions as the back stage of a *kaz creole* and is intended for the household's members only. It was therefore with considerable pride that I accepted an invitation to do laundry on a regular basis with one of my neighbours and key informants in her *kour derriere*. Nevertheless, even then, the distinction between the front and back stages was maintained when it was time for our coffee breaks, which always took place on the *varang*.

The actual *kaz* (the house) is situated between the *kour devant* and the *kour derriere*. Moreover, as Éliane Wolff (1991) notes, such living patterns are common not only in the traditional one-family *kaz creole* but also in terraced houses or blocks of flats. Here, the *kour devant* and the *kour derriere* are represented by, for example, terraces, smaller front- and backyards at street level, or even rooms at the front and the back of an apartment. As potted plants do not require any ground or space to speak of, a *jardin creole* is therefore also common in urban areas, including in social-housing estates.

Gardens and Mosquitoes: Touching on Human-Environment Interaction and Multispecies Ethnography

From a cultural perspective, the Réunionese garden is less about gardening than housekeeping and living in general. For decades, anthropologists have been investigating people's relations with their gardens as part of a wider social system (Hasna 2003). Early anthropological studies of gardens have illustrated the importance of these spaces for economic life, in addition to their symbolic importance as representations of society as a whole (ibid.). A classic example of this line of research is Bronislaw Malinowski's (1935) study 'Coral Gardens and their Magic'. From a functionalist perspective, Malinowski documents the interrelations between the Trobriand Islanders' gardening activities, religious practices,

and agricultural economy, all of which he claims form a holistic part of the Trobrianders' organisation of social life.

More recent studies such as Conan (1999) consider that on the one hand gardening reproduces societal structures and on the other illustrates people's relationship to nature (ibid., 202). Contextual human-environment interaction—that is, the interrelations between people and their surroundings—entails how people act on and respond to the environment and how such exchanges influence the social organisation of a given society (Hasna 2003). Mahbuba Hasna (ibid.) claims, for example, that gardening connects people to their plants and environment through a systemic process. However, her fieldwork in Inhambane, Mozambique, leads Julie Archaumbault (2016) to argue that gardening is also an aesthetic and often profoundly affective endeavour. According to Archaumbault (ibid., 246), human-plant relations should be understood not merely as a way in which societies reproduce their social bonds, but also a reflection of genuine love for plants.

While clearly stating the relevance of continued anthropocentric analyses, Archaumbault (ibid., 248) also engages with the growing field of multispecies ethnography, which focuses both on humans and other organisms that are linked to human worlds and livelihoods (Kirksey and Helmreich 2010, 545). The core relationships scrutinised are both human-to-human and human-to-nonhuman. The latter can include gardeners' relationship with their plants, as discussed by Archaumbault (2016), or people's relations with animals, insects, fungi, and microbes. Multispecies ethnography focuses on multispecies mingling in zones of contact where the dichotomy between nature and culture is challenged in ways that generate new ecologies and 'becomings' (Kirksey and Helmreich 2010, 546). In this context, in his analysis of how residents of Ciudad Sandino in Nicaragua share their lives with dengue-carrying *Aedes aegypti* mosquitoes, Alex Nading (2014) uses the concept of 'entanglement'. He argues that in studying the diffusion of dengue, it is both difficult but also analytically unproductive to separate humans from nonhumans, such as mosquitoes, because their lives are so intertwined (ibid., 19). Kirksey and Helmreich (2010, 545) take the concept further by suggesting that such entanglements can be perceived as 'symbiopolitical', whereby a 'multitude of organisms' livelihoods shape and are shaped by political, economic, and cultural forces'. As pointed out by Hugh Raffles (2010), people's relationships with mosquitoes are usually less affectionate than, say, Inhambane gardeners' love for their plants (Archaumbault 2016). Not only are mosquitoes a nuisance to most people, but they can also spread

life-threatening diseases (Raffles 2010). Nevertheless, as Nading points out, while the language of risk prompts us to think in terms of the body's proximity to mosquitoes and viruses, risk also comes in the form of the body's entanglement with them (2014, 92). In Réunion, both flower pots in the *kour devant* and the accumulation of items in the *kour derriere* may lead to a flourishing environment for the *Aedes* mosquito. Mosquitoes, sometimes disease carrying ones, are familiar features of Réunionese landscapes. As M. Noel, a 56-year-old informant, noted:

> The chikungunya mosquito, it lives in the environment where we live, and thus we don't really notice it [...] It's not a new mosquito. When I was a child, I lived in St Rose where I often frequented the vanilla fields. And in those fields there was only this type of mosquito, the white and black mosquito. I have always known the white and black mosquito. (Interview, 14 September 2009)

Several other informants also brought up the fact that the *Aedes* mosquito, with its characteristic white- and black-striped body, was a long-standing and well-known part of Réunion's environment. However, the fact that such a familiar part of the island's ecosystem suddenly began spreading a previously unknown disease led many Réunionese to question the aetiological origin of chikungunya. While mosquitoes were commonly presented to me by research participants as an intrinsic part of Réunionese gardens, chikungunya was often depicted as something alien and aerial (Jansen 2013, 180–181). In the following section, I will discuss how this interpretation of chikungunya was based on previous experiences with public health measures against vector-borne diseases, particularly malaria, and the post-colonial and political discourses surrounding these interventions.

Public Health Interventions and Past and Present Stigmatisation of Vector-Borne Diseases

According to DRASS, about 70 per cent of mosquito breeding grounds are located close to people's homes.[5] Consequently, public health interventions have been primarily directed towards the control and eradication

[5] This information is from the DRASS preventive campaign pamphlet '*Adoptons les bons gestes*!'/'Embrace the right actions!'.

of mosquito breeding grounds through so-called 'demosquitofication' of people's gardens in Réunion.

During the chikungunya epidemic, such 'demosquitofications' were mostly conducted by French metropolitan soldiers working with local DRASS officials. However, the cultural differences between the two often created problems. In addition, the soldiers' ignorance of local practices and concepts of intimate and public spheres within the *jardin creole* led many of the soldiers to disregard the cultural-spatial boundaries of the *kour devant* and the *kour derriere* in their search for mosquito breeding grounds. This in turn could result in friction between the soldiers and local residents. For example, one of my key informants, 72-year-old Gabrielle, who had suffered from chikungunya, risked a substantial fine for denying soldiers access to her *jardin* to conduct a demosquitofication. She also declined their offer of free anti-repellents, as did 41-year old Nathalie: 'I didn't protect myself! There were no products in my house. I walked normally, I dressed normally, and that's it' (Interview, 16 July 2009).

Moreover, Gabrielle and other elderly informants often compared these garden inspections to those that took place during the extensive governmental anti-malaria campaigns on the island during the 1950s (Zettor 2010). With the development of the insecticide DDT in 1939, the fight against malaria accelerated worldwide. In Réunion, anti-malaria work was undertaken by the governmental Prophylactic Services (SDP) and was later taken over by DRASS. Officials from these agencies not only conducted garden inspections similar to those that took place some 50 years later during the chikungunya epidemic, but also sprayed the insides and outsides of peoples' houses with large amounts of pulverised DDT mixed with petrol (ibid.). This method was known locally as 'house painting' and was considered to be highly effective, as it rid people's homes of not only mosquitoes but also of lice, bedbugs, fleas, scabies, and ticks. Despite the island's departmentalisation and full integration with metropolitan France in 1946, French welfare provision and social reforms were only put on the agenda in Réunion during the latter half of the 1960s. Consequently, between 1946 and the late 1960s, the majority of Réunionese suffered from severe poverty and poor sanitary conditions, which contributed to the spread of various vector-borne diseases. As I have discussed in previous papers (Jansen 2013, 2016), even during my fieldwork in 2009 and 2010, Réunionese people commonly associated vector-borne diseases with the work of the SDP. As Raffles (2010) noted, when preventive measures were

lacking, living with vectors was a serious and potentially lethal health hazard. For example, in 1948, malaria was responsible for 38 per cent of deaths annually in Réunion (Vaxelaire 2009, 608; Zettor 2010, 18).[6]

Many Réunionese, particularly the elderly, associate these past experiences of unsanitary conditions and malaria with French public health measures. As Gabrielle put it, if one kept one's *jardin* well-kept and clean, consequently there would be no mosquitoes there to transmit disease. Since she fell ill with chikungunya herself, the disease could therefore not be vector-borne since, in her opinion, her *jardin* was immaculate (Jansen 2013, 182). The problem, according to her, was rather the nearby *quartier lavoir* and its Mahoran immigrants. Similarly, Nading (2014) documents the common association of mosquitoes with poor housekeeping in Ciudad Sandino, where residents believe that their neighbours' lack of appropriate domestic cleanliness contributes to the spreading of dengue. Seventy-two-year-old Sylvaine, who lived in a social-housing estate, compared mosquitoes to rats, the classic symbol of the spreading of bubonic plague. To my surprise, several younger informants who had no personal experience with the previous malaria-eradication programmes also claimed that chikungunya was not a vector-borne disease. For example, Nathalie compared chikungunya to 'a passage of polluted air', a bad-air hypothesis of chikungunya infection similar to traditional miasma theories that were also held by many other informants (Jansen 2013).

Miasmatic Disease, Colonial Medicine, and Power Play in Réunion

In miasma theory, which originated in the Middle Ages, diseases were believed to be caused by the presence in the air of 'miasma', a poisonous vapour caused by rotting, foul-smelling organic material. Miasma theory remained the dominant aetiological explanation for disease diffusion well into the nineteenth century when rapid industrialisation and urbanisation in Europe created many poor and unsanitary neighbourhoods that often tended to be focal points of epidemics. Improved housing and sanitation removed bacteria and mosquito breeding grounds.[7]

[6] Malaria was eradicated in Réunion in 1979 (Fontenille et al. 2009, 79).

[7] For example, malaria originates from the Italian words *mala* (bad) and *aria* (air).

Although germ theory largely replaced miasma as an explanation of contagion from around the 1870s, it is still used to make cultural sense of disease by many ordinary people worldwide (Herring and Swedlund 2010; Rosenberg 1992). Certainly, many Réunionese, even today, believe that miasma is the cause of chikungunya. Miasma theory implies exposure to unsanitary environments, in particular noxious mists or vapours from decomposing organic matter near human dwellings, such as the dirty and stagnant water in which mosquitos can breed. Of my 16 informants, 9 claimed that chikungunya was transmitted through foul air. The seven others favoured a mixture of miasma theory and biomedical explanations in which mosquitoes were considered to have first contracted the chikungunya virus from rotting organic material and then spread it to humans (Jansen 2013).

As noted, local people doubted that chikungunya was a vector-borne disease, in large part due to their suspicions about the public health policies of DRASS officials and French soldiers during the 2005–2007 chikungunya epidemic. The discourse surrounding the anti-malaria campaigns of the 1950s and the general political climate on the island at the time also contributed to popular antipathy to the authorities. There was, for example, mounting criticism of the failure of the French government to improve local economic and sanitary conditions. Such sentiments were in part responsible for the 1959 Réunion Communist Party declaration in favour of independence from France. However, such a prospect was anathema to Paris, which considered Réunion to be a key military base in the region (Finch-Boyer 2010). Consequently, France withheld welfare provisions until the island's residents voted against independence in the 1963 election, swayed by local politicians loyal to France, who argued that independence would result in greater poverty, while continued attachment to France would be rewarded by substantial economic, social, and health benefits (Vergès 1999; Jansen 2016).

Following the pro-France vote in the 1963 election, substantial infrastructural changes were finally initiated in housing, schools, roads, and electricity (Vaxelaire 2009), and the same rights to health insurance, social and family allocations, and social housing were provided as to metropolitan French citizens (Finch-Boyer 2013; Vergès 1999). This 'welfare colonialism' continued to ensure that Réunion remained French by making it economically dependent on the metropole (Finch-Boyer 2013). I here contend that 'welfare colonialism' also made Réunion French with regard to disease on the island. Thus, the metropolitan French notion of 'tropi-

calism', meaning the association between disease and so-called 'exotic' and 'primitive' places and people (Weinstein and Ravi 2008), became current in Réunion where it continued to be reflected in many local illness narratives also during the 2005–2007 chikungunya epidemic. This association was also reinforced by scientific communication of chikungunya. For example, the dust jacket of the popular scientific book '*Le chik, le choc, le cheque*' (The chik[ungunya], the shock, the check) published in 2006 by two locally based medical doctors notes:

> Réunion has painfully reconnected, due to the interference of chikungunya, with its ancestral tropical and African roots. What is this mysterious virus with a cannibal name which bends the spine and eats at the cartilage and pride? (Gaüzère and Aubry 2006)

Moreover, despite the lasting threat of subtropical and vector-borne diseases in all French DOMs in the Indian Ocean and the Caribbean, not until 2006 were chikungunya and dengue included in the French notifiable disease surveillance system (Weinstein and Ravi 2008, 227). These tropical diseases have thus literally not been on Réunion's epidemiological radar. Thus, many Réunionese relate tropical disease, chikungunya included, to the island's colonial past rather than its French present, perceived as the implementation of sanitation and civilisation through welfare colonialism. For example, Gabrielle compared the previous poor health and sanitary conditions of Réunion to what she perceives to be the present state of Mauritius, which she considers to be, in negative terms, highly 'exotic' compared to Réunion.

Conclusion

Réunionese responses to the chikungunya epidemic both challenge past and current public health measures, by employing miasma theory as 'alternative' disease aetiology to vector-borne theories, and simultaneously mirroring colonial racialist health discourses (Winterbottom and Tesfaye 2016). There were, for example, close connections between miasma theory, colonial medicine, and early hygienic approaches to public health in tropical locations (Greene et al. 2013, 50). While European colonists settled in hilltop areas where a vast supply of fresh air kept them above the range of mosquitoes, healthy native-borne populations, who were often considered to be vectors for disease themselves, were relegated to the

humid, hot, 'fetid', and densely populated lowlands, often in port cities. In Réunion, not only were local people relegated to the lowlands by colonists, but the mind-set accompanying the spread of vector-borne disease to particular environments also appears to have been adopted by many Réunionese. Moreover, it seems to continue to influence their view on disease. Since vector-borne diseases such as chikungunya have unsanitary connotations in Réunion, mosquitoes were not perceived as responsible for spreading the disease. Instead public health officials and soldiers with so-called accusatory attitudes concerning local domestic cleanliness were considered trespassers in ordinary people's gardens during the epidemic.

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CHAPTER 11

Inherited Without History? Maldive Fever and Its Aftermath

Eva-Maria Knoll

Although not been recognized by the international media, the population of the Republic of Maldives has had to face a major health dilemma—the world's highest prevalence of beta-thalassaemia, an inherited single-gene disorder that affects the body's ability to create haemoglobin, the red blood cells crucial to providing oxygen to the cell tissue. The most serious form, beta-thalassaemia major, results in severe anaemia in the first months of life and requires lifelong care.

In a Facebook posting, Mariyam, a 25-year-old beta-thalassaemia-major patient, revealed that every 20 days, she has to spend about seven hours in the transfusion clinic receiving donor blood. This procedure started when she was an infant, so she cannot recall her first blood transfusion. While regular blood transfusions have kept Mariyam alive and healthy, there is also the risk that they might poison her because each unit of blood adds critical amounts of iron to her body, which accumulates especially in her organs. To remove this excess iron, she uses a pump to inject iron chelator slowly into her body over a period of about eight hours, five days a week.

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