ABSTRACT

Cholera made a sudden, unexpected appearance at Conakry, Guinea in August, 1970 after a seventy-five year absence. Cholera has since spread throughout West Africa and to North Africa, East Africa, Angola and South-Eastern Africa. The patterns of inter-regional diffusion of cholera in Africa have been mapped. The study focused on West Africa, where four distinct forms of cholera diffusion were found. These are the coastal, riverine, urban hierarchical and radial contact diffusion types. Diffusion barriers and channels, plus distance and central place attraction were found to be important in determining the paths of inter-divisional diffusion of cholera. A summarizing model was made for each diffusion type. Local cholera survival and diffusion was examined through a case study of local diffusion in Lagos and brief summaries of published case studies. General systems modelling techniques were used to focus on key elements and relationships involved in inter-divisional cholera diffusion and local survival and transmission.

DIFFUSION OF CHOLERA IN WEST AFRICA, 1970-1974

by

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CHAPTER 1

INTRODUCTION

The Study of Infectious Diseases by Medical Geographers

A growing number of geographers are becoming interested in geographic relationships in the occurrence of infectious diseases. Studying the geography of infectious diseases is but one facet of medical geography, which has been defined as "the application of geographic concepts and techniques to health-related problems".[1] This definition readily facilitates the incorporation of the wide spectrum of health-related problems being studied by geographers, including psychosocial stress, location of health care facilities, and the impact of environmental pollution; and it broadens the definition given by Jacques May in American Geography Inventory and Prospect. May defined medical geography as "the study of the area distribution of disease and its relationship to the environment".[2]

Interest in geographic aspects of infectious disease is not a new phenomenon. Consideration of environmental factors in health was a basic feature of Greek medicine. The period between 1835 and 1855 has been called the "Golden Age" of medical cartography by Gilbert.[3] Cholera was one of the most intensively studied diseases during this era. John Snow's

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1 J.M. Hunter, "On the merits of holism in understanding societal health needs", The Centennial Review, vol. 17, No.1 (1973), p.4.

2 J. May, "Medical Geography", in P.E. James and C.F. Jones (eds.), American Geography: Inventory and Prospect, AAG, 1959, p.453.

3 E.W. Gilbert, "Pioneer maps of health and disease in England", Geographical Journal, vol. 124 (1958), pp.172-183.

treatise on cholera in London in 1849 was a landmark study. By careful mapping of cholera deaths in a part of central London, he was able to demonstrate a connection between the spread of cholera and the use of water from the pumps of a particular supply company.[4] Previously, cholera had been considered to be an air-borne disease. Interest in medical geography declined during the second half of the nineteenth century with the shift of attention to the study of pathogenic bacteria and the search for antibiotics to combat them. Interest in medical geography has gradually revived since about 1950. J.M. May's World Atlas of Disease (1950), Ecology of Human Disease (1958), and series on the ecology of malnutrition, L.D. Stamp's Geography of Life and Death (1964), R.M. Prothero's Migrants and Malaria (1965) and the volume of selected papers edited by N.D. McGlashan, Medical Geography Techniques and Field Studies (1972) are significant landmarks in the recent study of infectious disease by geographers.

There are four basic approaches used by medical geographers in the study of infectious disease. These are the map approach, map correlation approach, quantitative approach and ecological approach.

(a) Map approach: Spatial variations in the incidence of a disease are mapped. This is the approach used in most medical atlases. Maps showing patterns of disease diffusion may also be included as part of the map approach.

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[4] J. Snow, Snow on Cholera (reprint of 2 papers by J. Snow, M.D.). New York: Commonwealth Fund, 1936.

(b) Map correlation approach: The correlation between a disease and certain environmental conditions is demonstrated by showing the coincidence of their spatial distributions. This was the method used by Snow in his noted study of cholera.[5] Allen-Price's map relating cancer deaths to water supply sources is another example of this approach.[6] Computer mapping is a recent innovation in the analysis as well as the portrayal of disease distributions.

(c) Quantitative approach: Geographers are making increasing use of analytical models and such quantitative techniques as correlation and regression, factor analysis and graph theory to analyse statistical data and thereby understand the distribution or dissemination of a disease. Two examples of the quantitative-model approach are Haggett's modelling of measles outbreaks in South Western England[7] and Brownlea's development of a geographic model of infectious hepatitis.[8]

(d) Ecological approach: Studies of the disease transmission cycle and the environmental conditions which facilitate the persistence of infection in an environment or its spread to a new environment may be called the ecological approach. The study by Hunter in Northern Ghana of

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5 Snow, op.cit.

6 G.D. Allen-Price, "Uneven distribution of cancer in West Devon with reference to the divers water supplies", Lancet, June 4, 1960, pp.1235-1238.

7 P. Haggett, "Contagious processes in a planar graph", in N.D. McGlashan (ed.), Medical Geography: Techniques and Field Studies, London: Methuen, 1973, pp.307-324.

8 A.A. Brownlea, "Modelling the geographic epidemiology of infectious hepatitis", in N.D. McGlashan (ed.), op.cit., pp.279-300.

spatial and environmental relationships and implications of the river blindness transmission cycle demonstrates the potential of this approach.[9]

The four approaches listed are not mutually exclusive categories, but rather areas of emphasis. Distribution maps, consideration of the transmission cycle and some form of statistical analysis are basic to almost any study of infectious disease. The choice of the primary strategy for studying a problem generally is based on the researcher's training and philosophy of geography, on data availability and reliability, and on the scale of analysis.

Disease Diffusion

Either endemic or epidemic occurrence of a disease may be studied. Endemicity refers to the persistence of a disease in a given location. The transmission cycle is generally the key to this persistence. An epidemic may refer to either a seasonal or periodic flare-up of a disease in its endemic area, or its spread to a new area. Particularly with newly introduced epidemics, disease diffusion is an important concept. Disease diffusion refers to the process of spread of a disease through space and time.

Considerable research interest has focused in recent years on spatial diffusion. Many of the models and concepts used in studying the

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[9] J.M. Hunter, "River blindness in Nangodi, Northern Ghana: A hypothesis of cyclical advance and retreat", The Geographical Review, vol.56 (1966), pp.398-416.

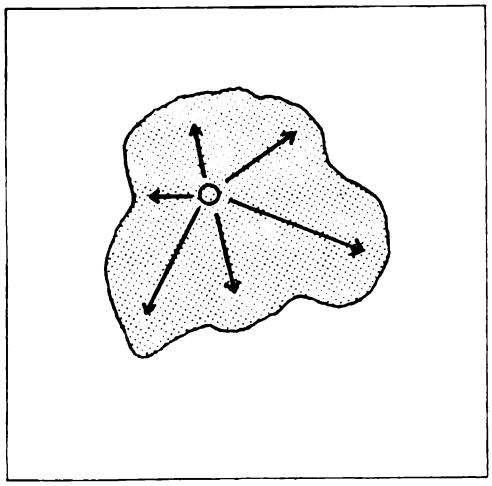
FIGURE 1

THREE TYPES OF SPATIAL DIFFUSION

Contact diffusion is a wave-like outward movement to adjoining territory commencing at a central point. In hierarchical diffusion, the direction of diffusion is from the largest and most important cities in a central place urban hierarchy to progressively smaller and less important ones. Relocation diffusion involves a sudden transfer over hundreds or thousands of miles with no impact on the intervening territory.

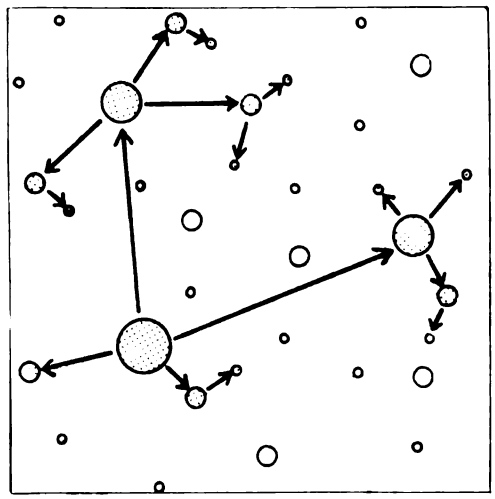
FIGURE 1

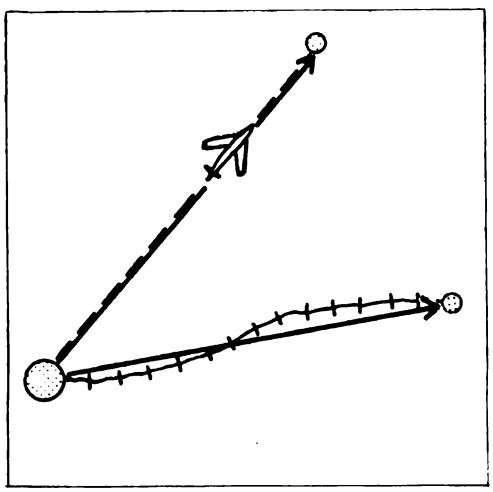
THREE TYPES OF SPATIAL DIFFUSION



Contact Diffusion

Hierarchical Diffusion





Relocation Diffusion

spatial diffusion of innovations are also applicable in disease diffusion. Three basic types of diffusion, which are shown in Figure 1, are expansion or contact diffusion, hierarchical diffusion and relocation diffusion.[10] An infectious disease may spread in any of these ways. Where transportation is evenly developed, a disease may spread outward like a wave from a central focus. This is a form of contact diffusion. If infection enters the largest city in a central place urban hierarchy, it is likely to diffuse in a stepwise manner down through the hierarchy. The long distance jump of an infection by means of a traveller on an airplane or other means of rapid transportation is essentially relocation diffusion. The amount and types of human interaction are crucial for both innovation and disease diffusion. Interaction is governed by such factors as distance, intervening opportunities, and the distribution of attractions and facilities in space.

The configuration of barriers and channels, both natural and man-made, is a very important determinant of diffusion patterns. The logistic curve of adoption roughly corresponds to the cumulative epidemic curve in a community.

Mathematical epidemiologists have developed models to predict the spread of epidemics through space and time. N.T.J. Bailey's The Mathematical Theory of Epidemics is one of the most important works in this field. A key element of the mathematical epidemiological models is the concept of a susceptible population, members of which are subject to

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[10] P. Gould, Spatial Diffusion, Washington: AAG. Commission on College Geography Resource Paper No.4, 1969, p.35.

infection. Immunity may be gained through exposure to infection and recovery, genetic immunity, or through vaccination or chemical prophylaxis. Unfortunately, the diversity of possible transmission media and the "iceberg effect of a preponderance of asymptomatic carriers in cholera would make it extremely difficult to use susceptible population-immune population models in the analysis of a cholera epidemic.

Study Objectives

This study focuses on the following objectives:

1. To determine and map spatial and temporal patterns of cholera diffusion in Africa since 1970. Emphasis will be placed on West Africa. Besides diffusion patterns at the national and international scales, more detailed local case studies from areas with differing environmental conditions will be examined. By examining cholera diffusion at different scales the effect of scale-sensitive factors in diffusion may be examined. For example, certain factors may operate only in diffusion within a small community, and the effects of others may be most clearly illustrated at the international scale of analysis.

2. To determine the key factors governing the patterns of diffusion. This requires a consideration of diffusion principles and their application under West African circumstances. A careful consideration is also needed of the cholera transmission cycle and its interactions with elements of the West African environment.

3. To determine ways in which this study of cholera in Africa can be shown to confirm, extend or refute existing spatial diffusion concepts and models.

4. To construct a systems model showing the key relationships involved in the survival and diffusion of cholera in Africa.

5. To determine areas where cholera has remained endemic following the initial epidemic invasion, and the characteristics of these endemic areas.

6. To use correlation analysis and analysis of variance to identify variables related to the observed diffusion pattern for selected countries.

Data Sources and Data Reliability

Cholera is one of four diseases which are governed by international regulations requiring prompt reporting of confirmed and suspected cases to the World Health Organization. The number of cases and deaths for each reporting country is published each week in the Weekly Epidemiological Record. These reports are usually broken down on a district, county or provincial basis for each week.

The Weekly Epidemiological Record, volumes 46 to 50 of 1970-74 comprised the main source of data. Published reports of specific cholera outbreaks from newspapers,[12] newsmagazines[13] and medical journals formed

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[12] The following newspapers were employed: New Nigerian (Kaduna, Nigeria), The Daily Times (Lagos, Nigeria), Gaskiya ta Fi Kwabo (Kaduna, Nigeria) and Fraternité Matin (Abidjan, Ivory Coast).

[13] in particular, West Africa and Africa Digest.

a secondary source of data. The date of onset or of the first reported case from each political unit was recorded as well as the weekly reports of cases and deaths.

The data, particularly those on the number of cases and deaths, are subject to various limitations. Four of these, which are discussed below, are lack of information on symptomless carriers, size of the reporting political units, failure to report the presence of cholera, and inaccurate information on the number of cases and deaths.

(a) Lack of information on symptomless carriers:

Cholera characteristically has up to 100 asymptomatic carriers for every patient with clinical symptoms.[14] Unless there is very extensive bacteriological investigation of the general population, these asymptomatic carriers remain unrecognized. The carrier state may exist in a location and subsequently spread to other locations with no clinical case developing.[15] There have been very few published studies focusing on the incidence of cholera carriers in Africa.

(b) Size of the reporting units:

The data published in the Weekly Epidemiological Record are typically at the district, county or provincial level. However, the

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[14] W.H. Mosley, "Epidemiology of Cholera", in Principles and Practice of Cholera Control. Geneva: WHO 1970, p.26.

[15] Joint ICMR-GWB-WHO Cholera Study Group, Calcutta, India. "Serological studies on cholera patients and their household contacts in Calcutta in 1968", Bull. WHO, vol.43 (1970c), pp.389-399, and G.I. Forbes, J.D.F. Lockhart, and R.K. Bowman, "Cholera and nightsoil infection in Hong Kong, 1966", Bull. WHO, vol.36 (1967), pp.367-373.

level of reporting is inconsistent. Some Nigerian states, such as Lagos provide a divisional breakdown of cholera incidence, while others give only provincial or state totals. After the initial epidemic phase, Ivory Coast provided only a monthly national total. The large areal extent and population of some political units presents another problem. For example, Sokoto Province, Nigeria had a 1963 population of 3,193,019[16] and an area of 36,477 square miles. When cholera is reported from "Sokoto Province", we have a very imprecise idea of its actual location and extent. Another problem arises from the wide range of size of populations and areas among the political units of a study area. Other things being equal, a large division has a greater probability of being infected than a small one. Thus, the diffusion pattern mapped is largely a function of the areas and populations of the political units in the study area.

(c) Failure to report the presence of cholera:

The lists of cholera infected territories were frequently incomplete. In some cases, cholera was denied or not reported for such reasons as prestige and minimizing economic and social disruption. Guinea refused to admit the presence of cholera, and left the World Health Organization in protest after WHO unilaterally announced the outbreak.[17] Imported cases of cholera originating in Tunisia were reported from

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[16] Nigeria Year Book 1971. Lagos: Times Press, 1971, p.27.

[17] West Africa, Sept.5, 1970, p.1039.

Great Britain, Germany and Italy late in 1973,[18] although Tunisia was reporting no cholera at the time, probably to protect its tourist industry. Especially in rural areas lacking medical facilities, individual cases or even fairly sizeable outbreaks doubtlessly remained undiagnosed and unreported.

(d) Inaccurate information on the number of cases and deaths:

The actual number of cases and deaths far exceeded the reported number in several countries. Félix has estimated that the actual incidence of cholera in Nigeria, Mali and Dahomey was five to ten times the reported incidence.[19] This sometimes resulted from deliberate underreporting to minimize adverse publicity. Several countries, including Liberia, Ivory Coast, Mauritania and certain Nigerian states furnished data to World Health Organization only sporadically, with the result that the apparent incidence of cholera is much smaller than the actual number of cases. Many thousands of cases occurred which were not reported to health officials. This was especially true in rural areas lacking medical facilities. In a study of attitudes toward cholera in Trengganu, Malaysia, Chen found that many cholera cases and deaths were not reported

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[18] Weekly Epidemiological Record, vol.48 (1973), p.358 and pp.363-364.

[19] H. Félix, "Le développement de l'épidemie de choléra en Afrique de l'Ouest", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971C), pp.565 and 572.

because of skepticism about Western treatment methods and a fear that the authorities would interfere with traditional burial ceremonies and the functioning of markets.[20] Similar fears of the consequences of reporting cases may well have existed in Africa. For example, the first cholera cases in Togo were among Ghanaian fishermen. They returned at night to Ghana with cholera patients and victims' bodies to avoid being caught by Togolese authorities.[21]

Another source of error in the Weekly Epidemiological Record morbidity and mortality data is the lack of a standardized procedure on bacteriological confirmation of cases. In some territories, for example, Lagos State, Nigeria, all suspected cases were bacteriologically checked. Only 26% of the suspected cases were cholera-positive.[22] Many territories reported only the number of suspected cases. Analysis and comparison of morbidity data submitted with such inconsistent criteria would be difficult and the results very tenuous.

The major limitations of the data for the study have been considered in this section. The limitations apply in particular to morbidity and mortality data. Where cholera was diagnosed and reported

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20 P.C. Chen, "Socio-cultural aspects of a cholera epidemic in Trengganu, Malaysia", Trop. Geog. Med., vol.23 (1971), p.298.

21 J.J. D'Almeida et al., "L'épidemie de choléra au Togo", Médecine d'Afrique Noire, vol.20, Nos.8-9 (1973), p.639.

22 Dada, B.A.A., "First sixteen weeks of cholera in Lagos State", J. Soc. Hlth. Nigeria, vol.6,No.3 (1971B), p.133.

the deviation of the reported date of onset from the actual date is thought to be relatively small. There are certain countries and regions where relatively good morbidity and mortality are available, especially where detailed local epidemiological studies have been made and published. However, the nature of data from the Weekly Epidemiological Record does restrict the possibilities for statistical analysis. Data limitations should also be kept in mind when evaluating conclusions reached in the study.

Interpretation of Diffusion Arrows

Several of the maps in the thesis are designed to show the major cholera diffusion routes. In the majority of cases, there are no published descriptions of particular outbreaks to provide conclusive evidence of the origin of infection. Where such evidence was unavailable, a subjective selection was made of the probable origin of infection. By considering the distribution and seriousness of outbreaks in the three or four weeks preceding and the amount of interaction between these previously-infected places and the newly-infected location, a "most probable" origin was selected. Estimating the amount of interaction involves consideration of several factors, including total population, urban population, transport routes, diffusion barriers, urban function, the flow of trade goods, seasonal population movements, ethnic distribution, international frontiers, and intervening opportunities.

It is thought that the maps convey a relatively accurate picture of the actual diffusion system. Published reports describing the sequence of infection in particular areas seem to confirm that actual diffusion patterns bear a close resemblance to the expected pattern based on interaction. However, it is important that individual arrows should be interpreted probabilistically rather than deterministically.

CHAPTER 2

CHOLERA

Causative Agent

Cholera is caused by vibrio cholerae, curved rod-like bacteria of the genus vibrio.[1] Cholera vibrios possess a complex chemical structure. On the basis of their antigenic properties, they may be divided into three serotypes. These are called Inaba, Ogawa and Hikojima.[2] Evidence of spontaneous sero-conversion exists. For example, during the West African cholera Ogawa outbreak, vibrios of the Inaba serotype were isolated in Southern Cameroun, Chad, Niger, and Liberia.[3] The East African epidemic began as Inaba, but Ogawa vibrios were later found in Uganda and Western Kenya.[4] The several types of non-agglutinable or NAG vibrios present special problems of classification.[5] They possess most of the characteristics of agglutinable vibrios, but are usually non-pathogenic. NAG vibrios are commonly found during the final stage of a cholera outbreak, probably developing as a result of mutation.

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[1] R. Sakazaki, "Classification and characteristics of vibrios", in Principles and Practice of Cholera Control, Geneva: WHO, 1970, p.33.

[2] R. Pollitzer, Cholera, Geneva: WHO, 1959, pp.232-234.

[3] B. Cvjetanovic and D. Barua, "The seventh pandemic of cholera", Nature, vol.239 (Sept.15, 1972), p.138, and Félix, H., "Le choléra Africain", Médecine Tropicale, vol.31, No.6 (1971D), p.627.

[4] D. Barua, "The global epidemiology of cholera in recent years", Proc. Royal Soc. Med., vol.68, No.5 (May, 1972), p.425.

[5] D. Felsenfeld, The Cholera Problem, St. Louis: W.H. Green, 1967, pp.29-30.

Cholera El Tor is a biotype of classical vibrio cholerae. It was first found in 1906 among pilgrims returning from the Mecca pilgrimmage at the El Tor health control station in Sinai.[6] It was next found in Celebes (Indonesia). From 1937 to 1949 there were occasional minor outbreaks of cholera-like diarrhoea in Celebes.[7] Isolated occurrences of cholera El Tor were also reported from Djakarta, Singapore, and India.[8] Prior to 1962, it was considered a separate disease, known as paracholera El Tor, because of its less severe clinical symptoms. It was reclassified by the World Health Organization in 1962 as vibrio cholerae biotype El Tor and hence became subject to international regulations.[9] Concern about cholera El Tor had grown as a result of its spread in a pandemic form from Celebes commencing in 1961. The designation vibrio cholerae biotype El Tor was supported by Hugh as a result of his investigations which showed it to be identical to classical vibrio cholerae in thirty positive and twenty negative characteristics, and different in only minor ways.[10] Differences in the epidemiology and clinical picture of classical and El Tor cholera will be discussed later in this chapter.

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[6] M. Khan, K.J. Bart and Z. Haq, "The changing pattern of cholera in East Pakistan: the appearance of Vibrio cholerae", J. Pakistan Med. Assoc., vol.20, No.2 (1970), p.43.

[7] C.E. DeMoor, "Paracholera (El Tor)", Bull. WHO, vol.2 (1948), pp.5-17.

[8] O. Felsenfeld, "Some observations on the cholera El Tor epidemic in 1961-62", Bull. WHO, vol.28 (1963), p.289.

[9] D. Barua, and B. Cvjetanovic, "Cholera during the period 1961-70", in Principles and Practice of Cholera Control, Geneva: WHO, 1970, p.16.

[10] R. Hugh, in Proceedings of the Cholera Research Symposium, PHS publication 1328, 1965.

Clinical Symptoms

Cholera infection usually results from the ingestion of an infected medium such as food or water. The illness may take various forms along a continuum from carriers displaying no clinical symptoms, to mildly symptomatic cases, to very severe, fatal illness. Various factors govern the severity of illness. These include the level of immunity of the victim, his general health and nutritional status, the quantity of vibrios ingested, the virulence of the vibrio strain, the length of the incubation period, and the speed of diagnosis and treatment. The incubation period is generally one to five days, but is occasionally longer.[11]

Cholera patients may display a variety of clinical symptoms. Typically, there is a sudden onset of effortless and profuse diarrhoea with a "rice water stools" appearance. Vomiting occurs among about 80% of the cases.[12] As the diarrhoea and vomiting continue, signs of dehydration and disturbed electrolyte balance become apparent. These symptoms include clammy skin, sunken eyes and cheeks, a drop in body temperature and blood pressure, rapid respiration, cramps, and marked saline depletion.[13] The progression of symptoms usually takes about five to twelve hours. If these symptoms are untreated, mortality may be 60% or higher. However, relatively simple treatment involving rehydration with saline solution

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[11] Pollitzer, op. cit., pp.684-686.

[12] Felsenfeld, op.cit., 1967, p.58.

[13] Felsenfeld, op.cit., p.58.

and the use of antibiotics to control vibrio multiplication may reduce the mortality to almost nil.[14]

Cholera does not assume the severe clinical picture outlined above in the majority of those infected. Most are carriers who have been infected by cholera vibrios and who may in turn reinfect the environment but who have no clinical symptoms such as vomiting or diarrhoea. Carriers may be classified as incubatory carriers, convalescent carriers, and symptomless carriers.[15] Incubatory carriers are persons who become infected and later develop clinical symtoms. Convalescent carriers are persons excreting vibrios during the period of their recovery from clinical cholera. Symptomless carriers generally excrete vibrios, but do not develop any clinical symptoms.

The carrier state normally lasts from one to five days. However, the carrier state with El Tor infections lasts, on the average, three times as long as that of classical vibrio cholera.[16] Long term, intermittent carriers have been found. The most publicized long term carrier is "Cholera Dolores" from the Philippines, who has continued to

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[14] W.H. Mosley, K.J. Bart, and A. Sommer, "An epidemiological assessment of cholera control programs in rural East Pakistan", Int. J. Epid., 1972, p.10.

[15] R. Sinha et al., "Cholera carrier studies in Calcutta in 1966-67", Bull. WHO, vol.37, No.1 (1967), pp.90-91.

[16] W.H. Mosley, K.J. Bart, and A. Sommer, op.cit., pp.9-10.

excrete vibrios intermittently for over ten years.[17] A comprehensive program of testing for cholera carriers and environmental contamination in Calcutta located several households in which the cholera infection seemed to be ingrained, repeatedly reappearing in a carrier or the environment despite the complete absence in the area of clinical cholera cases.[18]

The percentage of asymptomatic carriers is much higher with infections of vibrio cholerae biotype El Tor than with classical vibrio cholerae. There are normally five to ten carriers for every clinical cases of classical cholera, and 25 to 100 carriers for every El Tor case.[19] This "iceberg effect" of numerous unrecognized infections is a major problem of cholera control, especially in outbreaks of cholera El Tor.

Immunity

Some degree of immunity may be obtained as a result of vaccination, recovery from an attack of cholera, and through the barrier effects of saliva, gastric acidity, and bacterial competition in the intestine.[20]

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17 J.C. Azurin, et al., "A long-term carrier of cholera - Cholera Dolores", Bull. WHO, vol.37 (1967), pp.745-750.

18 R. Sinha et al., "Role of carriers in the epidemiology of cholera in Calcutta", Indian J. Med. Res., vol.56, No.7 (1968), pp.964-978.

19 W.H. Mosley, "Epidemiology of cholera", in Principles and Practice of Cholera Control, Geneva: WHO, 1970, p.26.

20 J. LeViguelloux, and J.C. Doury, "Épidémiologie du choléra moderne", Médecine Tropicale, vol.31, No.1 (1971),p.27.

Vibrio cholerae is acid-sensitive, and it would appear that stomach acids must be at least temporarily neutralized before cholera can survive.[21] Stomach acid is governed by physiology, diet, and intestinal parasites. Persons with heavy worm loads and inadequate diets are more susceptible to cholera.[22] Eating a high protein meal may also neutralize stomach acid.[23]

Cholera antibody levels rise after the ingestion of cholera vibrios. Even inapparent infections may cause a significant rise in antibody titer.[24] The longer the period of excretion the higher the probability of a significant increase in antibodies. In cholera-endemic areas, there is a linear increase of vibriocidal antibody with age.[25] Therefore, children are most frequently affected when cholera occurs in an endemic area, while in newly-infected areas, adults are commoner among the early victims.[26]

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[21] S. DeMaeyer-Cleempoel, "Quelques réflexious à propos du choléra...", Arch. Belg. Méd. Soc., vol.30, No.6 (1972), p.381. An experiment showed that after neutralization of stomach acid among volunteer subjects, ingestion of 10^6 cholera vibrios caused heavy diarrhoea, while among those with un-neutralized stomach acid a dose of 10^{11} vibrios caused only light diarrhoea.

[22] C.K. Wallace, et al., "The 1961 cholera epidemic in Manila, Republic of the Philippines", Bull. WHO, vol.30 (1964), p.808.

[23] C.C.J. Carpenter, "Pathogenesis and pathophysiology of cholera", in Principles and Practice of Cholera Control, Geneva: WHO, 1970, pp. 53-54.

[24] W.E. Woodward and W.H. Mosley, "The spectrum of cholera in rural Bangladesh. II Comparison of El Tor Ogawa and classical Inaba infection", Am. J. Epidem., vol.96 (1972), pp.345-349.

[25] W.H. Mosley, A.S. Benenson, and R. Barui, "A serological survey for cholera antibodies in rural East Pakistan", Bull. WHO, vol.38 (1968), pp.327-334. None of the sampled Pakistani children under one year had cholera antibodies, while 90% of the adults over 30 did. A control experiment in a non-endemic area (Czechoslovakia) showed no change in antibodies with age.

[26] W.M. McCormack, et al., "Endemic cholera in rural East Pakistan", Am.J. Epidem., vol.89 (1969), p.403.

Vaccination for cholera is a controversial subject. Available vaccines give only about 50% protection for three to six months.[27] While vaccination may reduce the number of severe cases, it has been claimed that there is no reduction in the number of vibrio excretors or the length of excretion.[28] Cost-benefit analysis has been used to show that, in most outbreaks, the small protection given by vaccination cannot economically justify its use. It is cheaper instead to treat clinical cases.[29]

Transmission Cycle

Man is the only known natural host of vibrio cholerae. The infection is maintained as a result of the establishment of a man-environment or person to person transmission cycle. The vibrios, which multiply in the gut of a carrier or cholera patient are excreted. Where environmental sanitation is poor, the vibrio-carrying feces may directly contaminate the soil, water, or food. The transmission cycle is completed when a new subject ingests the contaminated medium and becomes a carrier. Environmental contamination may also be indirect, such as vibrio transmission by

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[27] A.M. McBean et al., "Comparison of intradermal and subcutaneous routes of cholera vaccine administration", Lancet, Mar.4, 1972, p.528, and L. La Peyssonnie, "Chemioprophylaxie de l'infection cholérique: intérêt, espoirs, et limites", Médecine Tropicale, vol. 31 (1971B), p.127.

[28] La Peyssonnie, op.cit., p.128.

[29] D. Barua, "Cholera vaccination as a tool for cholera control", Bull.Soc. Pathol. Exotique, vol.64 (1971), pp.652-659.

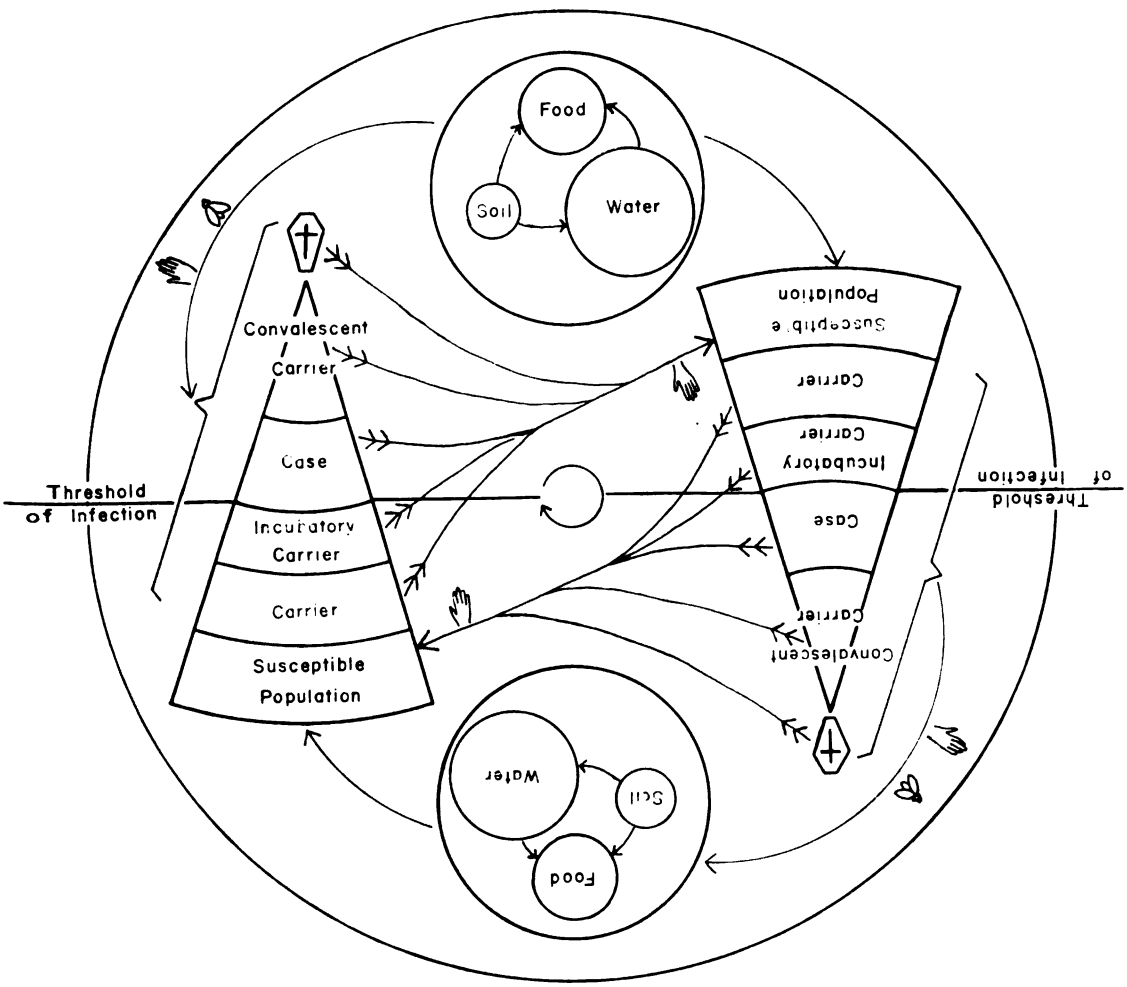
FIGURE 2

CHOLERA TRANSMISSION CYCLE

Cholera may spread directly from the infected to the susceptible population (person to person spread) or indirectly as a result of environmental contamination and subsequent ingestion of the infected medium by members of the susceptible population.

FIGURE 2

CHOLERA TRANSMISSION CYCLE



Source : Le Viguelloux & Doury, 1971

flies from contaminated nightsoil to food, or from contaminated nightsoil fertilizer to vegetables grown with it.[30]

Cholera may also be spread directly from person to person. This commonly occurs when a patient is being treated for cholera at home, or during the ritual preparation for burial of the body of a cholera victim.[31] Cholera vibrios may survive on the body or clothing of a cholera victim and be passed to the hands of anyone in close contact. These vibrios are then ingested as a result of hand-mouth contact or the contamination of food or water. Cholera patients excrete several liters each day including about 10^6 to 10^8 vibrios per milliliter.[32] Thus contacts of cholera victims run a considerable risk of infection.

The survival of cholera vibrios in the environment depends on the favorability of the transporting medium. Three important factors in survival are moisture content, temperature, and acidity. Cholera vibrios cannot survive in high temperatures, although they are resistant to cold.[33]

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30 J. Cohen, et al., "Epidemiological aspects of cholera El Tor in a non-endemic area", Lancet, July 10, 1971, pp.86-89. The article attributes the spread of cholera in Jerusalem in 1970 to the sale of nightsoil-infected vegetables.

31 Le Viguelloux and Doury, op.cit., pp.24-25.

32 Le Viguelloux and Doury, op.cit., p.19.

33 Felsenfeld, op.cit., 1967, p.32.

They are also sensitive to acid. A slightly alkaline medium, such as brackish lagoon water, is ideal.[34] El Tor vibrios usually remain viable for two to three weeks. Under ideal conditions, they may survive for two weeks or more in foods such as milk, seafood and cooked rice.[35] Vibrios usually die within hours on acidic fruit. Sunlight, drying, and ultra-violet light are all fatal to cholera vibrios.

In many areas with a long history of cholera occurrence, the disease displays a distinct seasonal pattern. However, areas with apparently identical seasonal climatic regimes may display different cholera regimes. For example, cholera in Dacca peaks late in the monsoon and disappears during the hot, dry season. The pattern in Calcutta is the opposite.[36] The two cities are located relatively close to each other in the Ganges delta.

Vibrio cholerae biotype El Tor survives for a longer period in the environment and under more adverse conditions than does classical vibrio cholerae.[37] As a result, El Tor outbreaks are harder to control, and endemicity is more common than with classical cholera.

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34 H. Wollf, "A quantitative approach to the epidemiology and control of cholera", Bull. Soc. Pathol. Exotique, vol.64, No.5 (1971), pp.583-584.

35 Felsenfeld, op.cit., Ch.2, 1967, p.33 and D. Barua, "Survival of cholera vibrios in food, water and fomites", in Principles and Practice of Cholera Control, Geneva: WHO, 1970, pp.29-31.

36 Mosley, op.cit., 1970, p.24.

37 A. Sommer and W.E. Woodward, "The influence of protected water supplies on the spread of classical/Inaba and El Tor/Ogawa cholera in rural East Bengal.", Lancet, Nov.11, 1972, pp.985-987.

Control Measures[38]

Cholera is a very adaptable disease which can survive in a variety of environments and be transmitted in a variety of ways. With cholera El Tor, the large incidence of symptomless carriers relative to symptomatic cases, and its ability to survive in adverse environments multiplies the problems of cholera control.

The key to control is environmental sanitation. Where there is adequate sewage and garbage disposal, it is improbable that a cholera transmission cycle could become established. However, modern sewage facilities are lacking in many large cities in the Third World, much less in the rural areas. Measures such as improved garbage collection, fly control, and construction of simple pit latrines are effective control measures. Sources of drinking water, whether wells, ponds or a piped water system may be protected by chlorination.

Restrictions of large gatherings for festivals or markets is frequently used as a control measure. A large gathering can easily promote an epidemic explosion by bringing carriers into contact with a large susceptible population from different locations. One of the earliest control measures was establishment of a travel cordon around an infected town or along an international boundary. These cordons have usually failed to stop travel and hence the dissemination of cholera.[39]

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[38] Extensive discussions of control measures are found in Pollitzer, op.cit., pp.893-987 and Felsenfeld, op.cit., 1967, pp.108-152.

[39] Pollitzer, op.cit., p.965.

Public education is a key element of a cholera control program. The public must be told about the symptoms of cholera and the importance of environmental sanitation in stopping its transmission.

Mass vaccination of the susceptible population is a popular control measure, largely because of it is an easily understood and visible approach. However, it is costly, limited in its effectiveness, and may instill a false sense of security in the population.

Constant surveillance and bacteriological examination of suspected cases and their contacts, water sources and nightsoil are also necessary. It is only through such surveillance that early recognition of an outbreak or the determination of the means of infection transmission is possible.

CHAPTER 3

HISTORY OF CHOLERA

Occurrence of Cholera Before 1817

Controversy surrounds early reports of cholera-like illnesses made by Greek, Indian, and Chinese physicians. There is no consensus on whether the descriptions are of cholera or other diseases with similar symptoms. Extensive reports of a cholera-like disease are found in the writings of the Indian physician Shushruta in the fifth century A.D.[1] The earliest definite evidence is from the journals of European travellers to India following Vasco da Gama's pioneering voyage in 1498. Gaspar Correa, in Lendas da India described an illness which are almost certainly cholera which had been observed in 1503 and 1543 in India.[2] There are many more well-documented, independent references to cholera outbreaks in various parts of the Indian sub-continent from 1503 to 1817.

Before the nineteenth century, cholera did not spread westward from India. However, whether or not it had moved eastward into China and the East Indies before 1817 is less certain.[3]

Nineteenth Century Cholera Pandemics

The year 1817 marked a crucial turning point in the history of cholera. The years 1815 and 1817 were exceptionally rainy, causing

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[1] L.M. Bhattacharji, et al., "Changing phases of cholera in India", Indian J. Med. Res., vol.52 (1964), p.751.

[2] R. Pollitzer, Cholera, Geneva: WHO, 1959, pp.12-13.

[3] Pollitzer, op. cit., pp.14-15.

floods and crop failures. The weather in 1816 was extremely hot and dry. During this period, cholera assumed such a violent form that it was thought to be a new disease.[4] It also spread out of the Indian sub-continent into South-East and East Asia and the Middle East. This marked the beginning of the first cholera pandemic. During the nineteenth century there were six pandemics in which cholera spread from country to country and continent to continent. In each case, the pandemics originated in the Indian sub-continent. After epidemic outbursts in the newly-infected countries, the infection tended to disappear for a few years until reintroduced.

Table 1 shows the extent of cholera infection during each pandemic and the approximate dates of the pandemics.[5]

Transportation routes and commercial centers played key roles in the dissemination of cholera during each pandemic. Caravans and sailing ships were especially important. Cholera reached the shores of the Mediterranean from China via the Central Asian caravan route during the second pandemic.[6] Mecca was repeatedly infected by caravans arriving from Persia.[7] In East Africa, caravans were responsible for bringing cholera from the Red Sea coast inland and south to the Somali coast.[8]

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[4] Pollitzer, op. cit., pp.17-18.

[5] Maps of the diffusion and extent of cholera during each pandemic are found in J. May, The Ecology of Human Disease, New York: MD publications, 1958, pp.39-43. A relatively detailed account of the occurrence of cholera and routes of diffusion is contained in Pollitzer, op.cit.,pp.17-48.

[6] May, op.cit., pp.38-40.

[7] J. Christie, Cholera Epidemics in East Africa, London, McMillan, 1876, p.79.

[8] Christie, op.cit., p.103.

TABLE 1

Extent of the First Six Cholera Pandemics

Pandemic	Date	Indian Sub-continent	SE Asia	East Asia	Middle East	S Europe	NW Europe	NE Europe	N Africa	E Africa	W Africa	N America	C America	S America
First	1817-23	x	x	x	x					x				
Second	1826-37	x	x	x	x	x	x	x	x	x		x	x	x
Third	1842-62	x	x	x	x	x	x	x	x	x		x	x	x
Fourth	1865-75	x	x	x	x	x	x	x	x	x	x	x	x	x
Fifth	1881-96	x	x	x	x	x	x	x	x	x	x	x	x	
Sixth	1899-1923	x	x	x	x	x		x						

Note: an "x" denotes the occurrence of cholera during a pandemic in a particular geographical area.

Sailing routes also served as cholera diffusion routes. Among the more important of these were the routes from India to the East India, Arabia and East Africa, from Europe to the Americas, and between the numerous ports of the European perimeter from the Baltic to the Mediterranean and Black Seas. Typically, the infection would be introduced at a sea port and then would spread inland. With the coming of the Railway Age in Europe and North America after the mid-nineteenth century, the infection diffused along railway lines from ports to their hinterlands.

The most notorious cholera diffusion center was Mecca. The annual Hajj attracted tens of thousands of pilgrims from all parts of the Muslin World. Because of the lack of understanding of the causes or control of cholera, it could easily be introduced by any group of pilgrims. Pilgrims from other countries would then contact the infection and disseminate it on their homeward journeys. Between 1830 and 1912 there were thirty-three major cholera epidemics originating in Mecca.[9]

Pyle[10] has studied the patterns of cholera diffusion in the United States in 1832, 1849 and 1866. In 1832, the United States was still a frontier country lacking a well-developed transportation network. Cholera spread from Quebec and New York along the East Coast and the major Eastern river systems. Distance from the points of introduction

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9 May, op.cit., p.52.

10 G.F. Pyle, "The diffusion of cholera in the United States in the Nineteenth century", Geog. Analysis, vol.1, No.1 (1969), pp.59-75.

was strongly correlated with the date of onset. By 1866, the United States had become a highly urbanized nation with a dense railway network. Cholera quickly spread from New York and New Orleans along railways to the largest towns and then diffused to nearby smaller centers. City size, rather than distance had become the key variable. The 1849 epidemic showed elements of both the 1832 and 1866 patterns.

Mortality was very heavy during all of the nineteenth century cholera pandemics. For example, in 1866 there were over 90,000 cholera deaths in Russia, 115,000 in Austria-Hungary, 130,000 in Italy, 30,000 in Belgium and 20,000 in the Netherlands.[11]

During the nineteenth century two discoveries were made which had great significance for the possibilities of cholera control. The first was John Snow's discovery in 1849 that cholera was spread by water and that epidemic control could be achieved by stopping the distribution of infected water.[12] Robert Koch made the second important discovery in 1883, namely that cholera is caused by bacteria.[13] These two findings formed the basis for subsequent cholera control strategies.

Cholera in Africa during the Nineteenth Century

East Africa

The cholera epidemics of East Africa prior to 1870 have been described in considerable detail in an 1876 book by Christie.[14] Cholera

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[11] Pollitzer, op.cit., p.33.

[12] J. Snow, Snow on Cholera, New York: Commonwealth Fund, 1936.

[13] May, op.cit., p.35.

[14] Christie, op.cit.

was first noted in East Africa in 1821 when a slave ship arrived from Zanzibar via Muscat and Oman with cholera on board.[15] The second cholera invasion was in 1836-37 following the Mecca pilgrimmage. Dhows engaged in the coastal trade carried the infection from Arabia along the East African coast to the Somali Coast, Zanzibar and Mocambique.[16] It was also carried along caravan routes into East and Central Africa.

During the Third Pandemic (1852-68) the West African coast was infected several times. Abyssinia was reached in 1853, 1855 and 1858. Speke referred to the frequent occurrence of cholera in Central Africa (Uganda) in his journal of 1858-59.[17] Cholera arrived in Zanzibar from Oman on the ship Maryland on November 15, 1865.[18] Very heavy mortality was reported in Zanzibar and the adjacent mainland around Kilwa. The diary of the explorer, Burton, included a very vivid picture of the impact of the epidemic. It also illustrates the prevailing lack of understanding of cholera epidemiology, particularly the need for environmental sanitation, ensured massive mortality.

> "The smooth, oily water was dotted with fragments and remnants of humanity; black and brown when freshly thrown in; patched, mottled and party coloured when in a state of half-pickle; and ghastly white, like scalded pig when the pigmentum nigrum had become thoroughly macerated."[19]

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15 Christie, op.cit., pp.97-98.

16 Christie, op.cit., pp.102-103.

17 Christie, op.cit., p.107.

18 Christie, op.cit., p.111.

19 Christie, op.cit., p.114.

Christie's most detailed account is of the 1864-71 East African epidemic. The pattern of diffusion is shown in Figure 3. Cholera was introduced to Berbera in November, 1864 by a ship which had probably come from Bombay. It then moved along caravan routes to the coast in the vicinity of the Jub River. In May, 1865 Jidda and Mecca were infected by a ship coming from Singapore via India. The most severe epidemic in the history of Mecca ensued.[20] From Abyssinia in 1865, it gradually spread south, reaching the Masai country in 1869. The Masai spread it to the south during raids on neighboring peoples. A party of traders carried the infection from Laikepya to the coast near Zanzibar in 1869. Of the 150 men in the party, only seven survived the trip.[21] Cholera's arrival in the headwaters of the Congo beyond Lake Tanganyika was recorded by Livingstone.[22] Livingstone apparently contacted cholera infection himself, but recovered. Traders carried the disease south to Lake Nyassa and on to the Moçambique coast in 1870. It spread from Moçambique north and south along the coast and to the Comoro Islands and Madagascar.

In 1869, Zanzibar had been infected and sustained heavy mortality. In a single year, cholera claimed 70,000 victims in Zanzibar.[23] Ships took water on board in Zanzibar from infected ponds and wells near the harbour before setting sail.[24] As a result, cholera was reintroduced to the East African coast from Cabo Delgado to the Horn in 1870.

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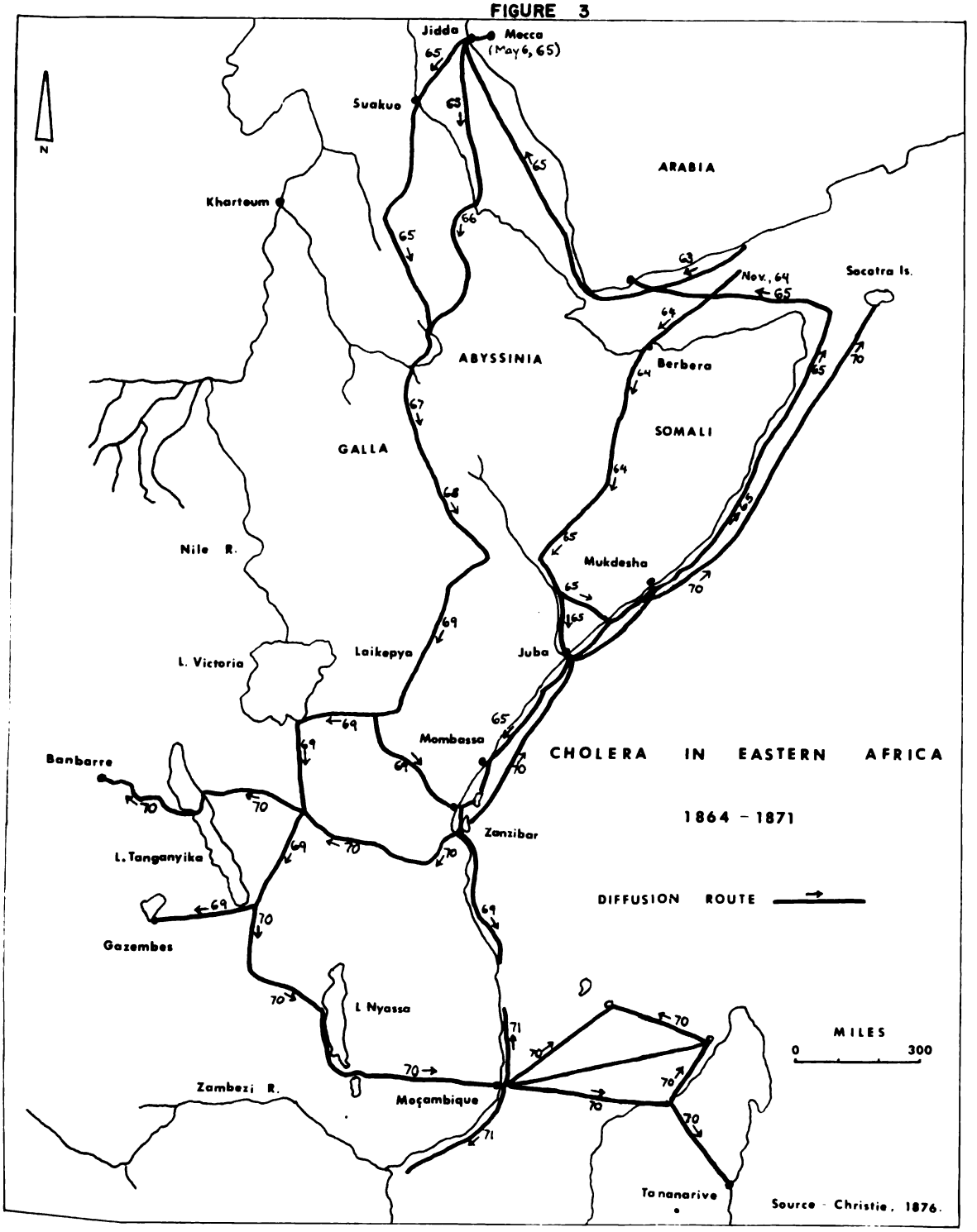
[20] Christie, op.cit., p.150.
[21] Christie, op.cit., p.228.
[22] Christie, op.cit., p.245.
[23] Pollitzer, op.cit., p.340.
[24] Christie, op.cit., p.420.

FIGURE 3

CHOLERA IN EASTERN AFRICA 1864-1871

This is a slightly simplified version of a map appearing in an 1876 book by James Christie on cholera empidemics in East Africa to 1870. It shows the southward movement of cholera from Berbera and Northern Abyssinia along caravan routes, eventually reaching Zanzibar, the headwaters of the Congo and Moçambique. Trading ships also carried cholera along the East African coast. The numbers on the map represent the year of onset in a particular area.

FIGURE 3


Jidda
Mecca
(May 6, 65)
Suakue
Kharteum
ARABIA
Socotra Is.
Nov., 64
Berbera
ABYSSINIA
SOMALI
GALLA
Nile R.
Mukdesha
L. Victoria
Laikepya
Juba
Mombassa
Banbarre
Zanzibar
L. Tanganyika
Gazembes
L Nyassa
Zambezi R.
Moçambique
Tananarive
CHOLERA IN EASTERN AFRICA
1864 - 1871
DIFFUSION ROUTE
MILES
0
300
Source - Christie, 1876.

Several of Christie's conclusions are noteworthy. He emphasized the importance of highways of human travel for the propagation of the disease. He noted that cholera dissemination peaked during the dry season, the rainy season being a "time of isolation".[25] He also stressed the importance of fixed local foci of dissemination, particularly where the cholera generative agent was discharged into more or less stagnant water. He concluded that because cholera is essentially a disease of filth; "a community can secure immunity by sanitary measures alone."[26]

Between 1879 and 1900, only the Red Sea and Somali coasts reported cholera in East Africa.

North Africa

North Africa was subjected to repeated introductions of cholera during the Second through Fifth Pandemics. There were two main sources of infection.[27] Cholera was frequently introduced to the Alexandria-Cairo area by returning pilgrims from Mecca. It typically spread up the Nile into Upper Egypt and sometimes the Sudan. Tripolitania (Libya), Tunisia, Algeria, and Morocco were also infected via Egypt by epidemics originating in Mecca. The second source of infection was Mediterranean trading ships calling at North African ports. The 1867 epidemic in Tunisia was blamed on Tunisian smugglers returning from Sicily.[28]

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[25] Christie, op.cit., p.502.
[26] Christie, op.cit., p.505.
[27] Pollitzer, op.cit., p.28 and p.32.
[28] Pollitzer, op.cit., p.34.

Heavy mortality was frequently reported, especially from populous areas in Upper Egypt, Algeria and Morocco. For example, 80,000 died in Algeria in 1867 in an epidemic of French origin, and over 58,500 cholera victims were recorded in 1883 in Egypt.[29]

West Africa

Cholera was introduced to Podor along the Senegal River by caravans from Morocco in 1868.[30] It then spread to St. Louis, Bathurst (Gambia) and Bissau (Portuguese Guinea) in 1869. A reported 1,700 of the 5,000 residents of Bathurst died of cholera.[31] It again arrived in the Senegal Valley by caravans in 1893-94.[32]

Worldwide Incidence of Cholera from 1923-1960

Between 1923 and 1960 cholera ceased to exhibit any pandemic tendencies. During this period there were no cholera deaths in Europe.[33] In South-East and Eastern Asia the number of cases declined, and after 1945 only sporadic, minor outbreaks were reported. There was a steady decline in the number of deaths due to cholera in the Indian sub-continent. Between 1930 and 1949 there were 3,580,000 cholera deaths in

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[29] Pollitzer, op.cit., p.38.

[30] Pollitzer, op.cit., p.34.

[31] R. Schram, A History of the Nigerian Health Services, Ibadan, 1971, p.102.

[32] D.J.M. Mackenzie, "Cholera, its nature, management, and prevention", S. Afr. Med. J., vol.45 (1971), p.2.

[33] Pollitzer, op.cit., p.59.

India and Pakistan, compared to 8,050,000 deaths from 1900 to 1919.[34] The geographical extent of cholera in the Indian sub-continent also became more confined. Cholera was restricted to a few endemic foci from which it periodically spread to neighboring regions. The major endemic areas were concentrated around the lower Ganges, Bramaputra and Hooghly Rivers.[35] The periodic epidemic outbursts from the endemic areas have been found to be highly correlated with the staging of large fairs and festivals.[36]

The most important cholera epidemic outside the Indian Sub-continent between 1923 and 1960 was in Egypt between September 18 and December 5, 1947. There were 32,978 recorded cases and 20,474 deaths.[37] The infection is thought to have been introduced in conjunction with the movement of military personnel from India to Egypt.[38] The first reported cases were at the village of El Korein which was the home of many workers from a nearby British military base. A major date fair, attracting thousands of traders from all parts of Egypt coincided with the cholera outbreak. Either through the contamination of the dates or the infection

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[34] Calculated from annual mortality data in Pollitzer, op.cit., p.58.

[35] S. Swaroop, "Endemicity of cholera in India", Indian J. Med. Res., vol.39, No.2 (1951A), pp.141-184.

[36] S. Swaroop, and M.V. Raman, "Endemicity of cholera in relation to fairs and festivals in India", Indian J. Med. Res., vol. 39 (1951), pp.41-49, and A.C. Banjera, "Note on cholera in the United Provinces (Uttar Pradesh)", Indian J. Med. Res., vol.39, No.1 (1951), p.31.

[37] Pollitzer, op.cit., p.63.

[38] S. Abdou, "The cholera epidemic in Egypt: Mode of spread", Lancet, vol.2 (Nov.8, 1947), pp.696-698.

of some of the traders themselves, cholera was disseminated in a matter of days throughout the Nile Delta. Smaller outbreaks were reported along the Nile as far south as Qena.[39] Because of its mode of introduction and transmission, its rapid dissemination and quick disappearance, and the very large number of cases and deaths the 1947 Egyptian epidemic remains somewhat enigmatic.

Cholera El Tor and the Seventh Pandemic

The unexpected development after 1960 of a new cholera pandemic, involving a local and apparently mild biotype of vibrio cholerae from the Celebes forced a sudden reassessment of the consensus that cholera was a geographically-limited and declining disease.

Cholera El Tor began to spread from its home territory in Southern Celebes following movements by troops and Celebes' Chinese population and after boats from the area had participated in a regatta in Sarawak.[40] The progression of the Seventh, or El Tor pandemic is shown in Figure 4.[41]

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39 "Trend of cholera in Egypt", WER, vol.22 (1947), pp.383-385.

40 D. Barua, and B. Cvjetanovic, "Cholera during the period 1961-70", in Principles and Practice of Cholera Control. Geneva: WHO, 1970, p.5.

41 Several accounts of the dissemination of cholera during the seventh pandemic exist, including Barua and Cvjetanovic, op.cit., pp.15-21, J. Gallut, "Actualité du choléra. Évolution des problèmes épidémiologiques et bactériologiques", Bull. Inst. Pasteur, vol.66, No.2 (1968), p.219-248, and S. Mukerjee, "Recent incidence of cholera outside India", Indian J. Med. Res., vol.52 (1964), pp.771-776.

FIGURE 4

TREND OF THE GLOBAL SPREAD OF CHOLERA 1960-1974

Classical vibrio cholerae was superseded after 1960 by cholera El Tor. Originating in the Celebes, cholera El Tor had by 1974 advanced westward through Southern Asia and the Middle East into Africa and limited parts of Europe. The map shows the migration of the "cholera front" from 1961 to June, 1974.

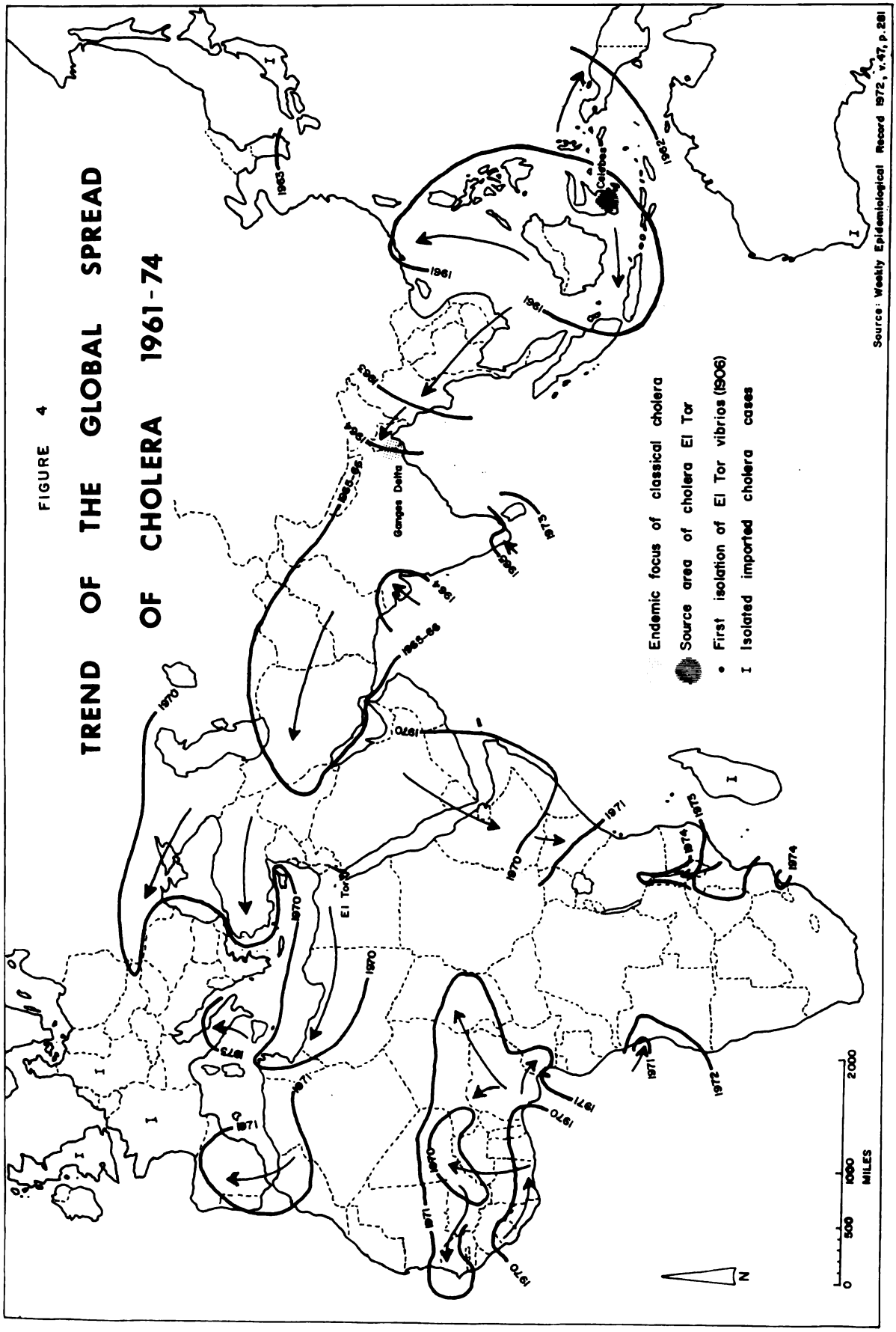
TREND OF THE GLOBAL SPREAD OF CHOLERA 1961-74
Endemic focus of classical cholera
Source area of cholera El Tor
• First isolation of El Tor vibrios (1906)
I Isolated imported cholera cases
Source: Weekly Epidemiological Record 1972, v.47, p.281
Ganges Delta
Celebes
El Tor
0 500 1000 2000
MILES
N

FIGURE 4

During 1961 cholera reached several other islands of the Indonesian archipelago, Kwangtung Province in China, Hong Kong, Macao, and the Philippines. Taiwan and West Irian were infected in 1962. In 1963, Malaya, Cambodia, Vietnam, Thailand, Burma, South Korea and Japan experienced cholera outbreaks. The most serious epidemics were in Indonesia, the Philippines and South Vietnam.

Late in 1963 cholera El Tor first appeared in the Indian subcontinent at Chittagong.[42] By 1966, cholera El Tor had completely eliminated classical vibrio cholerae in India,[43] although classical cholera remained dominant in East Pakistan (Bangladesh). Since 1968 there has been a resurgence of classical cholera in India and an increase in the frequency of El Tor isolation in Bangladesh.[44] The two strains now appear to be coexisting in both countries.

Cholera El Tor moved into West Pakistan in 1964. Afghanistan, Iran and limited areas in USSR, near the Iranian frontier were infected in 1965, followed by Iraq in 1966. The western cholera front remained relatively stable until 1970 when it advanced into all countries in the Middle East, as well as the Soviet Black Sea coast, eastern Czechoslovakia, North Africa, East Africa, and West Africa. The sudden appearance and rapid diffusion of cholera in West Africa was probably the most noteworthy of these developments.

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[42] S. Mukerjee et al., "A new trend in cholera epidemiology", Brit. Med. J., vol.2 (1965), p.837.

[43] S. Mukerjee, "A decade's tracking of cholera with bacteriophage", Indian J. Med. Res., vol.55, No.4 (1967), pp.310-311.

[44] K.J. Bart, Z. Huq, et al., "Sero-epidemiologic studies during a simultaneous epidemic of infection with El Tor Ogawa and classical Inaba vibrio cholerae", J. Infect. Dis., vol.121 (1970), pp.S17-S24.

During 1971, cholera affected all countries in West Africa from Chad to Senegal, with the exception of Guinea-Bissau and Guinea (Conakry). In East Africa, cholera occurred in Ethiopia, Somalia, Kenya, Uganda, and the French Territory of Afars and Issas. In North Africa, Morocco and Algeria reported cholera for the first time in 1971. A new focus developed in Angola in 1971. Moçambique, Malawi, Rhodesia and Tanzania have reported cholera for the first time in 1973 and 1974. There are indications that cholera El Tor may be becoming endemic in certain areas of Africa, in particular in West Africa.

Health officials have been somewhat disturbed by the appearance of cholera in Europe since 1970. Because of the higher standards of living and environmental sanitation, and because of fifty years of cholera-free status, Europe had been thought to be immune from infection.[45] However, European USSR and Eastern Czechoslovakia had cholera outbreaks in 1970, Spain and Portugal in 1971, and Italy in 1973. Portugal is reported to have had 827 cases of cholera between April and July, 1974.[46] In addition isolated imported cases have been reported from such countries as France, Germany, Britain and Canada. Although the epidemics have been confined to the poorest areas of Southern Europe, their size and the mere fact that cholera has been able to establish itself in Europe has been cause for concern.

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[45] "Tip of the iceberg shows", Nature, vol.227 (1970), p.766.

[46] Radio station CKOX, Woodstock, Ontario. Noon news report, August 3, 1974.

CHAPTER 4

CHOLERA EPIDEMIC ZONES IN AFRICA

Following the initial establishment of cholera in Africa in 1970, the disease has spread to thirty-two countries. These countries form five discrete clusters or infection foci, located in West Africa, North Africa, East Africa, Angola and South-Eastern Africa (Figure 5). The major axes of diffusion in each of these areas are described in this chapter.

West Africa

On August 18, 1970, Guinea summoned World Health Organization officials because of the outbreak of a serious epidemic suspected to be cholera.[1] Guinean students returning from USSR are suspected to have brought it from the Black Sea coast.[2]

Coastal Sierra Leone and Liberia first reported cholera in the period of September 19-25 and October 2-8, 1970, respectively.[3] The infection was carried along the coast to the vicinity of Abidjan by

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[1] L. La Peyssonnie, "Le choléra, An II", Médecine Tropicale, vol.31, No.6 (1971D), p.615.

[2] L. La Peyssonnie, "Acquisitions récentes en matière d'épidémiologie et de prophylaxe du choléra en Afrique", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971A), p.644.

[3] All dates of onset and numbers of cases and deaths have been obtained from the Weekly Epidemiological Record, vols.45-49 (1970-1974) unless footnotes indicate another source.

FIGURE 5

CHOLERA-AFFECTED AREAS IN AFRICA 1970-1974

Between 1970 and 1974 cholera has established five discrete fronts in its invasion of Africa. These are in North Africa, West Africa, East Africa, Angola, and South-Eastern Africa. Long distance air travel played an important role in the initial establishment of three of the fronts. Despite diffusion over long distances in each of the five cholera zones, and despite the fact that thirty-two countries have had cholera outbreaks, roughly 80% of the continent has yet to experience any cholera cases.

FIGURE 5

CHOLERA - AFFECTED AREAS IN AFRICA

1970 - 74

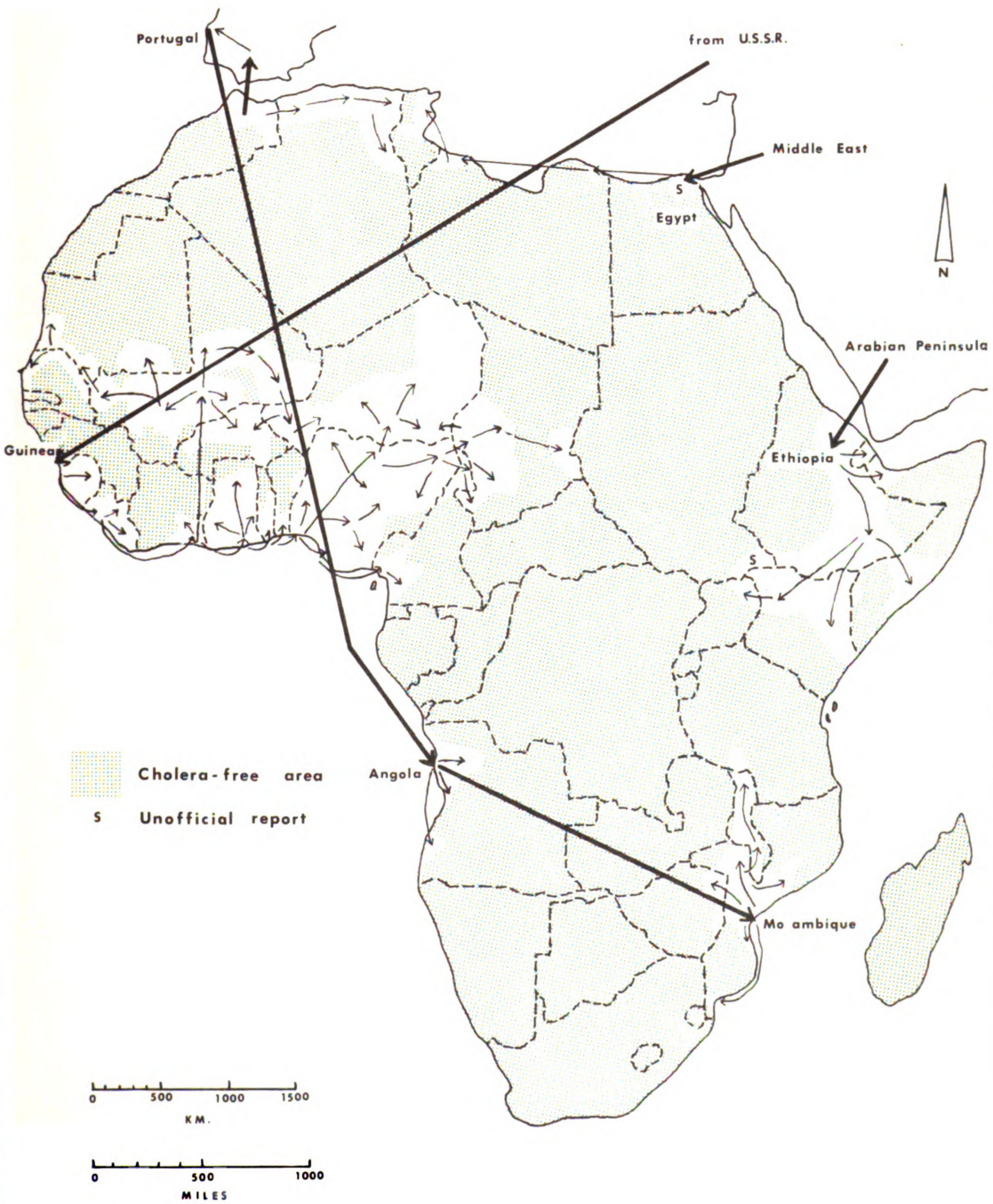


FIGURE 6

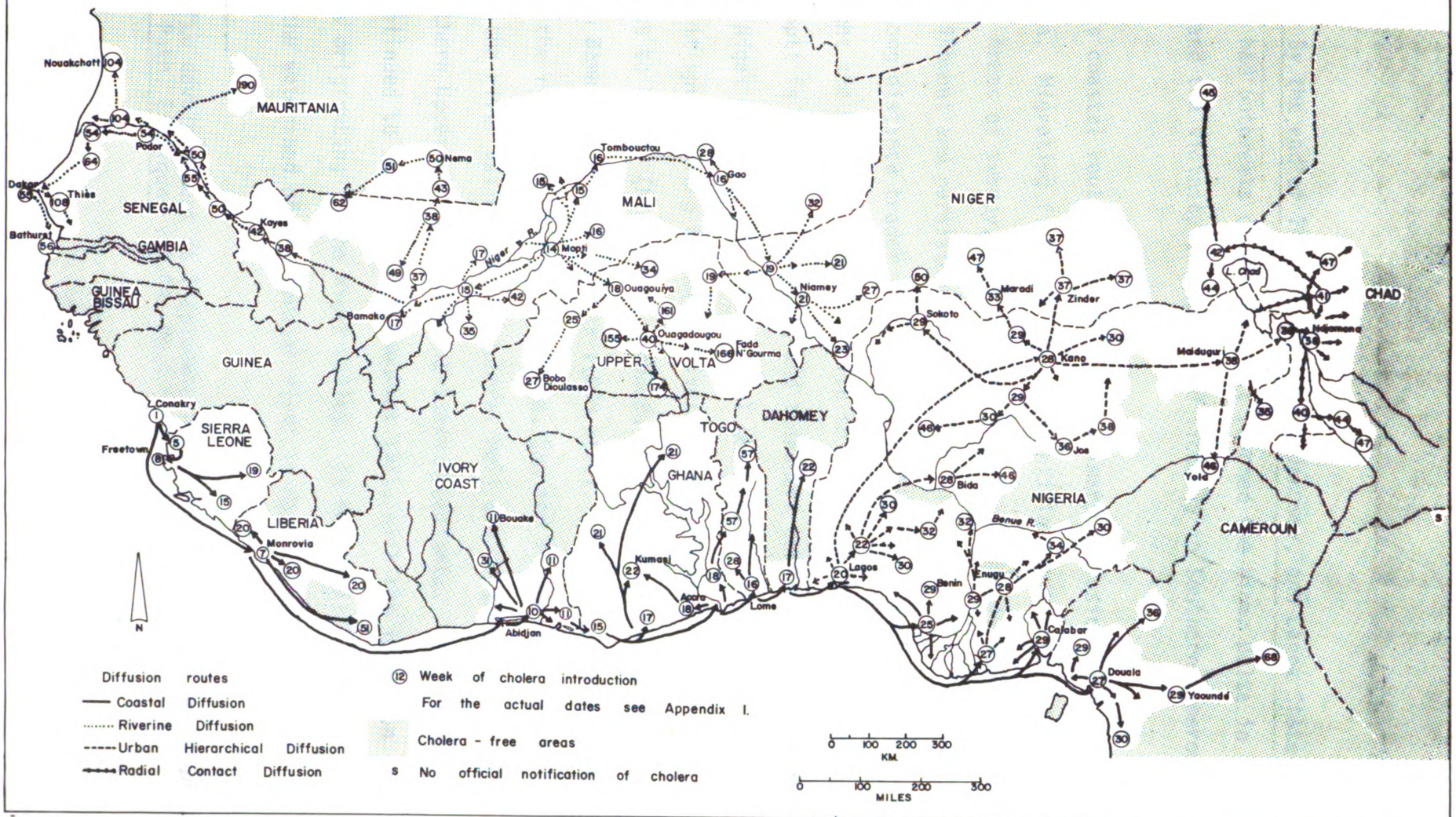
DIFFUSION OF CHOLERA IN WEST AFRICA

After the introduction of cholera at Conakry in mid-August, 1970, it spread along the coast as far as Southern Cameroun by February, 1971. There were three other types of diffusion. One was the riverine epidemic centered on Mopti (Mali) and extending from central Niger in the East to the Atlantic coast of Senegal and Mauritania in the West. Nigeria and South-Central Niger experienced urban-based hierarchical diffusion. Radial contact diffusion occurred in the vicinity of Lake Chad.

Guinea-Bissau was the only country to completely escape infection. Substantial areas in the Middle Belt have remained cholera-free.

FIGURE 6

DIFFUSION OF CHOLERA IN WEST AFRICA



mid-October. By the end of 1970, Ghana, Togo, Dahomey, and Lagos State (Nigeria) had had outbreaks of cholera. It reached the Niger Delta in late January and the vicinity of Douala and Victoria in Southern Cameroun in February.

This coastal route constituted the primary diffusion axis in West Africa. Migrating fishermen were responsible for the spread of the disease. Areas of swampy, lagoon coast were particularly susceptible to the establishment and persistence of cholera.

A long distance trader from Abidjan who was also a cholera carrier brought the disease 1,500 km. north to Mopti (Mali) on November 5, 1970.[4] Mopti is the main marketing and transport center of the Inland Delta of the Niger River. After the initial outbreak in Mopti on November 19, it spread along the Niger River, west to Bamako in four weeks and east to Niamey (Niger) via Timbuctu and Gao in eight weeks. The distance from Bamako to Niamey is over 1,000 miles. From important staging points along the river, such as Mopti, Segou, Gao and Niamey, the disease moved out of the river valley into peripheral Mali and Western Niger, and into Northern Upper Volta and South-Eastern Mauritania. New upland districts continued to be affected throughout 1971, with the infection almost always originating in the Niger Valley. In May, 1971, cholera spread into the watershed of the Senegal River in Kayes division (Mali).

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[4] H. Félix, "Le développement de l'épidémie de choléra en Afrique de l'Ouest", Bull. Soc. Path. Exotique, vol.64, No.4 (1971C), p.566.

It moved steadily down the Senegal Valley and reached the Atlantic Coast late in August, 1971.

The riverine diffusion route, which has been described in the preceding paragraph, featured rapid linear, contact diffusion along the channel of the Niger-Senegal valley and a slower penetration of valley-fringing uplands.

The third diffusion axis involves the spread of cholera from Lagos throughout Nigeria and South-Central Niger. Unlike Mali, Nigeria has a well-developed urban hierarchy and relatively good road and rail communications. A hierarchical diffusion pattern developed, with the infection moving along major transportation arteries between the largest cities and from the important regional cities to smaller places having more localized influence. The main secondary centers of diffusion were Ibadan, Kano, Zaria, Maiduguri, Sokoto, Enugu, Onitsha and Port Harcourt in Nigeria, and Maradi and Zinder in Niger.

From Maiduguri (Nigeria) cholera was carried to portions of Chad, Cameroun and Niger located near Lake Chad. The disease became more virulent here than anywhere else in Africa. A total of 12,255 cases and 3,448 deaths (28.1% mortality) were reported from Chad up to October 1, 1970.[5] The severity of the epidemic precipitated the flight of people from affected areas. As a result, the infection spread outward like a wave from the centers of introduction toward Eastern Chad and south to the border of the Central African Republic along the Logone and Chari Rivers.

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[5] J. Gallut, "La Septième Pandémie cholérique 1961-1966, 1970...", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971C), p.555.

North Africa

Very little is known about the early stages of the introduction of cholera to North Africa. The August 1, 1970 New York Herald-Tribune reported that cholera had claimed 1,500 victims in Alexandria and several in Cairo.[6] However, no cholera outbreak was reported to the World Health Organization by Egyptian authorities.

The only cases from Libya were reported on August 23, 1970. There were a total of twenty-eight cases at Tobruk near the Egyptian border and at Tripoli, over 700 miles further east.

Smugglers crossing the Libya-Tunisia border have been blamed for the spread of cholera to Tunisia.[7] The outbreak, occurred between August 22 and November 11, 1970 east of Tunis in Cape Bon. It was centered in the fishing village of Kéliéba. Thirty-nine cases were recorded in Tunisia, and a further fifty-two health carriers were found through bacteriological investigation.[8]

No further occurrence of cholera was reported from North Africa until July, 1971. This new outbreak was from Nador Province, Morocco. Nador is located on the Mediterranean coast near the Algerian Border. How the infection reached Nador without appearing in Algeria is unknown.[9]

Cholera quickly moved from Morocco into Western Algeria. Tlemcen was the first town reached. Cholera spread eastward to Oran

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[6] Gallut, op.cit., p.553.

[7] M.S. Ben Rachid et al., "Étude épidémiologique du foyer de choléra de Kéliéba (Tunisie)", Arch. Inst. Pasteur Tunis, vol.48, No.3 (1971), pp.255-292.

[8] Ben Rachid, op.cit., p.269.

[9] B. Mered et al. ("L'épidémie de choléra en Algérie en 1971", Arch. Inst. Pasteur Algérie, vol.49 (1971), pp.85-97) have described the comprehensive program of cholera surveillance established in Algeria after the beginning of the Tunisian outbreak. They found no trace of cholera prior to July 1971.

FIGURE 7

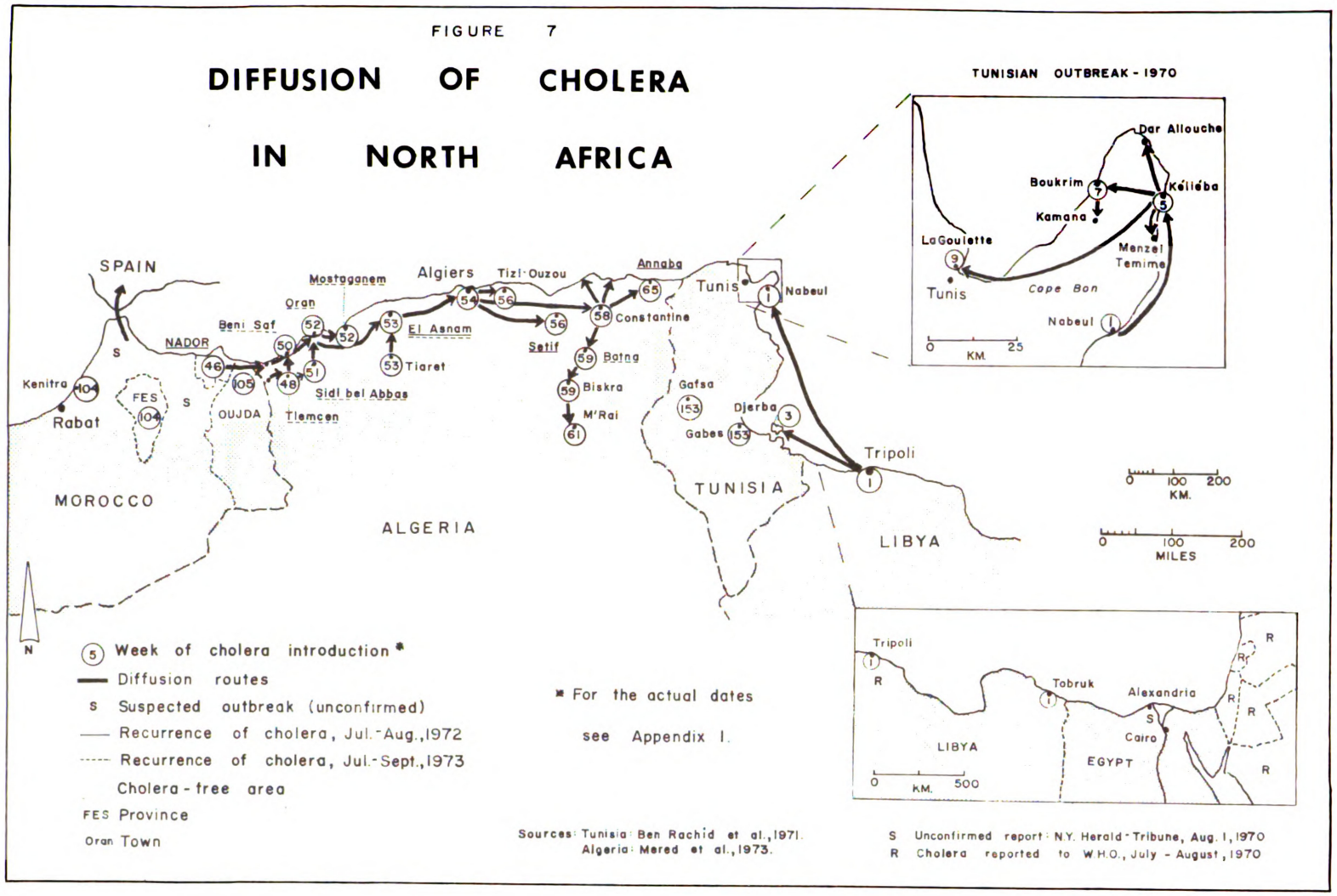
DIFFUSION OF CHOLERA IN NORTH AFRICA

In 1970, cholera occurred in Tunisia and Libya following a widespread outbreak in the Middle East and unconfirmed reports of cholera in Egypt. An inset map shows the distribution of cholera cases in the Cap Bon (Tunisia) area.

In 1971 cholera spread from Morocco into Algeria and Spain. Outbreaks have been virtually confined to high population density areas near the coast. Places experiencing recurrences of cholera in 1972 and 1973 have been underlined.

FIGURE 7

DIFFUSION OF CHOLERA IN NORTH AFRICA



El Asnam and Algiers in August and Sétif, Constantine and Biskra in September. A total of 1,332 clinical cases and 417 symptomless carriers were recorded in the Algerian epidemic, which ended on November 30, 1970.[10]

In 1972, Algeria reported twenty-seven cases from four locations in Central and Eastern Algeria on July 31. Four provinces in Northern Morocco between the Algerian border and the Atlantic Ocean (Kenitra) were reported as infected in mid-August, 1971. During 1973, cholera was reported from two Tunisian districts in July and seven Algerian divisions in September.

The diffusion of cholera in North Africa has not followed any easily discernable pattern. The infrequent notification of cases and notification from widely separated areas make interpretation of the diffusion patterns difficult. The sequence of reported cases suggests that cholera moved over 400 miles from Alexandria (Egypt) to Tobruk (Libya), followed by jumps of 700 miles to Tripoli, 400 miles to Cape Bon (Tunisia) and 800 miles to Nador (Morocco). The time interval between the Cape Bon and Nador occurrences exceeded six months. The 1972 and 1973 recurrences of cholera exhibit no obvious spatial pattern.

Three key questions about cholera in North Africa concern the degree of under-reporting of cases by health officials, the means of transmission, and its mode of survival between the apparently infrequent outbreaks. Failure to report all outbreaks would be a plausible explanation for the confusing patterns of cholera occurrence. An alternate explanation would involve the endemic survival of the vibrio through a

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[10] Mered, op.cit., p.91.

man-environment transmission cycle with periodic diffusion from these endemic foci. In both the Algerian and Tunisian epidemics, water supplies were strongly linked to the spread of cholera. Thirty-seven cholera vibrios and ninety-two NAG vibrios were found in 1,019 Algerian water samples examined.[11] Infected seafood has also been implicated as a result of the 1973 Italian cholera outbreak which was linked to the importation of North African mussels.[12]

East Africa

A serious outbreak of cholera occurred in the South-Western Arabian Peninsula in August, 1970, although it was officially reported to be bacillary dysentry.[13] The first East African cases were reported from Harar, Shoa and Wollo Provinces of Ethiopia for the period of November 6-10, 1970. The actual date of introduction was probably somewhat earlier. This initial report was the only one made by Ethiopian authorities. However, there were repeated importations of cholera from Wollo, Harar and Sidamo Provinces of Ethiopia into the French Territory of Afars and Issas, Somali Republic, Northern Kenya and probably South-Western Sudan between November, 1970 and July, 1972. This would indicate the presence of a widespread, prolonged, and relatively serious epidemic in Southern and South-Western Ethiopia.

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[11] Mered, op.cit., p.93.

[12] Weekly Epidemiological Record, vol.48 (1973), p.358.

[13] B. Carteron, "Le choléra dans le Territoire Français des Afars et des Issas", Médecine Tropicale, vol.33, No.3 (1973), p.229.

FIGURE 8

DIFFUSION OF CHOLERA IN EAST AFRICA

Cholera entered Ethiopia from the Arabian Peninsula in November, 1970. It spread from Ethiopia into French Territory of Afars and Issas, Somali Republic, Northern Kenya, Eastern Uganda, and is suspected to have spread to Southern Sudan.

The epidemic was primarily among highly mobile nomadic peoples occupying desert and semi-desert areas.

The French Territory of Afars and Issas was subjected to repeated introductions of cholera from Ethiopia and Somalia. Prior to the fortieth week these were all successfully contained. The introductions of cholera documented in an article by Carteron and Artus have been shown in the inset map.

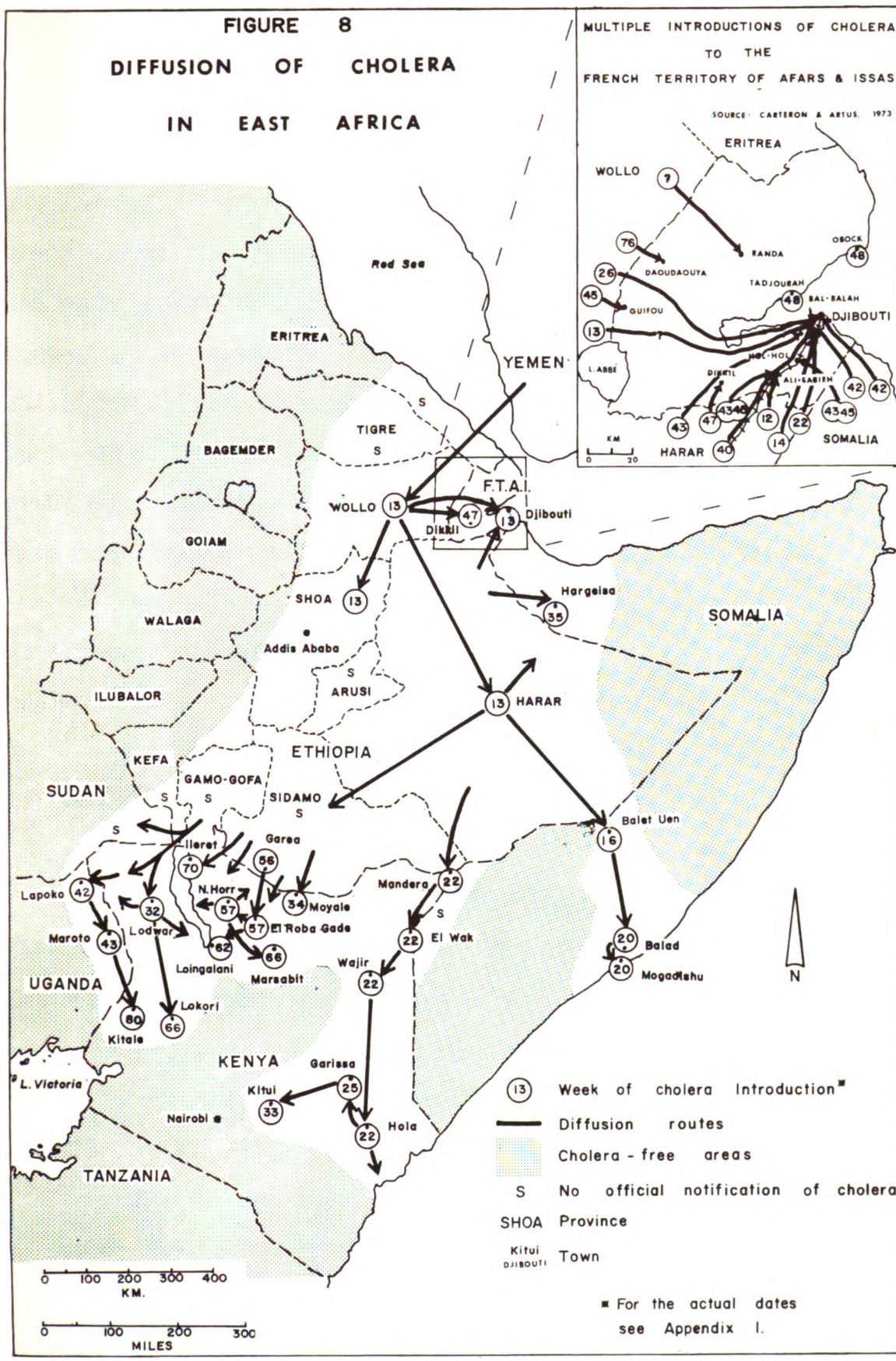
FIGURE 8
DIFFUSION OF CHOLERA
IN EAST AFRICA
MULTIPLE INTRODUCTIONS OF CHOLERA
TO THE
FRENCH TERRITORY OF AFARS & ISSAS
SOURCE: CARTERON & ARTUS, 1973
ERITREA
WOLLO
RANDA
DAOUDAOUYA
GUIFOU
OBOCK
TADJOURAH
BAL-BALAH
DJIBOUTI
DIKHIL
ALI-SABIEH
L. ABBÉ
HARAR
SOMALIA
KM
0 20
Red Sea
YEMEN
ERITREA
TIGRE
BAGEMDER
GOIAM
WALAGA
ILUBALOR
KEFA
GAMO-GOFA
SIDAMO
SHOA
Addis Ababa
ARUSI
ETHIOPIA
SUDAN
F.T.A.I.
Djibouti
Hargeisa
SOMALIA
HARAR
Balet Uen
Baled
Mogadishu
Ileret
Garea
N.Horr
Moyale
El Roba Gade
Lodwar
Lapoko
Maroto
Loingalani
Marsabit
Mandera
El Wak
Wajir
UGANDA
Lokori
Kitale
KENYA
Garissa
Kitui
Nairobi
Hola
L. Victoria
TANZANIA
N
Week of cholera Introduction*
Diffusion routes
Cholera - free areas
S No official notification of cholera
SHOA Province
Kitui Town
DJIBOUTI
* For the actual dates
see Appendix I.
0 100 200 300 400
KM.
0 100 200 300
MILES

There were numerous independent introductions of cholera into the French Territory of Afars and Issas[14] (inset of Figure 7). Most of these importations of cholera involved travellers arriving from Ethiopia. The early importations were all discovered in time to prevent the development of a local epidemic. However, a local epidemic commenced on June 22, 1971. Between June 22 and August 18 there were 282 cases and sixty-two deaths in Djibouti and smaller numbers in other towns in frequent contact with Ethiopia and Somalia.[15] There were sporadic occurrences resulting from fresh importations of infection between August, 1971 and July, 1972.

Cholera was introduced to Belet Uen and Mogadiscio in Central Somali Republic from Harar (Ethiopia) in December, 1970. The Hargeisa region in Northern Somalia reported cholera in April, 1970.

Northern Kenya was infected along three main routes.[16] The earliest introductions were in the Hola-Garissa area along the Tana River and at Wajir and Mandera in the extreme North-Eastern corner of Kenya in January, 1971. The second importation was west of Lake Rudolf in Turkana District in June. It appears that the disease entered from Ethiopia via Southern Sudan. There were 457 cases in Turkana District up

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[14] B. Carteron and J.C. Artus, "Épidémiologie du choléra au Territoire Français des Afars et des Issas", Médecine Tropicale, vol.33, No.3 (1973), pp.235-248.

[15] Carteron and Artus, op.cit., pp.240-241.

[16] Descriptions of cholera in Northern Kenya have been written by P.S.V. Cox, "Cholera in Northern Kenya", E. Afr. Med. J., vol.49, No.6 (1972), pp.440-447, by E.N. Mngola, "Cholera epidemic in Kenya", E. Afr. Med. J., vol.49, No.2 (1972), pp.151-158, and by J.M.D. Roberts, "Cholera detection and surveillance in Kenya", E. Afr. Med. J., vol.49, No.2 (1972), pp.159-162.

to October, 1971.[17] The third axis of penetration was east of Lake Rudolf in the North Horr and Marsabit areas during September and October, 1971. The infection was introduced by a party of travellers from Garea (Ethiopia) who walked along a dry river bed to North Horr, infecting at least three major water holes along the way.[18] There have been several other localized introductions across the Ethiopian frontier into such towns as Mandera, Sololo, Sabarei and Ilaret. None of these towns apparently became the source of further diffusion. The last report of cholera from Kenya to the World Health Organization was in April, 1972.

Cholera has not been officially reported from the Sudan, but Félix reports its presence there.[19] The Turkana (Kenya) epidemic appears to have resulted from an importation of cholera from Southern Sudan.

Between June 1 and mid-July, 1971, a total of 757 cases were recorded in Karamoja Province in North-Western Uganda. After entering Uganda from Turkana District (Kenya), it moved along a southerly route parallel to the Kenya border, extending as far to the south as Kitale (Kenya) near Mount Elgon.

Cholera diffusion in East Africa followed the paths of movement of nomadic herders and trading caravans. It was the highly mobile nomadic population which was primarily affected and was responsible for carrying

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[17] Roberts, op.cit., p.161.

[18] Cox, Op.cit., pp.440-441.

[19] Félix, op.cit., 1971C, p.572.

the disease from place to place.[20] A severe drought in this desert and semi-desert region forced many nomads to travel longer distances than usual in search of water and pasture, and caused a concentration of the population around the remaining water holes.[21] This increased travelling and concentration of population both facilitated the establishment and spread of cholera. Because water holes are unprotected and the water is alkaline, it is an ideal survival medium for vibrio cholerae. The mobility of the nomads, low density of population, inaccessibility of the area and absence of cooperation in cholera control between neighboring countries made the control of cholera in East Africa very difficult.[22]

Angola

The first reported case of cholera in Angola was in the capital city of Luanda on December 29, 1971. Determining the source of the infection is problematical. Portugal would seem to be the logical source, as Angola is a Portuguese colony, and because Portugal was experiencing a cholera epidemic. However, the Inaba serotype was found in Angola and Ogawa in Portugal.[23] Serotype conversion is sufficiently rare that the chance of it occurring in the particular individual carrying the infection to Angola is remote. It is possible that the infection may

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[20] R.M. Prothero, in Migrants and Malaria (London: Longmans, 1964, pp.63-78) has documented the difficulties of malaria control in this area due to pastoral movements.

[21] Mngola, op.cit., p.153.

[22] Mngola, op.cit., p.154.

[23] B. Cvjetanovic and D. Barua, "The Seventh Pandemic of cholera", Nature, vol.239 (Sept.5, 1972), p.138.

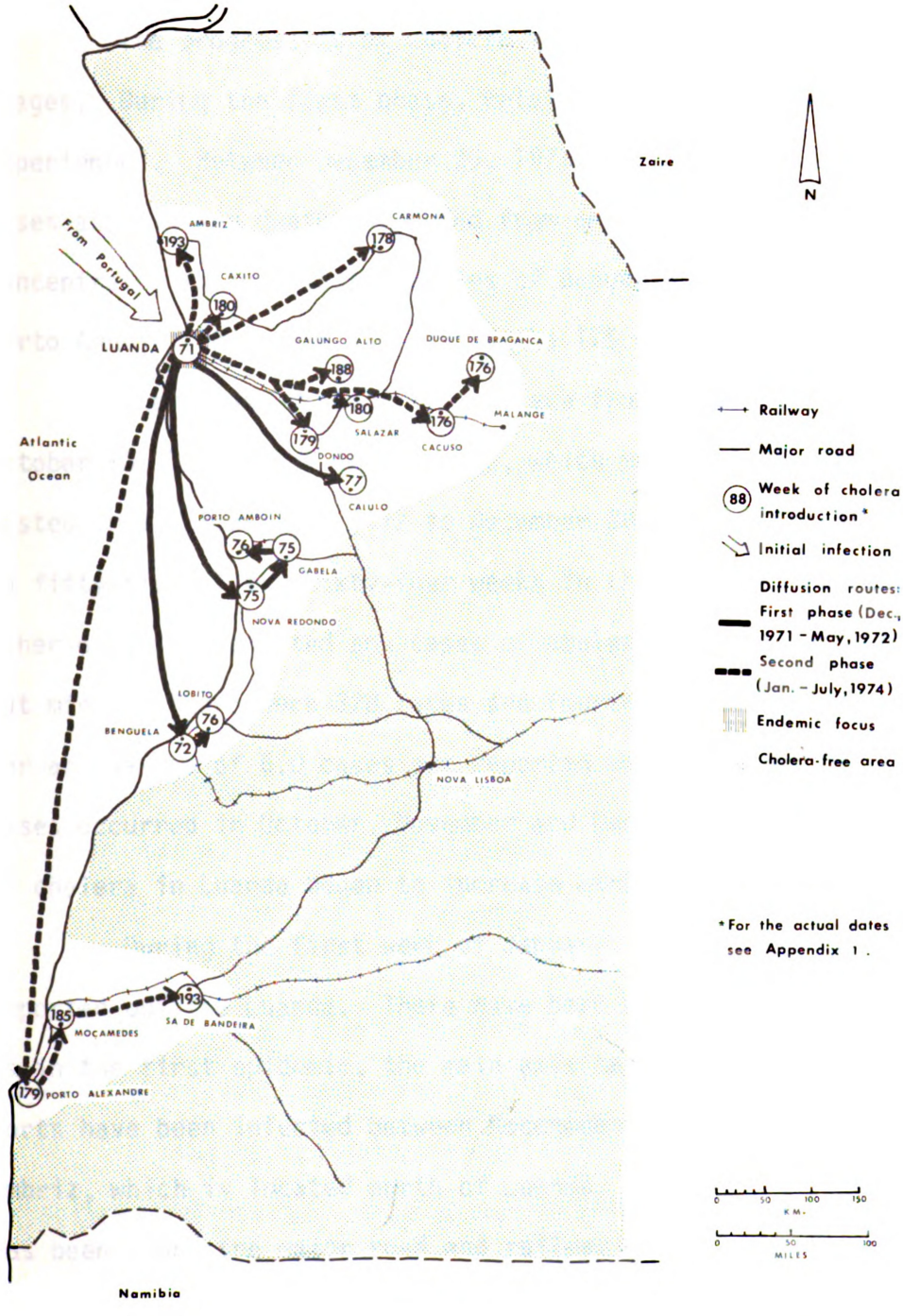
FIGURE 9

DIFFUSION OF CHOLERA IN ANGOLA

Cholera was introduced to Luanda at the end of December, 1971. During the first phase, it spread to six other locations, mostly other ports. The infection disappeared between May and October, 1972. From October, 1972 to December, 1973 a low-profile, persistent infection occurred in Luanda, but other places were cholera-free. A second epidemic phase began at the end of December, 1973. Cholera has since spread along the Luanda-Malange railway and along the coast north and south of Luanda.

FIGURE 9

DIFFUSION OF CHOLERA IN ANGOLA



have been introduced by a carrier from East Africa or India, areas which had the Inaba serotype.

The progression of cholera in Angola may be divided into four stages. During the first phase, relatively mild epidemic cholera was experienced. Between December 29, 1971 and May 1, 1972, there were 179 cases and sixteen deaths reported from seven districts. The outbreak was concentrated in the coastal cities of Benguela, Lobito, Nova Redondo and Porto Amboin. Benguela had 129 of the 179 cases and ten of the deaths.

There was no report of cholera from Angola between May 1, and October 13, 1972. The third phase, which may be called the endemic phase, lasted from October 13, 1972 to December 30, 1973. Cholera was reported in fifty-five of the sixty-four weeks in this period from Luanda. No other district reported any cases of cholera. The infection was persistent but minor. There were 328 cases and fourteen deaths in the endemic period, for an average of 6.0 cases per reported week. Almost half of the 328 cases occurred in October, November and December, 1973 when the incidence of cholera in Luanda began to increase markedly.

During the first week of January, 1974, cholera was again reported outside Luanda. There have been two main axes of diffusion. As in the first epidemic, the main axis has been along the coast. Eight ports have been infected between Moçamedes in Southern Angola and Ambriz, which is located north of Luanda. The second axis of diffusion has been along the major road and railway east of Luanda. The second epidemic phase has been relatively minor outside of Luanda, with a total

of 140 cases and five deaths to June 9, 1974. However, there were 574 cases and seven deaths reported from Luanda in the first twenty-two weeks of 1974. This represents an average of 26.1 cases per week.

There are some indications of a seasonal pattern of cholera occurrence in Angola. Both epidemics began in the last week of December, in 1971 and 1973. The endemic period in 1972 began in October, and it was in October, 1973 that the incidence of cholera in Luanda began to increase. The onset of epidemic cholera at the end of December may be related to an increase in interregional travel during the Christmas season. October is the end of the dry season at Luanda, which may account for the increased incidence of cholera in 1972 and 1973.

South-Eastern Africa

South-Eastern Africa is the term used to designate areas of Moçambique, Malawi, Eastern Zimbabwe and Southern Tanzania which have been infected with cholera El Tor between September, 1973 and July, 1974.

During the week of September 16-22, 1973, a new cholera outbreak was reported from the port of Beira in central coastal Moçambique. The infection was probably imported from Luanda (Angola) by air. Like Angola, Moçambique is a Portuguese colonial territory.

Diffusion took place along several routes. One axis was established within two weeks along the Beira-Zimbabwe railway in Manica and Chimoio districts. Cholera also spread north along the Beira-Malawi

FIGURE 10

DIFFUSION OF CHOLERA IN SOUTH-EASTERN AFRICA

Commencing in Beira in mid-September, 1973, cholera has to date spread through Central and Southern Moçambique, Eastern Zimbabwe and Malawi. Southern Tanzania first reported cases in June, 1974.

Railway lines appear to have played an important role in the diffusion patterns. The movements of Moçambique and Zimbabwe liberation forces are suspected to be involved in the diffusion of cholera in Tete Province (Moçambique) and adjoining Zimbabwe.

The epidemic continues to invade new areas as of mid-1974. Zambia, Swaziland and South Africa are countries threatened with infection because of their location close to the epidemic front.

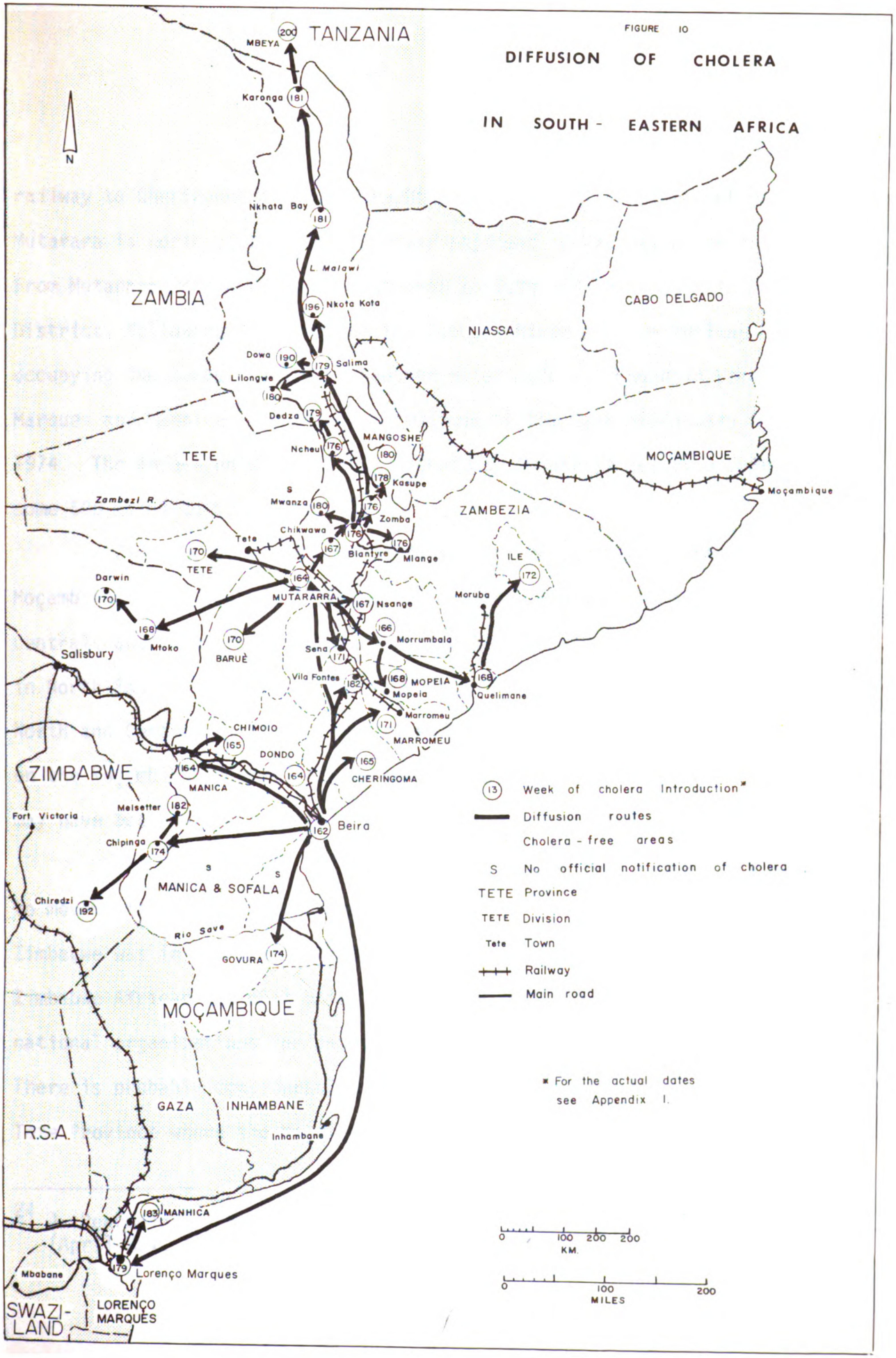


FIGURE 10
DIFFUSION OF CHOLERA
IN SOUTH - EASTERN AFRICA
TANZANIA
MBEYA
200
Karonga
181
Nkhata Bay
181
L. Malawi
ZAMBIA
196
Nkota Kota
NIASSA
CABO DELGADO
Dowa
190
Salima
179
Lilongwe
180
Dedza
179
MANGOSHE
Ncheu
176
180
TETE
MOÇAMBIQUE
178
Kasupe
Moçambique
S
Zambezi R.
Mwanza
180
176
Zomba
ZAMBEZIA
Chikwawa
176
Tete
167
176
Blantyre
Mlange
170
TETE
ILE
Darwin
164
172
170
MUTARARRA
167
Nsanga
Moruba
168
Mtoko
166
170
Morrumbala
Salisbury
BARUÈ
Sena
171
Vila Fontes
162
168
MOPEIA
168
Quelimane
Mopeia
Marromeu
171
CHIMOIO
MARROMEU
165
DONDO
ZIMBABWE
164
165
CHERINGOMA
164
MANICA
Melsetter
182
Fort Victoria
Beira
162
Chipinga
174
S
S
MANICA & SOFALA
Chiredzi
192
Rio Save
GOVURA
174
MOÇAMBIQUE
GAZA
INHAMBANE
R.S.A.
Inhambane
183
MANHICA
Mbabane
179
Lorenço Marques
SWAZI-
LAND
LORENÇO
MARQUES
13 Week of cholera Introduction*
Diffusion routes
Cholera - free areas
S No official notification of cholera
TETE Province
TETE Division
Tete Town
Railway
Main road
* For the actual dates
see Appendix I.
0 100 200 200
KM.
0 100 200
MILES

railway to Cheringoma and Mutarara Districts by the first week of October. Mutarara is north of the Zambezi River adjacent to the Malawi border. From Mutarara, cholera moved north-west to Tete and south-east to Marromeu District, following the trend of the Zambezi River and the railway line occupying the Zambezi Valley. Another major outbreak began in Lorenço Marques and Manhica in Southern Moçambique in the week of January 6-14, 1974. The infection was probably introduced by air or sea from Beira, some 500 miles to the north.

Zimbabwe's eastern and north-eastern borders are shared with Moçambique. Cholera has affected these border regions in Northern, Central, and Southern Zimbabwe. The first cases were reported from Mtoko in North-Eastern Zimbabwe. The fact that cholera was reported in both North and Central Zimbabwe before it had been reported from districts on the Moçambique side of the border suggests that the Moçambique epidemic may have been more extensive than what has been reported.

The introduction of cholera into Zimbabwe may have been related to movements of Zimbabwe liberation troops. The first outbreak in Zimbabwe was in the area with the most intensive liberation activity. The Zimbabwe African National Union (ZANU) sent requests to numerous international organizations for assistance in combating the epidemic.[24] There is probably considerable movement across the Moçambique border into Tete Province where the Moçambique Liberation Front (FRELIMO) is very active.

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[24] J. Pearpoint, "Cholera crisis reviewed", The CUSO Forum, vol.2, No.3 (April-May, 1974), p.24.

Cholera had appeared near the Malawi border in the first week of October, 1973. On October 24 it was reported from Nsanje and Chikwawa in Southern Malawi. These two locations reported a total of 332 cases in the first seven weeks. However, it was only on December 31, 1973 that it began to spread to other locations in Southern and Central Malawi. The key to this widespread diffusion was apparently the infection of Malawi's former capital city, Zomba, on December 31, 1973. By January 24, it had spread to eight other locations in Southern and Central Malawi. The infection followed the shore of Lake Malawi to Nkhata Bay and Karonga in Northern Malawi. The initial cases were reported early in March, 1974 in Northern Malawi.

Cholera has also crossed the Tanzanian border north of Lake Malawi. Five cases and two deaths were reported on June 6, 1974 from Kyella District in Mbeya Region.

As of early July, 1974, cholera was established in four countries in South-Eastern Africa. The infection front is also within twenty-five miles of Zambia, Swaziland and Republic of South Africa. South Africa is in particular danger of infection because of the constant movement of migrant workers from Malawi, Zimbabwe and Moçambique into South Africa. The outbreak at Lorenço Marques poses a major threat as a result of its location close to the South African border and its function as a very important port for South African trade.

The seriousness of the South-Eastern African outbreak is shown by the relatively large number of cases and deaths, and the persistence

of the infection. A total of 1,570 cases and 204 deaths (13.0% mortality) were reported from Moçambique up to June 1, 1974. Beira had 516 of these cases. Tete, Mutarara, Quelimane and Lourenço Marques each had over 100 cases. Malawi has reported 991 cases, but the somewhat irregular publishing of reports in the Weekly Epidemiological Record suggests that the outbreak may in fact be more extensive than officially reported. There were 535 cases and forty-two deaths (7.8% mortality) reported from Zimbabwe up to May 24, 1974.

Cholera was still being reported at the end of May, 1974 from all three Malawian provinces, four of the seven infected provinces of Moçambique, and two of the three infected Zimbabwe districts. However, the weekly number of reported cases has been declining since late March, 1974.

CHAPTER 5

INTER-REGIONAL DIFFUSION OF CHOLERA IN WEST AFRICA

Origin of Infection

The first indication that cholera had become established in West Africa came on August 18, 1970 when Guinea sent an appeal to the World Health Organization to investigate an outbreak of a disease feared to be cholera.[1] It was confirmed that the disease was cholera, but Guinea refused to make any official announcement of it. When the outbreak was unilaterally announced by the World Health Organization on September 4, 1970, Guinea withdrew in protest from the organization.[2]

The origin of the infection has not been definitely established. There were epidemics of cholera El Tor serotype Ogawa in the Middle East and along the Black Sea Coast of the USSR in mid-1970. The most likely explanation is that Guinean students in the USSR, who had holidayed in the Crimea just prior to their return to Guinea introduced the disease.[3]

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[1] L. La Peyssonnie, "Le choléra, An II, Médecine Tropicale, vol.31, No.6 (1971D), p.615.

[2] West Africa, September 5, 1970, p.1039.

[3] L. La Peyssonnie, "Acquisitions récentes en matière d'épidémiologie et de prophylaxe du choléra en Afrique", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971A), p.645. An alternate explanation of introduction by pilgrims from Saudi Arabia is given by La Peyssonnie, op.cit., 1971A and in "Cholera and politics", West Africa, September 8, 1972, p.1175. Introduction by pilgrims is improbable because August, 1970 was off-season for pilgrimmages.

From this initial focus in Conakry, cholera has spread throughout West Africa. Guinea-Bissau is the only country to escape infection.

It was noted in the previous chapter that four distinct patterns of diffusion characterized the spread of cholera between West African divisions or provinces. These were coastal, riverine, urban hierarchical and radial contact diffusion. A more comprehensive account of these four diffusion types follows. In addition, the chapter includes information on the morbidity and mortality associated with outbreaks in different parts of West Africa.

Coastal Diffusion

The term coastal diffusion is used for the movement of cholera along the coast from Guinea to Eastern Cameroun (Figure 11). The limited penetrations inland from the coast in several countries have been included where the base of the infection remained in the rural coastal zone. This definition excludes the majority of the Nigerian cholera epidemic which was essentially based in major cities, particularly Lagos.

Of the several West African ethnic groups specializing in fishing, the Fanti are most prominent. This homeland is coastal Ghana, but they are highly mobile and have established themselves along the length of the West African coast. These Fanti fishermen were apparently responsible for carrying cholera from Conakry along the West African coast.

Commencing in Conakry, the migrating fishermen moved along the coast. Their periodic stops along the coast allowed the cholera carriers

FIGURE 11

COASTAL DIFFUSION OF CHOLERA IN WEST AFRICA

Following the initial outbreak in Conakry in August, 1970, cholera was spread along the coast by fishermen. The infection reached Southern Cameroun in March, 1971. There were only limited penetrations inland, and these occurred in areas of relatively high population density such as South-Eastern Ivory Coast, Ghana and Togo.

The three insets show in greater detail the patterns of diffusion in South-Eastern Ivory Coast, Togo and Dahomey and Cameroun.

FIGURE II

COASTAL DIFFUSION OF CHOLERA IN WEST AFRICA

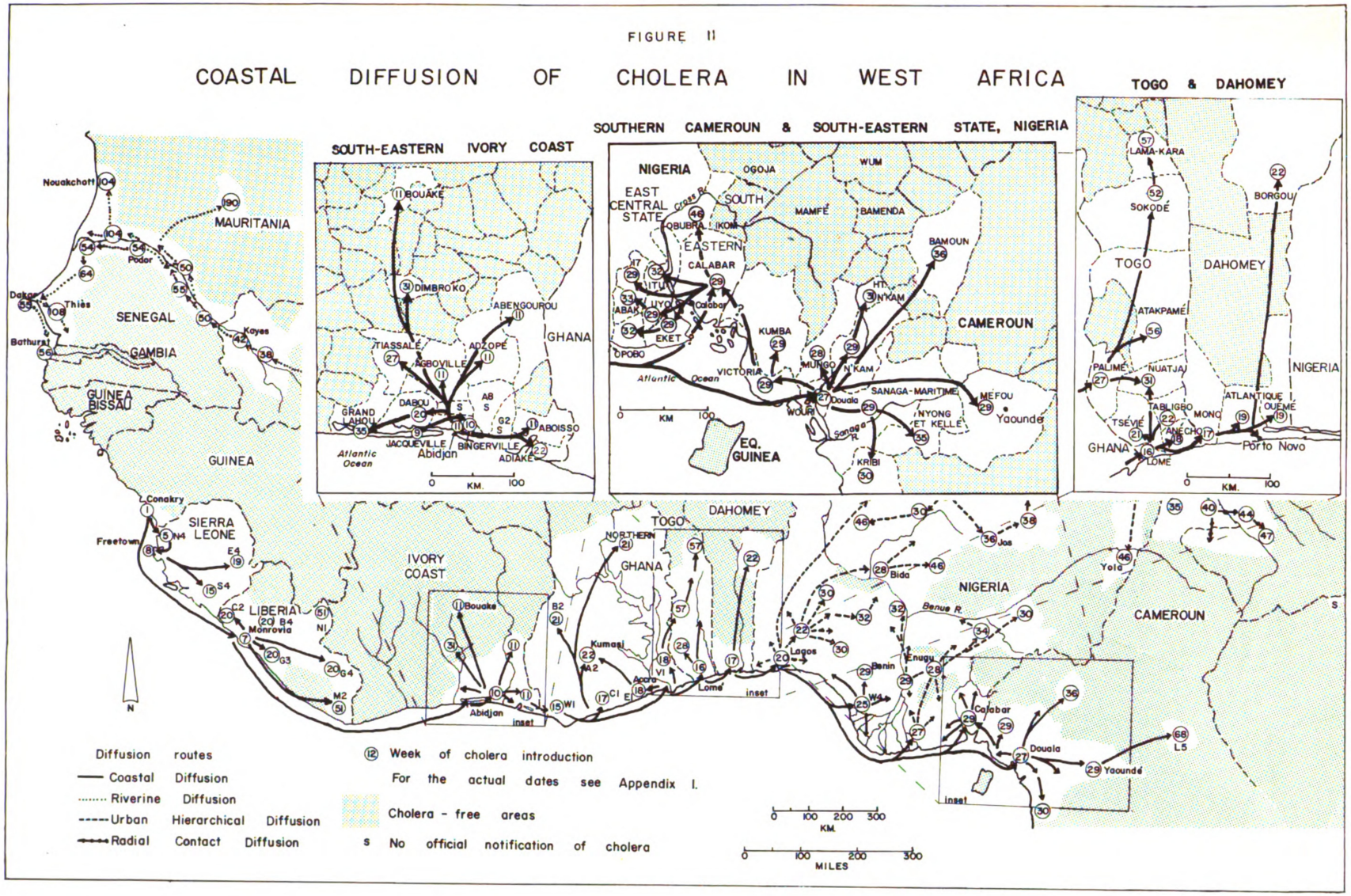

FIGURE 12

FANTI FISHERMEN, CAPE COAST, GHANA

The central portion of the Ghanaian coast is the Fanti homeland, but colonies of Fanti dot the West African coast from Senegal to Cameroun. The migration of Fanti fishermen expelled from Guinea along the West African coast in their canoes has been blamed for the dissemination of cholera in coastal West Africa.

in the group to introduce the infection to a new location. Where conditions permitted the survival of the vibrios and establishment of a local transmission cycle, a potential point of diffusion developed. The impact of a particular point depended largely on its location and the amount of interaction with other places. Lagoon villages near large cities such as Abidjan, Monrovia and Accra have been particularly dangerous sites. The swampy environment and brackish lagoon water are a suitable environment for long-term vibrio survival from which the infection may be repeatedly introduced to nearby cities. The risk of long distance transmission is greater from the cities because of the concentration of transport routes, commerce and travellers.

The role played by fresh and smoked fish in the transmission of cholera is unknown. Cholera vibrios survive readily on fish, and the large extent of the trade in fish suggests a possible role of cholera-infected fish in spreading the disease.

According to Dr. Joseph N. Togba, the migration responsible for the diffusion of cholera started as a result of their expulsion from Guinea.[4] There were major expulsions of Ghanaians from Sierra Leone in January, 1969,[5] but the major African news magazines contained no record of expulsions from Sierra Leone or Guinea in mid-1970. Unpublicized expulsions may have occurred, perhaps because of the existence of cholera infection among them.

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[4] University of Liberia Medical School. Lecture to I.D.C. 390 class at Michigan State University, February 7, 1973.

[5] Africa Diary, vol.9 (January 8-14, 1969), p.4261.

Guinea, Sierra Leone and Liberia

Very little is known about the extent of the Guinean epidemic. The World Health Organization estimated 2,000 cases and sixty deaths up to September 4, 1970,[6] which were apparently confined to Conakry and vicinity.

Sierra Leone's first reported cholera outbreak was in the fishing village of Bailoh located some 100 km. north of Freetown during the week of September 19-25, 1970. There were 293 reported cases in 1970 and 211 in 1971, but none since December, 1971. The infection was more persistent in Northern and Southern Provinces and Western Area which together occupy the coast than in landlocked Eastern Province or in the city of Freetown.

Cholera has been more serious and persistent in Liberia. Unfortunately, because their early reports to the World Health Organization were very sporadic, little is known of the diffusion or extent of cholera in Liberia. Monrovia reported the onset of cholera during the first week of October, 1970. All counties, except Lofa and Sinoe have reported cholera outbreaks. Data supplied to the World Health Organization indicate a persistent infection averaging about 100 cases per month in Monrovia. There has been no indication of any cholera outside Montserrado County since March, 1972.

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[6] West Africa, September 12, 1970, p.1070.

Ivory Coast

After the infection of Monrovia, cholera next appeared 1,000 km. to the east in Bingerville, a village located near Abidjan. The index case had apparently contacted the infection from a fisherman at Jacqueville, 40 km. west of Abidjan.[7] The apparent lack of infection of the area between Monrovia and Abidjan may be related to differences in coastal morphology, population density, or accessibility. Eastern Liberia and Western Ivory Coasts are not lagoon coasts, so they provide a less favorable environment for vibrio survival. It is also a relatively remote area with poor communications, low population density, and no major cities. Small, local outbreaks could easily have gone untreated and unreported.

An explosive outbreak resulted from the spread of cholera from Bingerville during the week of October 23-29, 1970. Twenty-five deaths and 447 cases were reported in Abidjan in the first week alone. A detailed bacteriological survey of a lagoon village near Abidjan in October showed that 20% of the population were carriers and that there was massive vibrio contamination of the environment.[8] The infection of several villages along Aby and Ebrié lagoons resulted in about 1,500

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[7] H. Félix, "Le developpement de l'épidémie de choléra en Afrique de l'Ouest", Bull. Soc. Pathol. Exotique, vol.64 (1971C), p.562.

[8] M. Duchassin, et al., "Le vibrion cholérique. Diagnostic bactériologique", Médecine d'Afrique Noire, vol.20, No.3 (1973), pp.165-174.

cases and 120 deaths between October and December, 1970.[9] Small outbreaks continued in Abidjan and nearby villages during 1971 and early 1972. The last report was in October, 1972.

Minor outbreaks of cholera were reported from the inland towns of Bouaké in October, 1970 and Dimbroko, Tiassalé and Abengorou between January and March, 1971. These towns are all located on major transport arteries extending north from Abidjan.

Abidjan also was the starting point of the most spectacular long distance transfer of cholera, namely the 1,500 km. diffusion to Mopti (Mali).

Ghana

Ghana's first cholera victim was a Togolese citizen arriving by air from Guinea on September 1, 1970.[10] The disease did not gain a local base until the beginning of November. The first reported occurrence was at Half-Assini on the Ivoirian border .[11] Outbreaks occurred during November in other places along the coast, including the Winneba, Accra, Keta and Ada areas. By mid-January the Upper Region was the only uninfected region.

The impact of cholera has been quite serious in Ghana. The reported cases and deaths totalled 2,886 and 73, respectively, in 1970, 12,623 and 609 in 1971 and 1,187 and 57 in 1972-1973. Well over

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9 A.Bourgeade, J. Rive et al., "L'épidémiologie du choléra et ses problèmes", Médecine d'Afrique Noire, vol.20, No.3 (1973), p.181.

10 J.O. Pobee et al., "Case report of cholera", Ghana Med. J., vol.9, (1970), pp.306-309.

11 West Africa, November 7, 1970, p.1326.

90% of these cases have been in the Southern regions which are at least partly coastal, namely Western, Central, Eastern Volta and Accra. It has primarily been a rural epidemic affecting mainly the fishing peoples. Larger cities with protected water supplies have been repeatedly infected from rural areas but reported few cases.[12] The transportation of bodies and funerals were implicated in most of the new, explosive outbreaks in the early stages of the Ghana epidemic.[13]

The Upper Region of Ghana has not reported a single cholera case, and the Northern Region reported only nineteen cases. Cholera was also absent from Eastern Guinea, Northern Ivory Coast, Southern Upper Volta and Northern Dahomey. The absence of cases is surprising because of the large amount of travel between these areas and the coast, especially the movement of migrant laborers. Various hypotheses may be advanced to account for the cholera-free status of Upper Ghana and similar areas. The infection may have been introduced by individual carriers but gone unreported because of a scarcity of medical facilities. The dispersed settlement pattern and upland location of most settlement may have provided an unfavorable environment for the establishment and dissemination of cholera. The low population density and small urbanization of the Middle Belt would hamper contact or hierarchical

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[12] D. Barua, "The global epidemiology of cholera in recent years", Proc. Royal Soc. Med., vol.65, No.5 (May, 1972), p.427.

[13] "Cholera in 1971", Weekly Epidemiological Record, vol.47 (July 28, 1972), p.282.

diffusion. The considerable width of the Middle Belt may also have a barrier effect. The distance factor prompts many travellers from coastal Ghana to interrupt their journey to Northern Ghana in such intermediate cities as Kumasi and Tamale. As a result, most cholera carriers would become vibrio-free or develop clinical symptoms preventing travel before reaching their destination in Upper Ghana.

Western and Central Regions are two of the few places in Africa which have continued to report cholera relatively frequently. All but five of the 653 Ghanaian cases in 1973 were from these two regions. With the exception of the Half-Assini area, lagoons are less extensive in Central and Western Regions than in the adjoining areas in Eastern Ivory Coast and Eastern Ghana where cholera does not seem to be endemic. It is perhaps significant that the homeland of the Fanti people, who played such an important role in the initial dissemination of cholera, is in Western and Central Regions. Endemicity in this area therefore may be more a function of cultural than physical environmental factors.

Togo and Dahomey

Cholera was brought into Togo by Ghanaian fishermen the week of November 13-19, 1970.[14] The epidemic peaked in January, 1971, when 158 cases were reported. Sporadic, minor infections continued during 1971 in the coastal departments. Cholera has affected the fringes of Lomé, but areas with piped water have been almost unaffected.[15] There were

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[14] J.J. D'Almeida et al., "L'épidémie de choléra au Togo", Médecine d'Afrique Noire, vol.20, Nos.8/9 (1973), p.639.

[15] D'Almeida, op.cit., pp.640-641.

relatively small outbreaks in such Central Togo departments as Palimé, Klouto, Akposso, Sokodé and Lama-Kara. Most of these infections originated in the neighboring Volta Region of Ghana.[16] Cholera was not reported from June, 1972 until January, 1974, when it reappeared near Lome.

The cholera epidemic in Dahomey was also based in coastal lagoons and the delta of the Ouémé River. The first case was found in the fishing village of Agoné Kamé near the Togolese border on December 7, 1970.[17] Cotonou was infected on December 12 and Porto Novo on December 24. The epidemic in Cotonou was double-peaked, the first occurring in January, 1971 and the second during the short rainy season in September and October, 1971. Cotonou had 198 cases and Porto Novo had seventy-one.[18] The national total during the serious epidemic phase (December 7, 1970 to April 10, 1971) was 1,812 clinical cases and 261 deaths (14.5% mortality). The only significant inland penetration occurred in November, 1971-January, 1972 when Borgou Division reported a total of 296 cases. Cholera persisted in the coastal lagoons of Dahomey until April, 1972, but the prediction by Aubry[19] of continued endemicity and new epidemics has not materialized.

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16 D'Almeida, op.cit., p.643.

17 Comité Central de Lutte contre le choléra, "L'Épidémie de choléra au Dahomey", Médecine Tropicale, vol.31, No.6 (1971), p.644.

18 P. Aubry et al., "Une expérience du choléra Africain...", Bull. Soc. Pathol. Exotique, vol.65, No.3 (1972), p.353.

19 Aubry, op.cit., p.356.

Nigeria

Nigeria was the next country to be infected along the coastal diffusion axis. Cholera first appeared in Ajegunle-Owode village at the mouth of the Ogun River just north of Lagos.[20] Five members of one household were hospitalized on December 26 and December 29, 1970. The first case in Metropolitan Lagos also occurred on December 26. The city of Lagos became an endemic cholera focus from which other Nigerian cities were infected. This urban-based diffusion is described later in the chapter.

Coastal, lagoon-based diffusion continued beyond Lagos. Western Ijaw Division of Mid-Western State reported the presence of cholera in the last week of January, 1971. Western Ijaw is located in the Western Niger Delta. Cholera was apparently introduced by Ijaw fishermen and local gin dealers travelling by boat from Lagos to the popular markets at Bomadi and Ojobo.[21] The disease spread throughout the rural deltaic areas of Southern Mid-Western State, as well as to the cities of Warri, Sapele and Benin. The urban outbreaks were relatively minor compared to those in rural areas. There were 758 hospitalized cases and 39 deaths up to March 7, 1971.[22] However, a survey of only six

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[20] B.A.A. Dada, "First sixteen weeks of cholera in Lagos State", J. Soc. Hlth. Nigeria, vol.6, No.3 (1971), p.133.

[21] V.G.B. Amu, "Experience with cholera epidemic in Mid-Western State of Nigeria", J. Soc. Hlth. Nigeria, vol.6, No.3 (1971), p.107.

[22] Amu, op.cit., p.109.

villages revealed 135 suspected cases and 75 deaths before the provision of medical aid. There were at least 200 more unreported deaths in February from other riverine villages. The remoteness of the affected areas and the necessity of using boats to transport victims caused delays in treatment and elevated death rates.

Cholera also spread into Otikpupa Division in Western State and Brass Division in Rivers State in mid-February. Both areas adjoin Mid-Western State. The size of the Rivers State outbreak is uncertain because of the failure to report cases. With the infection of Port Harcourt, a base was obtained for the diffusion of cholera into East Central State.

South-Eastern State reported cholera for the first time in the week of February 28-March 6, 1971. However, the fact that Southern Cameroun had reported cholera almost four weeks earlier suggests the infection of South-Eastern State may have been earlier than reported. All divisions were infected except Ikom and Ogoja in the Northern part of the state. There were only six weekly reports between March and July. However, the average incidence of over 250 cases per week indicates a relatively serious epidemic.

Cameroun

Southern Cameroun marked the farthest extent of the coastal diffusion axis. The introduction was again attributed to the movement of

fishermen along the coast.[23] The first case was in a fishing village near Douala. There were 333 cases and 55 deaths during the four week Douala epidemic. The deltaic zone in the Victoria-Douala area became a persistent focus of endemic cholera.[24] Minor occurrences continued to be reported periodically up to August, 1973.

Several other divisions of Southern Cameroun were infected for short periods of time in 1971 and 1972 from the Victoria-Douala. These included Méfou (Yaoundé), the Southern border division of Kribi which adjoins Equatorial Guinea (Rio Muni), Lom et Djerem on the Central African Republic border , and Bamoun in the Bamenda Highlands. That cholera did not advance into these adjoining countries or into Central Cameroun is probably a function of low population density, poor transportation and relatively long distances from the endemic focus around Douala and Victoria.

Generalizations

A number of generalizations follow concerning the spread of cholera along the West African coast. These generalizations have also been incorporated into a systems diagram and a schematic map diagram (Figure 13).

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[23] J. Dutertre et al., "Le choléra au Cameroun", Médecine Tropicale, vol.32, No.5 (1972), p.608.

[24] Dutertre, op.cit., pp.607-624 includes a detailed account of cholera in Victoria and Douala.

FIGURE 13

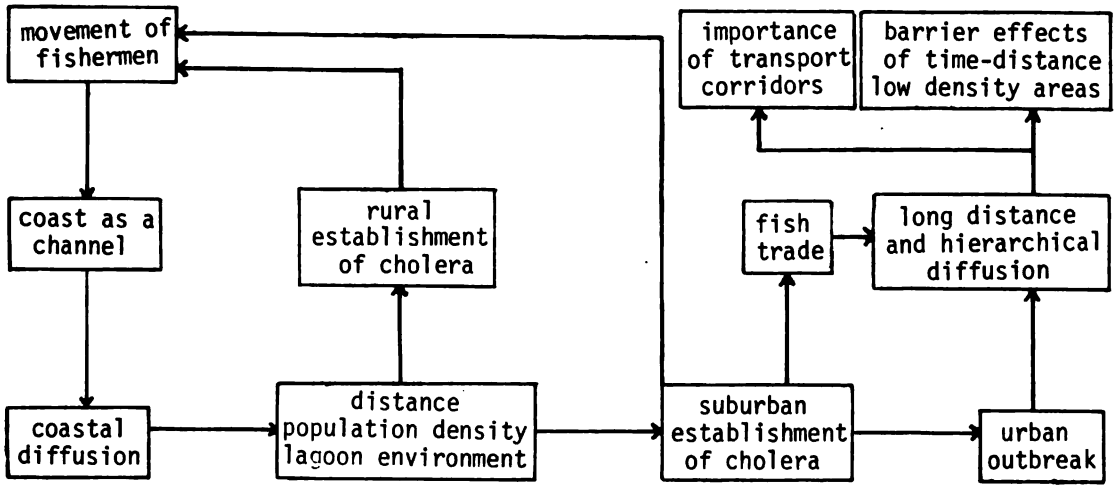
THE COASTAL DIFFUSION SYSTEM
SCHEMATIC MAP OF COASTAL DIFFUSION

The model shows the three phases of coastal diffusion. The first phase involves the spread of cholera along the coast to fishing villages in association with the movements of fishermen. Diffusion to surrounding villages from the points of introduction follows. Urban and hierarchical diffusion may follow the infection of major urban centers.

FIGURE 13

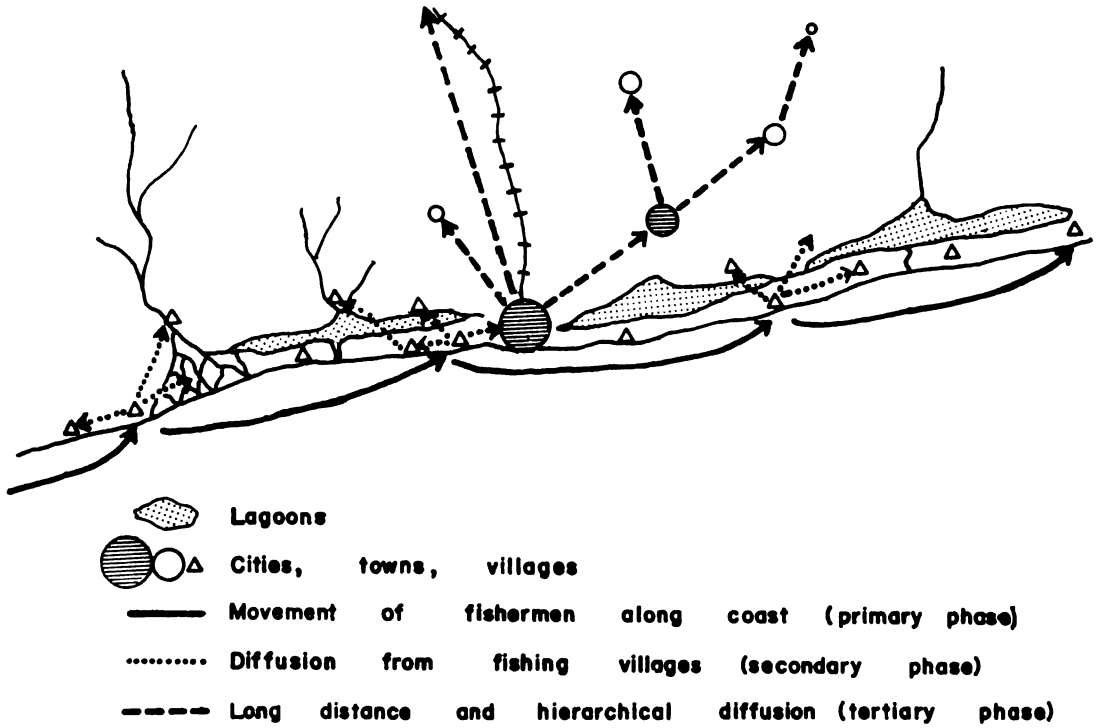
(A)

THE COASTAL DIFFUSION SYSTEM



(B)

SCHEMATIC MAP OF COASTAL DIFFUSION



1. The sequential west to east movement of the infection demonstrates that this was essentially contact diffusion with date of onset being primarily a function of distance from the point of origin.

2. In each of the infected coastal countries except Guinea, fishermen were responsible for the introduction of the infection and much of the subsequent internal diffusion. The Fanti were primarily responsible but other fishing people such as the Ijaw, Ewe, Kru and Ebrié were also involved.

3. The coast served as a diffusion channel for cholera. This may be attributed to the lack of east-west roads near much of the coast, and the use of canoes for both short and long distance travel.

4. The fishermen first introduced the disease in rural or suburban areas. This may be termed the primary phase of the coastal diffusion (Figure 13). The areas of introduction were physiographically distinctive, being either deltas or lagoon coasts. Cholera has also persisted longer in these deltaic and lagoon areas.

5. When villages near a major urban center were infected, the disease spread to the urban center (secondary diffusion phase) and subsequently diffused along transport axes as hierarchical or long distance diffusion (tertiary stage).

6. Distance from coastal endemic foci and low population density regions such as the Middle Belt and Southern Cameroun seem to have acted as a barrier to the disease. Despite considerable movement by migrants

and traders across the Middle Belt, it was breached in only two places, namely, Nigeria and Mali. Denser population and better transport reduce the barrier effect of the Middle Belt in Nigeria. As for the long distance transfer of cholera from Abidjan to Mopti, it is probably not unreasonable to consider it a long-shot possibility which happened to materialize.

Riverine Diffusion

Primary (River Valley) Phase

The spread of cholera to Mopti (Mali) from Abidjan, a distance of 1,500 km. has been noted in the previous section. Mopti is a very important center for the marketing of dried and smoked fish from the Niger River's Inland Delta. The index case was a trader who had arrived on November 5, 1970 to buy fish in the market.[25] He developed cholera symptoms on November 6 and spent two days frequenting the public latrines of the market.[26] An explosive outbreak occurred two weeks later at the time of the weekly market in Mopti. The large attendance at the market, much coming from a considerable distance explains the rapid diffusion of the infection from Mopti.

The Mopti epidemic corresponded to the classical epidemic model of four stages, namely, introduction of the contamination, latent phase with multiplication of the bacteria, explosive epidemic and finally dissemination outside with the departure of people.[27]

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[25] Félix, op.cit., 1971C, p.563.

[26] Félix, op.cit., 1971C, p.563.

[27] Félix, op.cit., 1971C, pp.563-564.

The diffusion of cholera from Mopti initially followed the axis of the Niger River. The week after the Mopti outbreak, five divisions in the Inland Delta, plus the important river town of Segou, located upstream from Mopti, reported cholera. The other two divisions partially located in the Inland Delta (Djenné and Douzentza) were infected in the third week. Tombouctou, Gao and Koulikoro also reported cholera in the third week of the riverine epidemic. Koulikoro and Gao are respectively the western and eastern terminuses of the Niger River navigation system, and Tombouctou is one of the main intermediate ports. The river is the main transportation channel in Eastern and Central Mali, and for most of the riverine areas the only access route from about July to November. The division containing the Malian capital, Bamako, was infected in the fifth week.

The next significant advance was on December 27, 1970 when the border of the Niger Republic was breached. The first outbreak in Niger was at Famalé in Tillabery Division. It subsequently spread within four weeks to Tera, Niamey and Gaya Divisions along the Niger and to three peripheral divisions. The only other occurrence of cholera in the first ten weeks of the riverine epidemic were in Ouagouiya and Tougan in Northern Upper Volta.

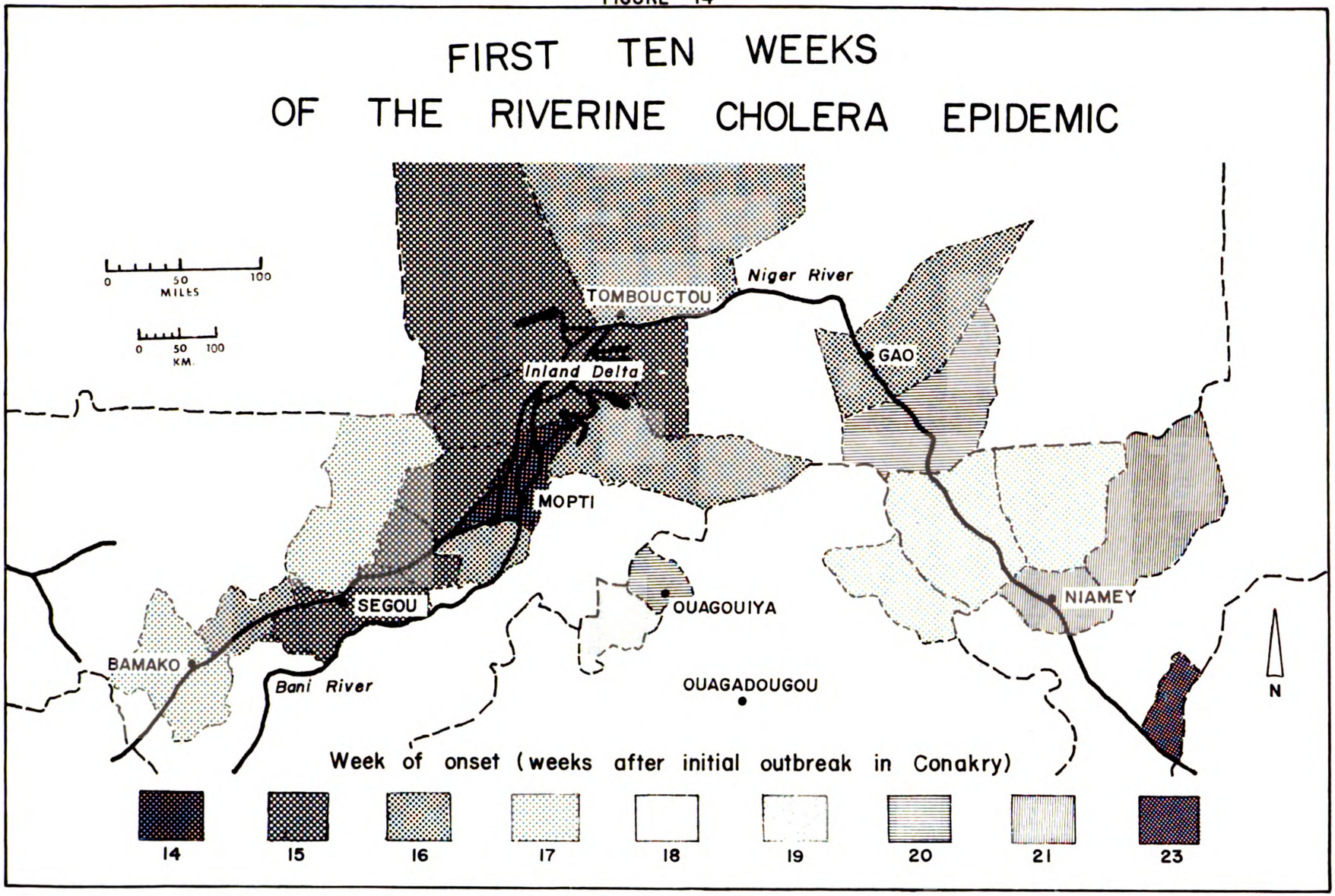
Figure 14 effectively shows the connection between location along the Niger River and the early stages of cholera in Mali, Niger and Upper Volta. Of the twenty-four divisions infected during the first ten

FIGURE 14

FIRST TEN WEEKS OF THE RIVERINE CHOLERA EPIDEMIC

All but five of the first twenty-four divisions infected during the riverine cholera epidemic were located along the Niger River. This map emphasizes the high correlation between riverine environment and early cholera onset.

FIGURE 14


FIRST TEN WEEKS
OF THE RIVERINE CHOLERA EPIDEMIC
0 50 100
MILES
0 50 100
KM.
TOMBOUCTOU
Niger River
GAO
Inland Delta
MOPTI
SEGOU
OUAGOUIYA
NIAMEY
BAMAKO
Bani River
OUAGADOUGOU
N
Week of onset (weeks after initial outbreak in Conakry)
14
15
16
17
18
19
20
21
23

weeks, nineteen were located along a 2,000 km. expanse of the Niger River between the Nigerian border in Gaya Division and the Guinean border in Bamako Division.

Various factors account for the channelling effect of the River Valley. Where navigable the rivers themselves provide a transportation route. When roads are constructed, river valleys often offer the path of least resistance. In Mali, the population density is higher in the valley than in the neighboring upland areas. Also, river valleys provide a suitable hydrological environment for the survival of vibrio cholerae. It is these characteristics of river valleys which facilitate cholera dissemination.

The introduction of cholera in the riverine divisions of Mali was typically followed by an epidemic lasting about three to seven weeks. It is very difficult to estimate the extent of these outbreaks. Because of the inaccessibility of many of the infected areas and the total lack of prior preparation by health authorities, mortality was very heavy and the majority of cases went unrecorded. For example, a total of 917 cases and 33 deaths were listed in the Weekly Epidemiological Record from Goundam and Diré Divisions in 1970. However, Félix reports that there were over 5,000 cases and 2,000 deaths among sedentary farmers alone, plus heavy mortality which is impossible to estimate among the nomads and fishermen.[28]

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[28] H. Félix, "Épidémie de choléra en Afrique. Note d'information sur l'évolution entre Août et Décembre, 1970", La Presse Médicale, vol.79, No.11 (1971), pp.476.

Secondary (Valley Periphery) Phase

The second phase of the riverine epidemic in Mali comprises the period between the tenth week of the Mopti epidemic (week 25 out of Conakry) and the forty-seventh week. Cholera remained endemic in the Inland Delta, particularly around Mopti. There were periodic new outbreaks in the important river towns of Ségou and Koulikoro. It was from this focus of infection that cholera gradually penetrated into the valley of the Bani River and the upland periphery of the Niger Valley via Mopti, Ségou and Koulikoro.

There were four principal thrusts into the peripheral divisions. One was south from Mopti into the vicinity of the Bandiagara Escarpment and the northern border divisions of Upper Volta. A second axis of diffusion was the movement from Segou into three divisions in the Valley of the Bani River, a major tributary of the Niger. The three divisions were San, Koutiala and Dioila. There was also a major northward thrust from Koulikoro into Banamba and Nara in arid North-Western Mali. Nara had a reported 1,129 cases and 332 deaths in thirteen weeks. Cholera reached South-Eastern Mauritania from Nara. Amourj, Néma and Timbédra were the infected Mauritanian divisions. The fourth diffusion axis, also originating in Koulikoro, extended along the Dakar-Mali railway into the headwaters of the Senegal River. Bafoulabé and Kayes Divisions were infected in Mali. The infection subsequently progressed down the valley of the Senegal River, reaching the coast at the beginning of September, 1971.

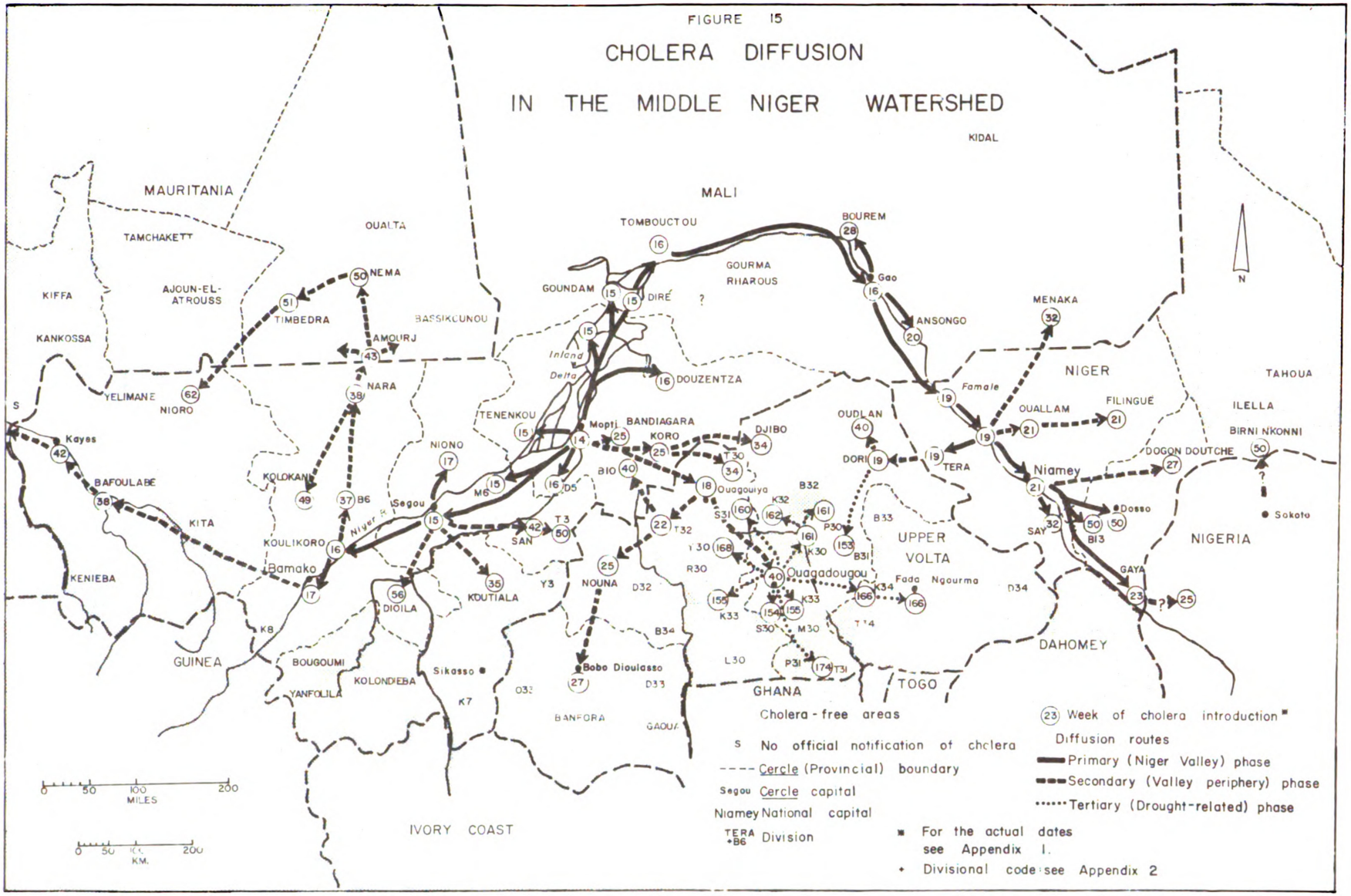
FIGURE 15

CHOLERA DIFFUSION IN THE MIDDLE NIGER WATERSHED

Cholera was introduced from Abidjan into the Niger Valley at Mopti in mid-November, 1970. During the first phase of the epidemic, cholera rapidly spread down the valley of the Niger River to Nigeria and upstream as far as Bamako. The secondary phase involved a more gradual penetration of the upland areas flanking the Niger River, the Bani River Valley and the valley of the Upper Senegal. Cholera has continued to spread in Upper Volta in connection with the West African drought. This has been called the tertiary diffusion phase.

FIGURE 15

CHOLERA DIFFUSION IN THE MIDDLE NIGER WATERSHED



Six divisions in Sikasso cercle in Southern Mali, as well as three other divisions bordering Guinea were uninfected. These areas were relatively far from the main centers of diffusion. However, the uninfected area included two important towns, Kita and Sikasso. The lack of cholera cases in these two towns provides further evidence that contact diffusion governed by distance from the endemic foci and river valley location is involved, rather than urban hierarchical diffusion.

The impact of cholera in Western Niger was most serious in Tera, Tillabery and Niamey Divisions along the Niger River. There were 1,677 cases and 239 deaths reported from Tera in 1971, 1,188 cases and 126 deaths in Tillabery and 850 cases with 95 deaths in Niamey. Cholera was reported during thirty-three weeks in Tera and Tillabery and twenty-three times from Niamey. The total reported incidence in 1971 in the eight other infected divisions in Western Niger was less than 275 cases.

Cholera reached Kayes Division along the Upper Senegal River in June, 1971. Bakel (Senegal) reported an outbreak between July 28 and August 13, 1971. There were eleven clinical cases along a thirty kilometer portion of the Senegal River. All of the victims were stricken after returning from funerals in Mali.[29] The infection progressed down the Senegal Valley, appearing almost simultaneously on the Mauritania and Senegal sides of the river. It reached St. Louis near the mouth of Senegal River on September 3, 1971.

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[29] S. Diop et al., "Considérations cliniques et épidémiologiques à propos de l'épidémie de choléra dans le département de Bakel (Sénégal)", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), pp.672-673.

FIGURE 16

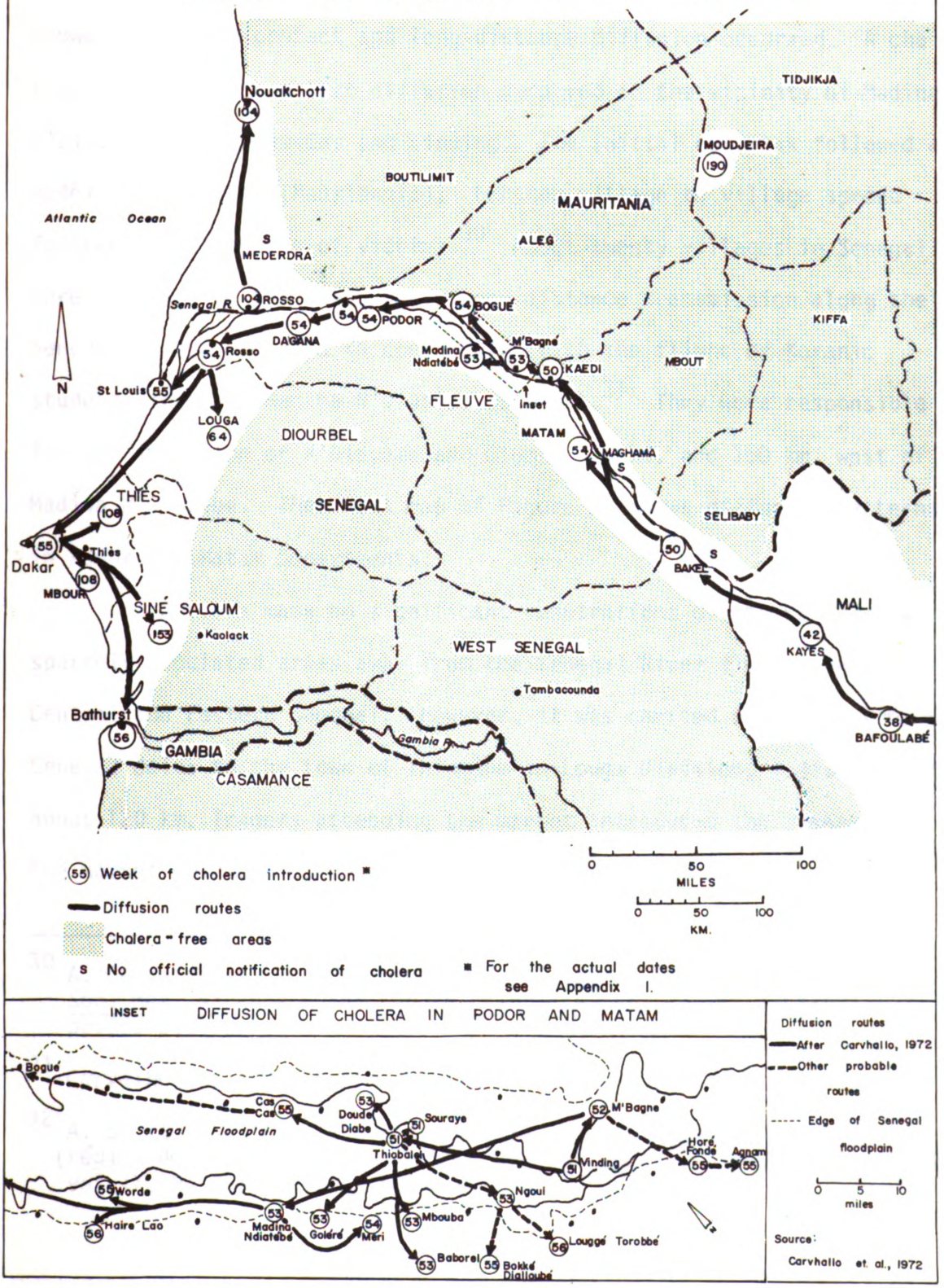
DIFFUSION OF CHOLERA ALONG THE SENEGAL RIVER CORRIDOR

Cholera entered Senegal from Kayes Division, Mali and moved down the Senegal River, appearing almost simultaneously in Mauritania and Senegal. It was only in the relatively populous North-Western Senegal that any significant extension beyond the Senegal Valley occurred. Cholera appeared in 1971, 1972 and 1973 and followed the same pattern each time, namely appearance in the Senegal Valley, followed by diffusion to Louga Division, the infection of Dakar, and spread to Thiès and Sine Saloum south and east of Dakar.

The inset shows in more detail the patterns of spread along the Senegal Valley in Podor and Matam Divisions. Weddings and funerals were instrumental in promoting this outbreak.

FIGURE 16

DIFFUSION OF CHOLERA ALONG THE SENEGAL VALLEY CORRIDOR



INSET DIFFUSION OF CHOLERA IN PODOR AND MATAM

Diffusion routes
After Carvhallo, 1972
Other probable routes
Edge of Senegal floodplain
0 5 10
miles

Source:
Carvhallo et. al., 1972

A study of cholera diffusion in Podor and Matam Departments showed that both contact and long-distance diffusion occurred. A chain reaction of local contact diffusion occurred in the vicinity of Madina N'Diatèbe, Meri, Cas-Cas and Vinding. The initial outbreak followed a wedding in Vinding (Mauritania). Further village to village spread followed the funerals of victims.[30] About twenty villages in Senegal were infected in this outbreak. Long distance transmission along the Senegal Valley occurred in conjunction with the flight of Koranic students from the Madina N'Diatèbe outbreak.[31] They were responsible for the infection of N'Diayème and Diamel, 60 km. and 150 km. west of Madina N'Diatèbe. The inset map of Figure 16 shows diffusion patterns in Podor and Matam Departments.

Cholera made no significant penetrations of the relatively sparsely populated areas away from the Senegal River floodplain in Central and Eastern Senegal. However, it was carried southward from the Senegal delta to the town of Thiomène in Louga Division, a distance of about 120 km. Traders attending the market introduced the disease.[32] The market became a center of diffusion from which cholera spread to

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30 A. Carvalho et al., "L'épidémie de choléra à Podor et Matam", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), pp.655-656.

31 Carvalho, op.cit., p.658.

32 A. Sy et al., "L'épidémie de choléra dans le département de Louga (région de Diourbel)", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), pp.662-669.

surrounding villages and to Dakar. There were only eight cases in Dakar in 1971.

Bathurst (Gambia) reported three cholera cases and one death on September 3, 1971. Dakar was the probable source of the infection.

Tertiary (Drought-related) Phase

Cholera remained endemic in various parts of West Africa after 1971, but the Niger-Senegal riverine diffusion system includes the only divisions to be newly-infected since 1971. Cholera has expanded into new divisions in Upper Volta, Senegal and Mauritania. The continuing diffusion of cholera in these countries may be partly attributed to the existence of a cholera-susceptible population, particularly in previously cholera-free areas. A more important reason is the serious drought afflicting these countries. Under-nutrition resulting from crop failures and the death of domestic animals reduces the body's resistance to infections. The drought has also forced millions of people to migrate, often to refugee camps with no protected water supply or waste disposal system.

The greatest expansion since 1971 has been in Upper Volta. Cholera was confined in 1971 to the divisions bordering on Mali and Niger and a minute outbreak in Ouagadougou. There was only one reported case in 1972. During 1973, cholera was reported from seventeen divisions. Twelve were infected for the first time. The 1973 outbreak was confined

to the Central and Eastern parts of the country. Dori and Ouagadougou were the main diffusion centers. The epidemic appears to have originated in Niger and spread to Ouagadougou via Dori.

Cholera advanced to new areas in Senegal and Mauritania in both 1972 and 1973. The newly-infected Mauritanian divisions included the capital city of Nouakchott and Moudjeira in the central part of the country. The extensions of cholera in Senegal have been east and south-east of Dakar. The pattern of occurrence has been the same in 1971, 1972 and 1973. Outbreaks have originated in the Senegal Valley (Fleuve Region) in the Middle of the rainy season, followed by explosive outbreaks in Diourbel midway between the Senegal Delta and Dakar, infection of Dakar and finally diffusion into Thiès and Sine Saloum Regions east and south-east of Dakar.

Analysis of variance and correlation analysis were used to evaluate the relative importance for the occurrence and diffusion of cholera of selected variables. Seven independent variables were recorded for each division in Mali, namely distance from Mopti, total population of the division, population of the largest urban center of each division, population density, a transportation score, a composite index of urban importance[33] and presence or absence of a river.

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[33] The index of urban importance includes urban population, government functions (divisional, regional or national capital), transportation, industry, and urban amenities such as hospitals, secondary schools, banks, and hotels. The data were obtained from Guid'Ouest-Africain 1971-72. Paris, 1971 and Annuaire Statistique:1971. Bamako: République du Mali-Direction nationale du plan et de la statistique, 1973.

Analysis of variance was used to determine the relationship between the first six of these variables and the presence or absence of cholera in a division. A chi-square test was used to evaluate the impact of the seventh variable. Analysis of variance was chosen as a suitable technique for testing the relationship between a nominal variable (presence or absence of cholera) and interval scales.[34] Because the "river" variable is itself nominal, it was necessary to use a chi-square test in this case.

The tests verified the considerable importance of distance from Mopti (F value of 8.07, significant at the .01 level) and presence or absence of a river (chi-square value of 5.13, significant at the .05 level) for the pattern of cholera occurrence. The second highest F value was only 0.88 for the transportation score, which would be significant at only a .35 level. Total population, urban population, urban importance and population density showed even weaker relationships with the occurrence of cholera.

Pearsonian correlation coefficients were also calculated for the variables listed above with the number of weeks necessary for the spread of cholera from the initial focus in Mopti. Divisions reporting no cholera cases were excluded. Pearsonian correlation coefficients may be calculated because the correlated variables are interval scales. Table 2 lists the Pearsonian (simple) and multiple correlation coefficients.

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[34] H.M. Blalock, Jr., Social Statistics (second edition). New York: McGraw-Hill, 1972, p.318.

TABLE 2

Mali: Correlation of the Week of Cholera Onset with Selected Variables

Riverine environment	.736 **
Urban index	.301
Distance to Mopti	.291
Population density	.267
Transportation score	.247
Urban population	.199
Total population	.186
R =	.789 **

(Level of significance: *: .05, **: .01, ***: .001)

Only one variable was significantly related to the week of onset, namely the presence or absence of a river. This high correlation was to be expected because of the virtual confinement of the infection to the Niger Valley during the first ten weeks of the epidemic. The .291 correlation between week of onset and distance from Mopti is lower than expected. Distance was the most important variable for the presence or absence of cholera.

Figure 17 shows that a disaggregation of the divisions into four categories, namely, ones located along the Niger, those along tributaries of the Niger, those in the Senegal watershed, and the remaining upland divisions more clearly shows the relationship between distance and

FIGURE 17

DISTANCE, URBAN HIERARCHY, VALLEY LOCATION AND CHOLERA DIFFUSION IN MALI AND WESTERN NIGER

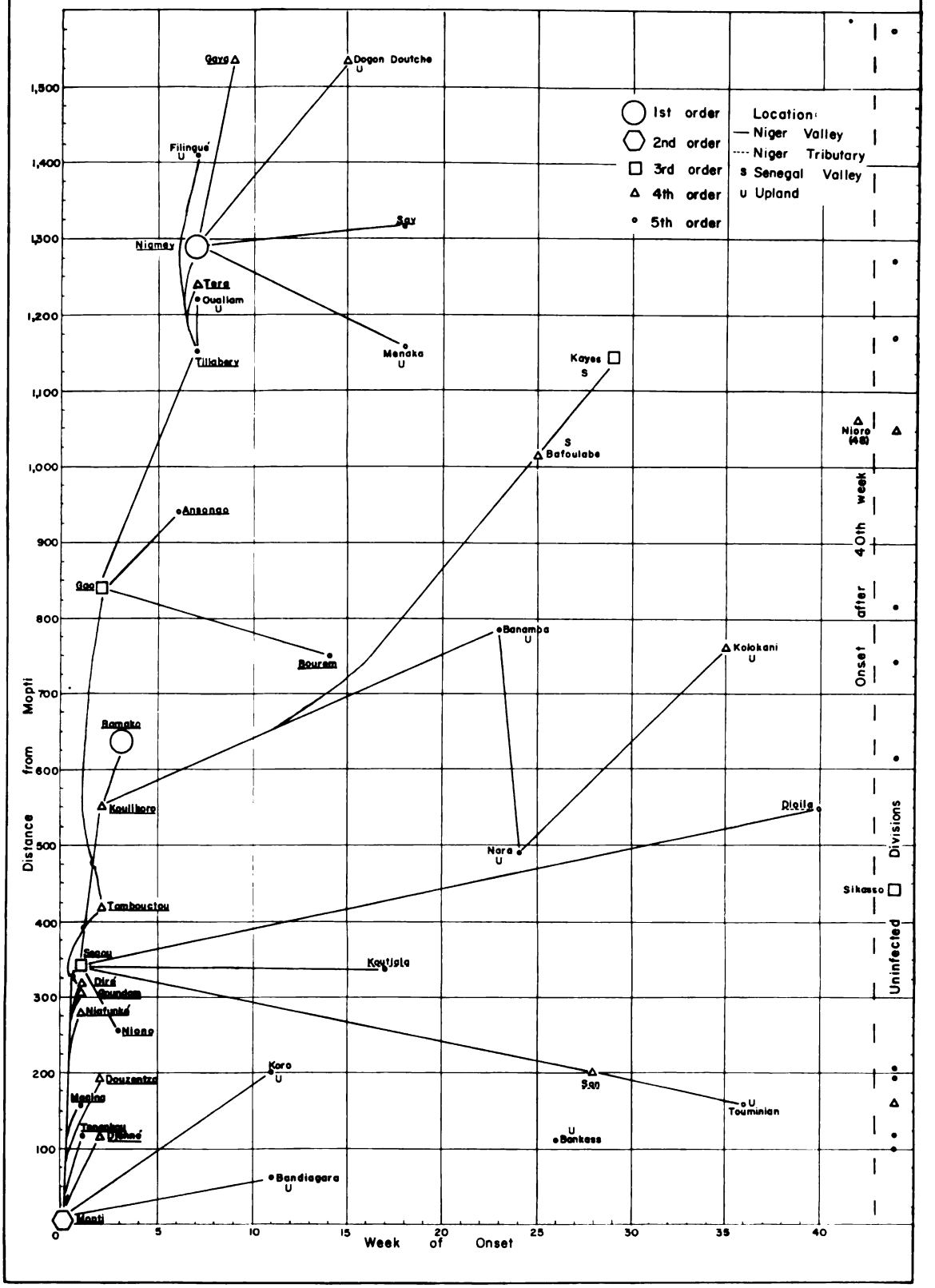
Distance from the initial outbreak at Mopti is plotted against the week of onset. The position in the urban hierarchy of the largest town in each division and the topographical location of each division is shown.

The relatively late infection of important cities such as Bamako, Niamey and Kayes and the cholera-free status of Sikasso shows that urban importance was not a primary factor in the date of onset. Bamako, Niamey and Kayes each were infected from a smaller urban center, providing evidence of the absence of urban hierarchical diffusion.

The relationship between distance to Mopti and week of onset at first appears weak. However, when topographical location is considered in conjunction with distance, a clear pattern emerges. Locations along the Niger Valley were clustered near the left axis of the graph with a nearly vertical trend. However, places in the Niger Valley close to Mopti were infected sooner than those located at a greater distance. Cholera took a longer time to spread a comparable distance in the Senegal Valley, and much longer into the valleys of Niger tributaries. In each of the above groups, the distance-time of onset relationship is almost linear. There is no such linear relationship for upland locations.

FIGURE 17

DISTANCE, URBAN HIERARCHY, VALLEY LOCATION & CHOLERA DIFFUSION IN MALI & WESTERN NIGER.



the week of onset. Within each of the four categories, places farther from Mopti tended to be infected later than those relatively near Mopti. This relationship is lost when the four categories are combined. The average speed of diffusion was greatest along the Niger Valley and slowest to places located along tributaries of the Niger and upland divisions.

The urban hierarchy, based on the index of urban importance[35] has also been shown in Figure 17. There is no clear pattern of larger centers having been infected before smaller ones, independent of the effects of distance and location in the Niger corridor. This provides further evidence that urban hierarchical diffusion was unimportant in Mali.

Generalizations

The following generalizations, illustrated in Figure 18, may be made about the riverine cholera epidemic.

1) Major river valleys, namely, the Niger and Senegal Valleys, served as channels along which the infection could travel with relative ease. The channel effect is largely a function of the importance of these valleys as transportation corridors, the relatively poor development of land transportation, the concentration of population in the valleys, and the suitability of the riverine environment for the survival of *vibrio cholerae*.

2) Low-lying, seasonally flooded areas such as the Inland Delta of the Niger and the Lower Senegal River served as endemic foci.

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[35] For a brief description, see footnote 33.

FIGURE 18

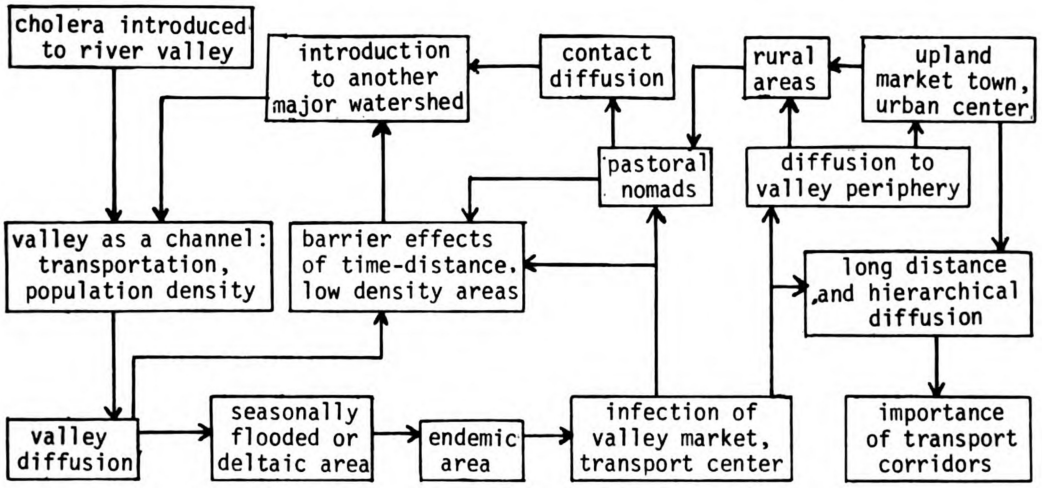
THE RIVERINE DIFFUSION SYSTEM
SCHEMATIC MAP OF RIVERINE DIFFUSION

Figure 18 is intended to schematically summarize the riverine form of cholera diffusion found in West Africa.

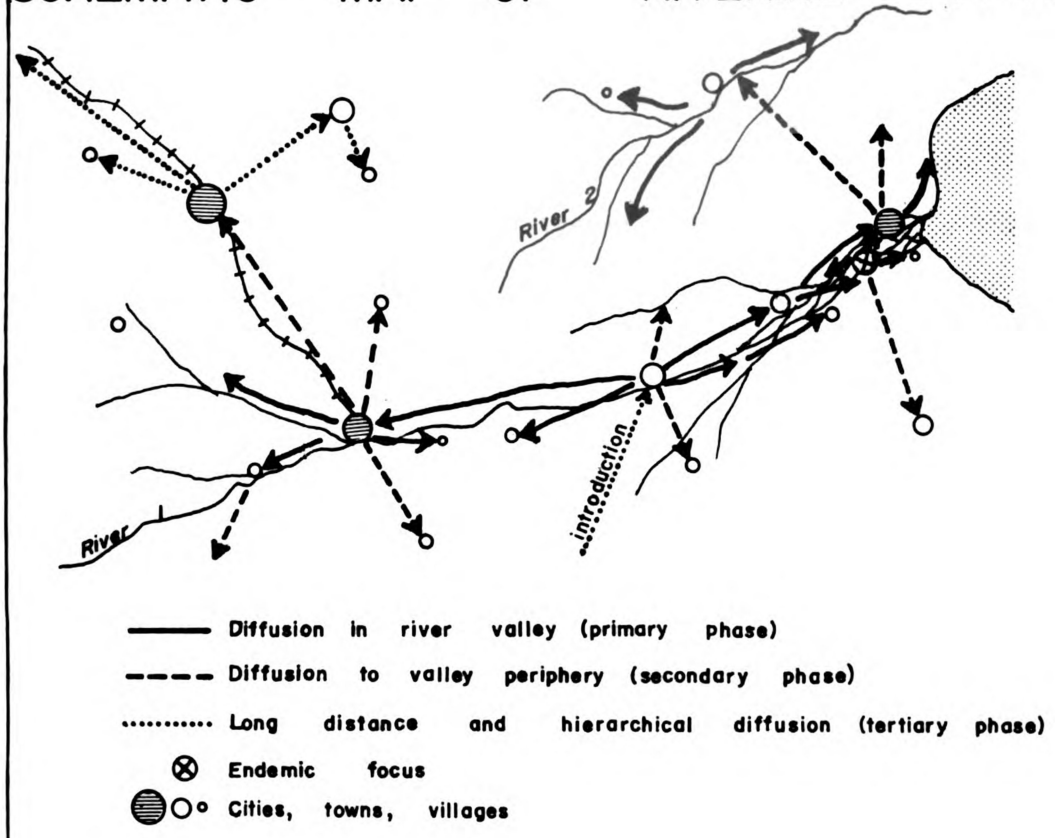
There is a three stage sequence of infection. During the initial phase, the infection is confined to a river valley. Distance along the valley from the initial outbreak is more important than urban hierarchical relationships in determining the sequence of onset. After this initial phase, cholera spreads to the upland areas flanking the valley, and may reach another river valley where the above sequence is repeated.

FIGURE 18

(A) THE RIVERINE DIFFUSION SYSTEM



(B) SCHEMATIC MAP OF RIVERINE DIFFUSION



3) Market towns and transportation nodes were the main points of dissemination as a result of their attraction of both carriers and susceptibles from a variety of locations. This process was most evident at Mopti, causing Félix to label it the "new Mecca" for cholera in Africa.[36]

4) Three stages may be recognized in both the Niger and Senegal Valleys, namely, rapid movement along the valley, more gradual penetration of the uplands and the continuing, drought-related outbreaks.

5) Riverine environment and distance from the index infection at Mopti are the two variables significantly related to both the presence or absence of cholera and the week of cholera onset.

Urban Hierarchical Diffusion

The coastal and riverine diffusion systems were examples of linear contact diffusion along a channel with major endemic foci lying outside the major cities. The dissemination of cholera in Nigeria involved a very different process. The infection was based in large cities and diffused hierarchically from higher to lower-ordered centers within the cities spheres of influence. Natural channels played no important role in inter-regional diffusion except along the coast. However, the configuration of man-made transportations was significant.

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[36] H. Félix, "Le choléra Africain", Médecine Tropicale, vol.31, No.6 (1971D), p.621.

The Hausa areas of South-Central Niger have been included in the urban hierarchical diffusion system. The cities of Maradi and Zinder fall within the sphere of influence of Kano (Nigeria). Cholera spread from Kano to Maradi and Zinder and subsequently to their hinterlands.

The spread of cholera beyond Lagos to the Niger Delta and Cross River Estuary forms a part of the coastal diffusion network. As previously noted, coastal diffusion has been distinguished from urban hierarchical diffusion according to whether the infection was primarily based inside or outside a major urban center.

The first three cases of cholera in Lagos State were in the rural areas. However, the infection quickly reached Lagos Metropolitan Area and became based there. Approximately 95% of the 922 confirmed cases in the first sixteen weeks of the epidemic came from Lagos and its suburbs. [37] A September, 1972 report showed a total of over 4,700 confirmed cholera cases in Lagos State between December, 1970 and September, 1972.[38] Cholera was continuously present during this period. The sporadic reporting of cases in the Weekly Epidemiological Record since 1972 prevents an assessment of continuing endemicity in Lagos.

Cholera spread from Lagos to the second order centers of Ibadan, Port Harcourt and Kano after one, seven and eight weeks, respectively.

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[37] Based on data in B.A.A. Dada, op.cit., 1971B, pp.133-138. An exact figure cannot be calculated because some districts are partly urban and partly rural.

[38] B.A.A. Dada, "Cholera in Lagos State: 26/12/70-2/9/72", Lagos: Ministry of Health and Social Welfare, 1972.

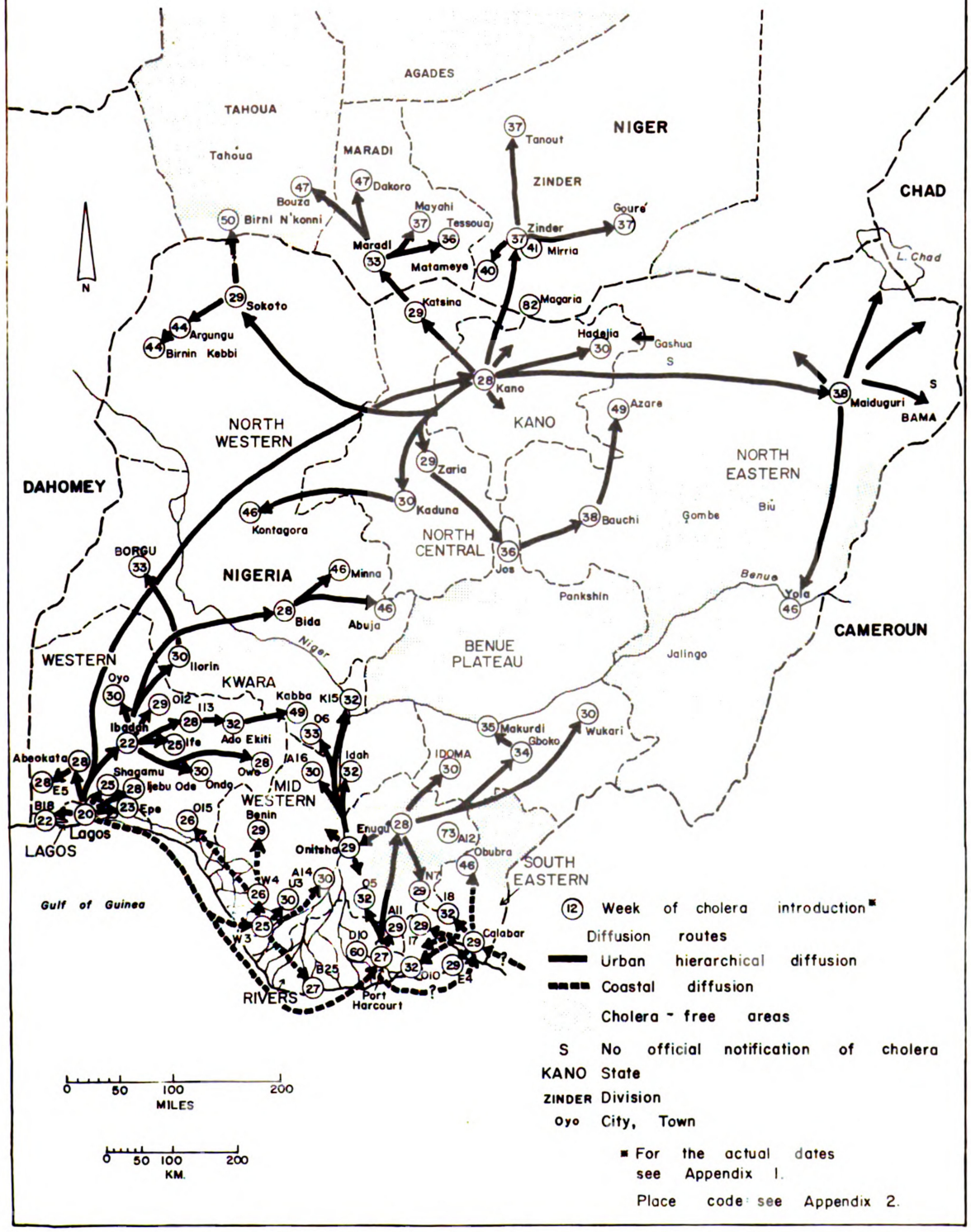
FIGURE 19

URBAN HIERARCHICAL DIFFUSION OF CHOLERA
IN NIGERIA AND SOUTH-CENTRAL NIGER

The map shows the paths of diffusion of cholera in Nigeria and South-Central Niger. The Nigerian epidemic commenced with the onset of cholera in Lagos on December 26, 1970. The disease spread from Lagos downward through the urban hierarchy, Ibadan, Port Harcourt and Kano were important secondary diffusion foci. Distance from Lagos and urban importance were the two variables most closely correlated with the occurrence and time of onset of cholera. Areas in Mid-Western and South-Eastern States, where the endemic foci remained in rural coastal areas, form part of the coastal diffusion system.

FIGURE 19

URBAN HIERARCHICAL DIFFUSION OF CHOLERA IN NIGERIA AND SOUTH - CENTRAL NIGER



Cholera also reached smaller places within a 100 km. radius of Lagos, such as Epe, Ikorodu, Shagamu, Ijebu-Ode, Abeokuta and Badagry from Lagos. This diffusion took place between the second and tenty week of the Nigerian epidemic.

Ibadan, a city of 1,000,000 located ninety miles north of Lagos, was infected on January 3, 1971. An explosive outbreak followed which reached a peak of 1,095 cases in its seventh week[39] and continued for over a year. Cholera spread from Ibadan to surrounding Yoruba cities such as Oyo, Ogbomosho, Ilesha, Ado-Ekiti, Ife and Ilorin. Most of them were infected at the time of the epidemic peak in Ibadan. Cholera diffused from these centers to smaller places on the periphery of Yorubaland including Kabba, Okene, and Borgu (New Bussa). Ibadan was also the probable origin of an outbreak in Bida during the week of February 14-20, 1971. Bida was the probable source of subsequent outbreaks in Abuja and Minna in mid-June.

Cholera probably reached Port Harcourt along the coastal diffusion route. Port Harcourt, located on the eastern side of the Niger Delta, is Nigeria's second port, a major industrial center, and shares with Warri the role of petroleum industry headquarters. Barua reports that morbidity in Port Harcourt was 3.3%[40], which would indicate a total of

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39 J.D. Adeniyi, "Cholera control: problems of beliefs and attitudes", Int. J. Hlth. Education, vol.15, No.4 (1972), p.238.

40 D. Barua, "The global epidemiology of cholera in recent years", Proc. Royal Soc. Med., vol.65, No.5 (May, 1972), p.427.

about 3,000 to 5,000 cases of cholera. The infection apparently spread to the nearby city of Aba and to Enugu, the capital of East-Central State. Enugu served as a focal point from which cholera spread to Onitsha and north to Idoma, Gboko and Wukari Divisions in Southern Benue-Plateau State. Onitsha, a very important market town and the site of the only bridge across the Niger River into Eastern Nigeria, became a center of diffusion. Surrounding towns in East-Central and Mid-Western States were infected, as well as Idah and Koton Karfi which are river towns north of Onitsha. Less than 100 cases were reported from East-Central State in 1971. Several divisions did not report a single case. Such minor incidence is surprising because of the population density which is the highest in Africa[41] and the disruption resulting from the 1967-1970 civil war which was staged in East Central State. It is suspected that the actual incidence was considerably greater than reported.

Cholera reached Kano in mid-February, probably from Lagos. Allegations by Schram[42] and Humponu-Wusu[43] that Kano was infected by pilgrims returning from Mecca are unlikely, as there was no report of cholera in Mecca, nor of any other place where Mecca was suspected to be the source. Kano is the largest city and most important business, industrial and transportation center in Nigeria. Although there are virtually

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[41] The population density of East-Central State averages 711 per square mile. Nigeria Year Book 1971. Lagos: Daily Times Press, 1971, p.22.

[42] R. Schram, "The 1971 cholera epidemic in Zaria", Savanna, vol.1, No.2 (1972), p.215.

[43] O.O. Humponu-Wusu, "Epidemiological aspects of an El Tor cholera outbreak in Kaduna, Nigeria", Trop. Geog. Medicine, vol.25 (1973), p.279.

In Niger Republic, the towns of Maradi and Zinder reported outbreaks on April 4-10 and May 2-8, respectively. Kano and Katsina were the probable sources of the infection. The predominantly rural districts around Maradi and Zinder reported serious outbreaks in April, May and June, 1971. Tessaoua District had a recorded total of 2,213 cases and 619 deaths (28.6% mortality) in ten weeks. There were 1,133 cases in Mayahi, 925 in Tanout, and a lesser number in the other seven infected districts.

The incidence of cholera since 1971 in Nigeria is difficult to assess. There have been no major extensions and only sporadic reports have appeared in the Weekly Epidemiological Record. The considerable spatial dispersion of the reported outbreaks, as well as a newspaper account of an officially-denied outbreak in Kano[46] suggest that cholera continues to well-established in Nigeria and is more serious than officially reported.

Several divisions have not yet reported any outbreak of cholera in Nigeria. Most of these are clustered in the Central and Eastern Middle Belt in North-Eastern and Benue-Plateau States. The apparent cholera-free status of some divisions may result from incomplete reporting. However, these areas are generally far from the major centers of diffusion

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46 F. Ogunleyi, "Filth: A Health Hazard in Kano", Lagos, Daily Times, No.20381 (September 8, 1973), p.7. Ogunleyi reports that cholera is claiming about two victims daily in Kano, although the Medical Officer of Health denies its occurrence.

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[46] F. Ogunleyi, "Filth: A Health Hazard in Kano", Lagos, Daily Times, No.20381 (September 8, 1973), p.7. Ogunleyi reports that cholera is claiming about two victims daily in Kano, although the Medical Officer of Health denies its occurrence.

and tend to have poor transportation connections. Their population density is low and they lack any major urban center. The uninfected areas which do not conform to these characteristics are certain high density but relatively unurbanized divisions in East-Central State.

Analysis of variance and the chi-square test were used to assess the relationship between the presence or absence of cholera in the Nigeria portion of the urban hierarchical diffusion system and six variables. Distance to the initial point of cholera infection (Lagos), total population of the division, urban population of division's largest city, a transportation score, and a composite index of urban importance similar to the one employed in analyzing the Mali epidemic were tested with analysis of variance, while chi-square was used for the nominal variable presence or absence of a major river or coastline.

Five of the variables were found to be significantly related to the occurrence of cholera (Table 3). Only total population was not significant at the .05 level or better. Distance to Lagos and the urban importance score were the two variables found to be most closely related to the presence or absence of cholera. They were significant at the .001 level. Urban population and transportation score were significant at the .05 level.

Pearsonian correlation coefficients were calculated between the above variables and the week of cholera onset. Although the simple correlation coefficients were only .429 or less, urban importance and distance to Lagos were significant at the .01 level, while transport

TABLE 3

STRENGTH OF RELATIONSHIP BETWEEN THE OCCURRENCE OF CHOLERA AND SELECTED VARIABLES IN NIGERIA

	F value
Distance to Lagos	18.42 ***
Urban importance	13.20 ***
Transportation score	6.06 *
Urban population	5.37 *
Total population	3.02
	Chi-square value
Riverine or coastal location	4.31 *

(Significance levels *: .05, **: .01, ***: .001)

TABLE 4

NIGERIA: CORRELATION COEFFICIENTS BETWEEN THE WEEK OF CHOLERA ONSET AND SELECTED VARIABLES

Urban importance	-.429 **
Distance to Lagos	.365 **
Urban Population	-.324 *
Transportation Score	-.294 *
Total population	-.135
Riverine or coastal location	-.053

R = .566 *** (first two variables)

or R = .585 ** (six variables)

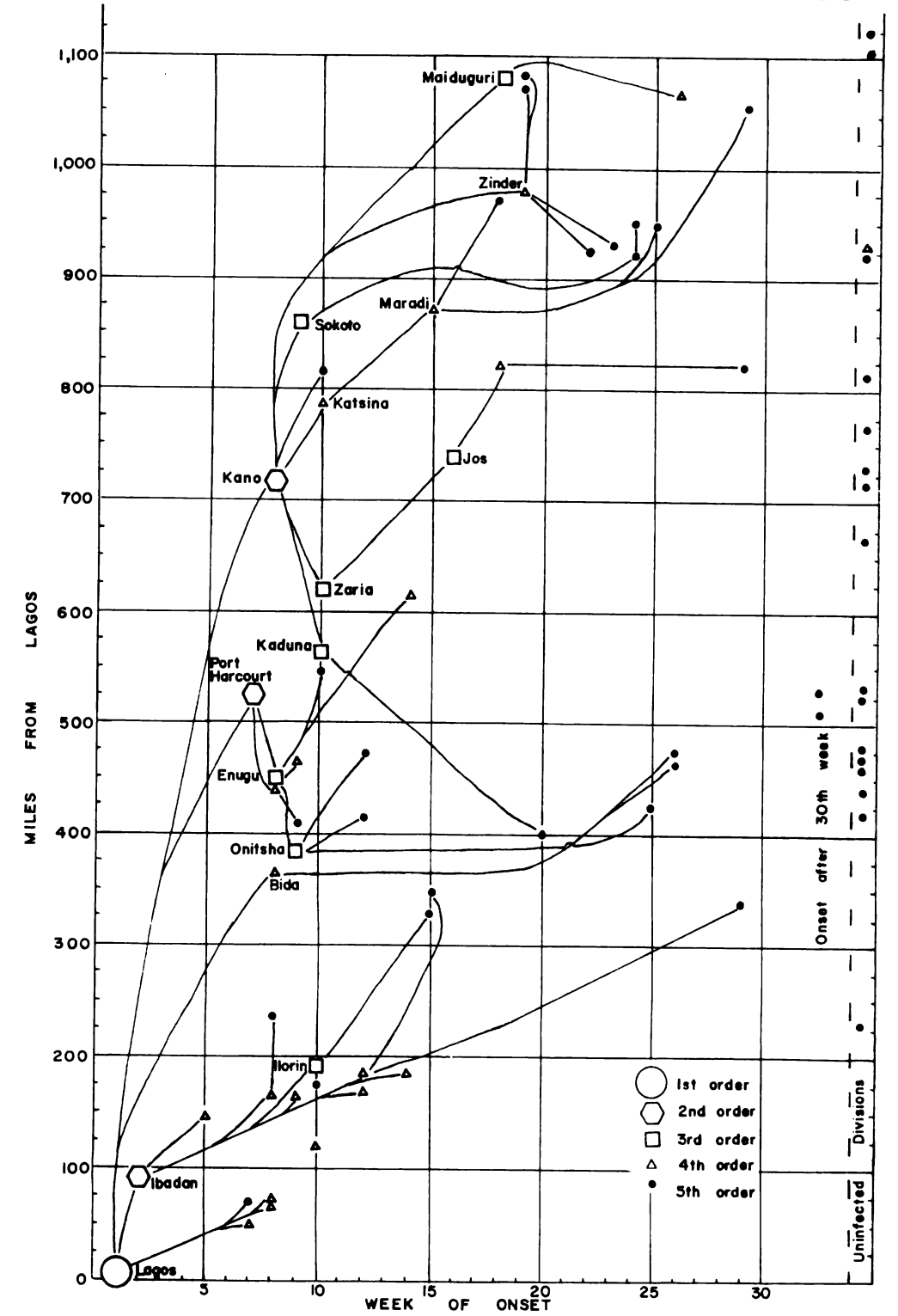
FIGURE 20

DISTANCE, URBAN HIERARCHY AND CHOLERA DIFFUSION
IN NIGERIA AND SOUTH-CENTRAL NIGER

Figure 20 illustrates the joint operation of urban importance and distance from the initial outbreak at Lagos as determinants of the diffusion patterns of cholera in Nigeria and South-Central Niger. The pattern of spread from higher-ordered to lower-ordered urban centers was observed consistently. Lagos, Ibadan, Port Harcourt and Kano were the most important diffusion foci.

FIGURE 20

DISTANCE, URBAN HIERARCHY & CHOLERA DIFFUSION IN NIGERIA & SOUTH-CENTRAL NIGER



score and urban population were significant at the .05 level. The multiple correlation coefficient, R, is .566 for the first two variables and is significant at the .001 level. The other variables contribute virtually no additional explanation.

Figure 20 effectively demonstrates the joint importance of distance from Lagos and position in the urban hierarchy in regulating the pattern of cholera diffusion in Nigeria. The hierarchical diffusion of cholera is also clearly shown. Of the fifty-two diffusion connections shown, forty-eight are from higher to lower ordered centers and four are between places of equal order. In contrast, six of the thirty-five diffusion paths in Mali were from lower to higher-ordered places.

Several generalizations follow which relate to the urban hierarchical diffusion system of Nigeria and South-Central Niger. A systems diagram and schematic map, Figure 21, illustrate these generalizations.

1) The diffusion of cholera in Nigeria was primarily influenced by two variables, namely, distance from the initial focus of infection in Lagos and importance of a city in the urban hierarchy. Urban population and transportation development are also significantly related to the occurrence and speed of onset of cholera.

2) Cholera diffused downward in the urban hierarchy from central places to lower ordered centers in their spheres of influence.

3) Transportation routes, rather than physical features formed the channels of diffusion. The rate of spread was greatest along major

FIGURE 21

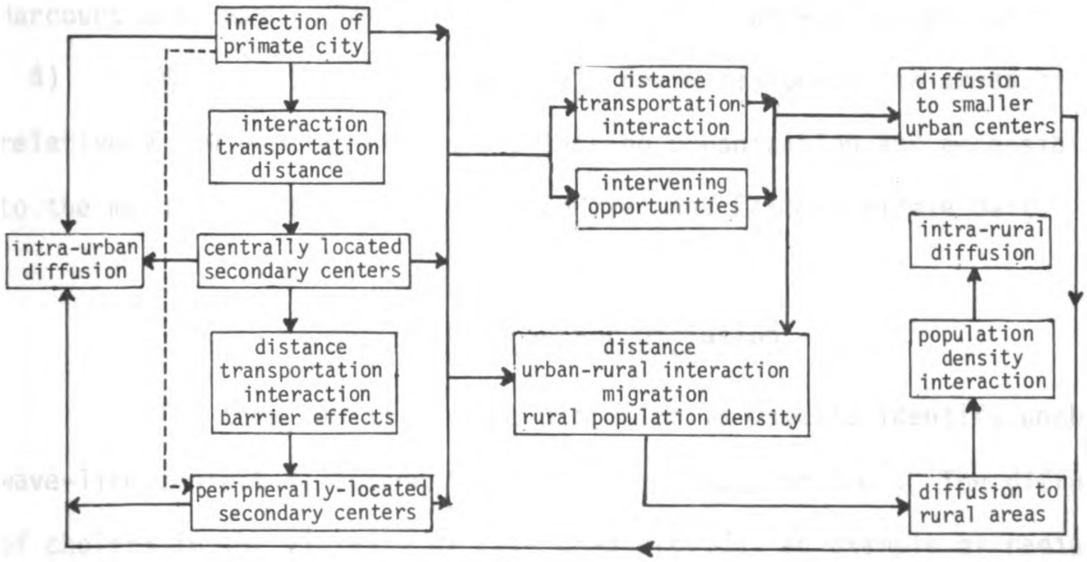
URBAN HIERARCHICAL DIFFUSION SYSTEM
SCHEMATIC MAP OF URBAN HIERARCHICAL DIFFUSION

In an urban hierarchical diffusion system, the pattern of diffusion is from larger to smaller centers within their spheres of influence. Distance, transportation channels, and the amount of interaction are the key controlling factors.

FIGURE 21

(A)

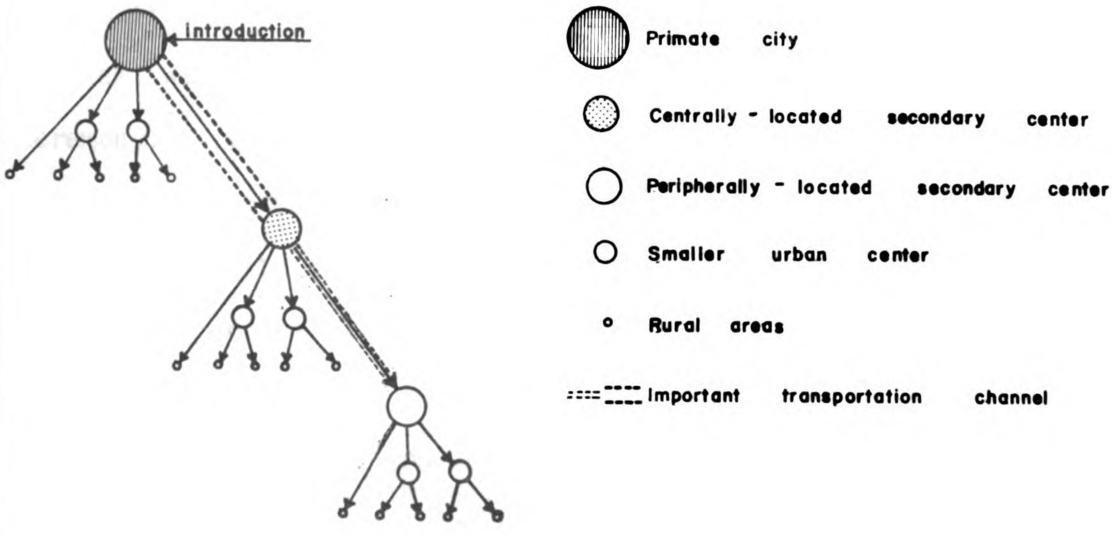
URBAN HIERARCHICAL DIFFUSION SYSTEM



(B)

SCHEMATIC MAP

OF URBAN HIERARCHICAL DIFFUSION



transportation arteries such as exist between Lagos and Kano or Port Harcourt and Enugu. Peripheral central places were affected later.

4) Most of the unaffected divisions were located in areas of relatively low population density lacking urbanization and accessibility to the main diffusion foci in the Central and Eastern Middle Belt.

Radial Contact Diffusion

The term radial contact diffusion is used to identify unchannelled, wave-like contact diffusion from a central focus or foci. The diffusion of cholera in the vicinity of Lake Chad provides an example of radial contact diffusion.

The first major introduction of cholera in the Lake Chad area was on May 7, 1971 at Goulfey, Northern Cameroun. Goulfey is a town of 3,500 located along the Chari River about 100 km. south of Lake Chad. The Goulfey outbreak, which is described more fully in the following chapter, was introduced by visitors coming from Nigeria to a circumcision ceremony.[47] The resulting outbreak was very serious with a total of 800 clinical cases in the town.

Despite stringent control measures which included travel prohibition in the Goulfey area and a mass vaccination program designed

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[47] B. Coulanges and P. Coulanges, "A propos de l'épidémie de choléra du Sultanat de Goulfey (Nord Cameroun: Mai-Juin, 1971). Considérations sur l'épidémiologie et la prophylaxe", Bull. Soc. Pathol. Exotique, vol.65, No.2 (1972), p.216.

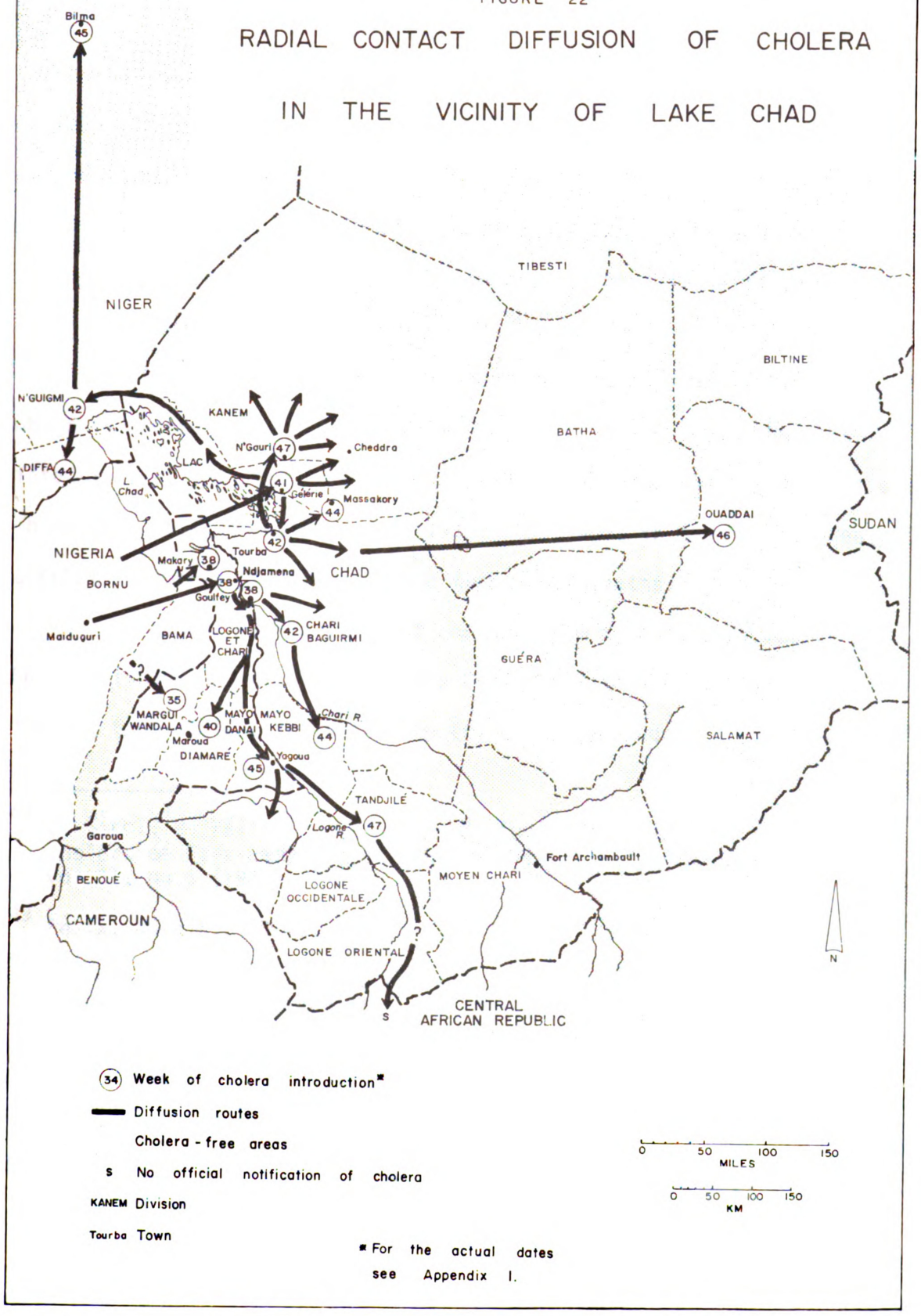
FIGURE 22

RADIAL CONTACT DIFFUSION OF CHOLERA IN THE VICINITY OF LAKE CHAD

The first major outbreak of cholera in the Lake Chad area was at Goulfey on May 5, 1971. Cholera spread from Goulfey south along the Logone and Chari rivers. A second major introduction was at Gélérie on May 25. It initiated a radial contact diffusion pattern resulting from the flight of refugees from cholera-infected villages. Nigeria was the source of the infection in both cases. There were at least four other independent introductions of cholera from Nigeria into Chad and Northern Cameroun.

FIGURE 22

RADIAL CONTACT DIFFUSION OF CHOLERA IN THE VICINITY OF LAKE CHAD



to protect Ndjamena (Fort Lamy), Chad[48], cholera quickly reached Fort Foureau (Cameroun) and Ndjamena. It subsequently spread south of these two centers along the Logone and Chari Rivers. The farthest confirmed penetration was at Kim in Tandjilé District (Chad), some 350 km. south of Ndjamena.[49] There is an unofficial report of the disease reaching the Central African Republic in June, 1971.[50]

A second introduction in Northern Cameroun was at Makary, which is located very close to Lake Chad. It is suspected that the infection was imported from Nigeria as a result of smuggling activities.[51] There were related outbreaks in fifteen villages in the Makary area, with a total incidence of 102 cases and twenty-six deaths.

Another introduction of cholera from Nigeria occurred at Gélérie (Chad), 50 km. east of Lake Chad. A Nigerian family, apparently fleeing from cholera in their home town, arrived and died on May 25, 1971.[52]

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[48] J. Sirol, H. Félix, P. Delpy and O. Bono, "A propos de l'épidémie de choléra de Fort-Lamy (Tchad) en Mai-Juin, 1971", Médecine Tropicale, vol.31, No.6 (1971), p.630.

[49] Félix, op.cit., 1971D, p.623.

[50] H. Félix, "Épidemie de choléra en Afrique (suite). Évolution de la situation pendant le premier semestre, 1971", La Presse Médicale, vol.79, No.41 (1971B), pp.1801-1804.

[51] B. Coulanges and P. Coulanges, "A propos de l'épidémie de choléra du Sultanat de Goulfey (Nord Cameroun: Mai-Juin, 1971)", Médecine Tropicale, vol.31, No.6 (1971), p.639.

[52] O. Bono et al., "Installation du choléra aux alentours du Lac Tchad", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971A), p.391.

A very serious epidemic ensued in Gélérie. Fleeing survivors carried the disease north, east and south of Gélérie. Within forty-eight hours of the arrival of a refugee there would be a new outbreak in the village, promoting a new wave of refugee movement.[53]

There were at least two other independent introductions from Nigerian markets to villages on the Chad side of the Lake.[54]

In addition to the dissemination of cholera in the Lake Chad vicinity, the infection reached Ouaddai Division which borders the Republic of Sudan in early July, 1971. There were reports of only four cases and three deaths from Ouaddai.

Cholera spread to N'Guigmi District in South-Western Niger during the first week of June, 1971. Cholera outbreaks were reported in N'Guigmi and Diffa Districts near Lake Chad and in Bilma, some 500 km. north of N'Guigmi. The infection of Bilma is probably related to the large trade in unrefined salt originating in Bilma.

The chain reaction flight of survivors from infected villages was prompted by the virulence of the epidemic. The official count of victims showed 8,225 cases and 2,337 deaths (28.9% mortality) in Chad in 1971. There were 4,836 cases and 1,586 deaths among 40,000 widely dispersed inhabitants of a 50,000 sq.km. area around Gelerie.[55]

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[53] Bono, op.cit., p.392.

[54] Bono, op.cit., p.395.

[55] O. Bono, et al., "Installation du choléra aux alentours du Lac Tchad", La Presse Médicale, vol.79, No.54 (1971B), p.2484.

Even if the entire 40,000 population had been vibrio carriers, the carrier to case ratio would be barely eight to one. In Goulfey there were 800 cases among 3,500 inhabitants (4.4 possible carriers per case). While such ratios would not be unusual with classical vibrio cholerae, they are extremely low for cholera El Tor epidemics which normally have twenty-five to one hundred carriers per clinical case. The explanation of the large incidence of clinical cholera lies in massive, undiluted vibrio pollution in the affected villages.

In some villages, as many as half to three quarters of the population died after a very short incubation period.[56] Tourba, a village of 1,000 south of Gélérie had 500 cases and 250 deaths. There were sixty-four cases and twenty-five deaths in Delboumou, a village of 150 inhabitants, within a twenty-four hour period.[57] These levels of morbidity and mortality appear to have been unprecedented in Asia as well as Africa for cholera El Tor.

The epidemic in the vicinity of Lake Chad has been cited as a definite example of person to person spread.[58] The infection was sufficiently massive to facilitate person to person spread. Cases were documented in which persons who had participated in the burial of cholera

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[56] Bono, op.cit., 1971B, p.2482.

[57] Bono, op.cit., 1971A, p.393.

[58] Bono, op.cit., 1971A and 1971B, Félix, op.cit., 1971B and 1971C, and Coulanges, op.cit., 1972.

victims became sick themselves.[59] Additional support for the hypothesis of person to person spread comes from Goulfey where a group of several hundred children who were isolated for the epidemic except when receiving food and water completely escaped being infected.[60] It has been stated that the high temperature and lack of water due to the late dry season timing of the epidemic exclude the possibility of water playing any role in the spread of cholera.[61] However, water was not completely absent. The water shortage could actually facilitate diffusion by concentrating people around the remaining water sources. Maximum contamination of these water sources would be assured by the small volume of water in them and the absence of sanitation around public wells. Once a well was infected, cholera vibrios could easily be transferred to wells in other towns in the leather pouches commonly used to draw water. Because they remain moist, such pouches would provide an ideal environment for vibrio survival. It is probable that cholera spread as a result of environmental contamination, as well as person to person spread.

The wave-like contact diffusion pattern near Lake Chad probably resulted from the lack of prominent urban centers, the poor transportation system and absence of natural barriers and channels. The diffusion, as a result, was not directed along particular corridors or toward particular urban centers. Instead, it went along the numerous paths chosen by individual refugee-carriers. However, a certain amount of

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59 Bono, op.cit., 1971B, p.2484.

60 Coulanges, op.cit., 1972, pp.223-224.

61 Félix, op.cit., 1971D, p.623.

directionality results from the fact that refugees would tend to flee away from the perceived source of the epidemic, and hence would tend to go inland rather than toward Nigeria or the shore of Lake Chad.

The incidence of cholera dropped sharply in late June and had virtually ended by late July. The second half of June would correspond to the usual beginning of the rainy season in the affected areas. It is hypothesized that the increased difficulty of travelling in the rainy season, plus the necessity of returning home to plant crops would overcome the urge to flee from cholera. This would effectively isolate the infection in small areas from which it would disappear with the exhaustion of the local cholera-susceptible population.

Radial contact cholera diffusion also occurred in Northern Kenya. Like the Lake Chad area, Northern Kenya has low population density, nomadic population, poor communications and an arid climate.

The following generalizations may be made about radial contact diffusion in the Lake Chad area:

1) The epidemic around Lake Chad had the highest morbidity and mortality in West Africa. As a result of this virulence, survivors fled infected villages and carried the infection with them.

2) Cholera spread radially outward from the centers of introduction. The lack of transportation, urban centers, barriers and channels resulted in a radial contact type of diffusion.

3) The flight of refugees away from the source of the epidemic established directional bias in the diffusion pattern.

FIGURE 23

RADIAL CONTACT DIFFUSION SYSTEM
SCHEMATIC MAP OF RADIAL CONTACT DIFFUSION

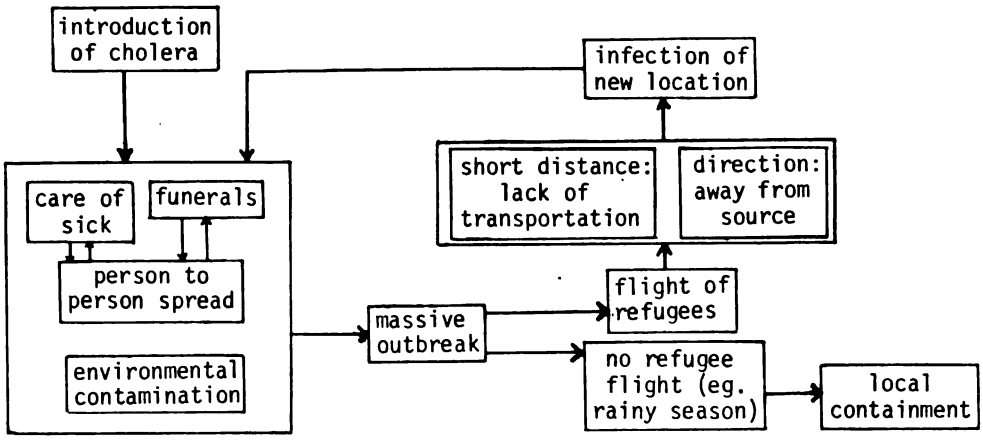
The model portrays the essential features of radial contact diffusion found in Chad. The diffusion pattern was outward from the center of introduction, and was not directed along physical or transportation channels. Because of the lack of prominent central places, it was not directed toward particular centers.

The pattern of radial contact diffusion near Lake Chad is similar to the diffusion sequence in Northern Kenya.

FIGURE 23

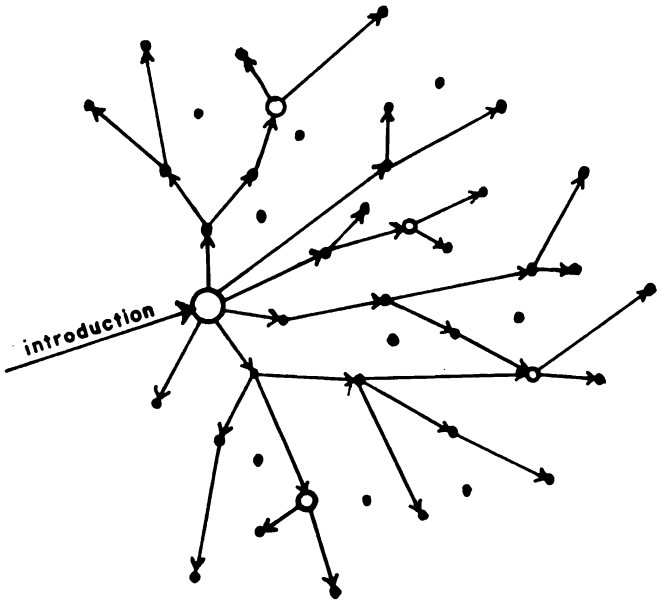
(A)

RADIAL CONTACT DIFFUSION SYSTEM



(B)

SCHEMATIC MAP OF RADIAL CONTACT DIFFUSION



4) There are indications of person to person spread, some as a result of the burial of cholera victims. However, spread through contamination of water sources cannot be ruled out.

5) The epidemic ended in the rainy season, possibly as a result of the seasonal decrease in travel.

CHAPTER 6

CASE STUDIES ILLUSTRATING THE LOCAL DISSEMINATION OF CHOLERA

The tracing of cholera diffusion of cholera within a community presents special problems. Symptomless carriers, who generally account for over 95% of all cholera victims, act as a reservoir of infection and as vibrio spreaders. It is virtually impossible to locate more than a small percentage of symptomless carriers or evaluate their impact during an epidemic because they show no symptoms and generally are vibrio-positive for only one or two days. The incidence of symptomless infections is also important because antibody levels of carriers are raised. As a result the size of the cholera-susceptible population decreases in proportion to the total number of carriers as well as cases.

The variety of possible means of cholera dissemination also complicates the tracing of intra-community spread. In the classical model of cholera dissemination, it progresses through a man-environment transmission cycle which usually involves infected water, but may also involve vibrio-infected goods, food or soil and transmission by flies. Cholera may also be spread through direct person to person contact. Because there are a variety of possible transmission processes, it is very difficult to trace the transmission cycle in a particular epidemic. The difficulty is greatest in large urban centers where there is high population density, greater opportunity for inter-human contact and exposure to a complex environment with a variety of possible sources of cholera infection.

A third source of difficulty, when published descriptions and analyses of particular epidemics are being used as a primary source of information is the failure of most medical researchers to take a holistic view. As a rule, spatial, environmental and cultural factors are ignored or considered very superficially. Because the survival and diffusion of cholera is a complex process, a holistic approach is essential.

This chapter consists of case study accounts of cholera in local areas in a variety of physical environments and involving different predisposing and precipitating factors. The case study locations are Lagos (Nigeria), Ibadan (Nigeria), Zaria (Nigeria), Goudoumé (Ivory Coast), Louga (Senegal), Akodesséwa-Plage, Togo, and Goulfey (Cameroun). The Lagos case study, which is based mainly on primary data, is more extensive than the others which were based entirely on secondary sources.

Lagos, Nigeria

Because of its location adjacent to a lagoon, its large population, busy commercial life, high incidence of crowded slum housing and the total inadequacy of municipal water, sewage and garbage disposal systems, Lagos provides virtual model environment for cholera survival and diffusion.

Cholera was first diagnosed just outside the urban fringe in a riverine village on December 26, 1970 (Figure 24). Another case was found in the Lagos suburb of Mushin on the same day. The onset of cholera

FIGURE 24

VILLAGE AT MOUTH OF OGUN RIVER, MILE 15
ON LAGOS-SHAGAMU HIGHWAY

The first outbreak of cholera in Nigeria involved five members of one family. It occurred at Ajegunle-Owode village on December 26, 1970. The photograph shows a scene very close to Ajegunle-Owode. It illustrates environmental conditions which proved to be conducive for cholera survival throughout coastal West Africa, namely, a low-lying fishing village adjoining a brackish lagoon and lacking any protected water supply or sewage disposal facilities.

corresponded to a city-wide breakdown of the water supply which forced people to seek water from wells and polluted streams.[1] Cholera quickly became established in Lagos Metropolitan Area and has continued to occur there. Between December 26, 1970 and September 2, 1972, there were 4,520 bacteriologically confirmed cases and 245 deaths from cholera in Lagos State.[2] It is probable that over nine tenths of these were in the Lagos Metropolitan Area. Between September, 1972 and August, 1973 a smaller number of cases continued to be reported, but in contrast to 1971, cholera was no longer considered among the most urgent health problems of the state.[3]

The epidemic curve of cholera morbidity during the first twenty-two lunar months (four weeks each) is shown in Figure 27B. There were four epidemic periods between December, 1970 and September, 1972. These occurred in January and February, 1971, July, 1971, October, 1971 and July, 1972. The number of cases in a four week period has ranged from a low of twelve in March-April, 1972 to a high of 657 between July 11 and August 7, 1971.

A list of the first 800 cholera victims in Lagos State (December 26, 1970-March 20. 1971)[4] was studied to obtain patterns of cholera

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[1] "Swift bid to restore normal water flow to Lagos", Daily Times, Lagos, No.19460 (January 1, 1971), p.1.

[2] B.A.A. Dada, "Cholera in Lagos State: 26/12/70-2/9/72", Lagos: Ministry of Health and Social Welfare, 1972.

[3] Personal communication, Dr. B.A.A. Dada, Lagos State Ministry of Health, August 17, 1973.

[4] "Positive cholera cases and deaths in Lagos State". Lagos: Ministry of Health and Social Welfare, 1971.

occurrence and spread in Lagos during the early stages of the epidemic. The age, sex, date and place of admission and home address were included in the list. Summary tables on the first sixteen weeks and 922 cases in Lagos State provided an additional source of information.[5]

Spatial distribution of cholera in Lagos

It was expected that the highest incidence of cholera would be found in crowded areas with slum housing. The main concentrations of poor and crowded housing are on Lagos Island near the Central Business District and more recently developed low class, high density suburbs such as Mushin, Somolu and Ajeromi on the northern and western periphery of Lagos. Conversely, it was expected that the lowest incidence would be in areas of modern, high class, low density housing. Ikoyi and Victoria Island (Ward G), Apapa and much of Surulere are such areas. The remaining areas, namely Yaba and Ebute Metta are composed of predominantly medium class housing and were expected to have moderate attack rates.

Mabogunje has described the old areas of Lagos Island as being "indescribably squalid. Access to many houses is by narrow footpaths which also serve as drains for household water. Household equipment and facilities are most inadequate, and even where they exist they are very unsatisfactory."[6] The density of population is very high. The local community of Inasa Court in Central Lagos has twenty-three houses on

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[5] B.A.A. Dada, "First sixteen weeks of cholera in Lagos State", J. Soc. Hlth. Nigeria, vol.6, No.3 (1971B), pp.133-138.

[6] A.L. Mabogunje, Urbanization in Nigeria, London: University of London Press, 1968, p.304.

FIGURE 25

HOUSING DENSITY IN CENTRAL LAGOS

This photo, which was taken in a market area near the edge of the Central Business District of Lagos shows the narrow streets, dilapitated housing and crowded conditions which typify the high density areas of Lagos Island.

about one acre, with an estimated population of 600 people.[7] In the peripheral low class suburbs the acute shortage of piped water was expected to be reflected in increased incidence of cholera. The fact that infant mortality in Peripheral Lagos is 172 per 1,000 live births compared to 79 per 1,000 in the inner core[8] is a clear indication of the poor conditions prevailing in Peripheral Lagos.

During the initial sixteen week period for which ward-level data are available, there was no clear correlation between the incidence of cholera and the quality of housing.[9] If the 1963 census population data are used to calculate morbidity rates, the highest rates are found in Ajeromi, Lagos Wards G and H and Ward F on the mainland. The location of the Wards is shown in Figure 26A. The population of Lagos, however, has grown rapidly since 1963, and this expansion has not been uniform. Therefore, 1970 rates based on the 1963 populations have little validity.

In the absence of published post-1963 data, the 1971 populations have been estimated, based on personal observation during several visits to Lagos between 1968 and 1973. Because of the high population density in central Lagos in 1963 and the expansion of non-residential functions around the Central Business District, the population of Lagos Island has

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[7] Mabogunje, op.cit., p.304.

[8] U.L.C.M. Cholera Research Team, "Prevalence and behaviour of vibrio cholerae in a newly infected country (Nigeria)", J. Nigeria Med. Assoc., vol.1, No.3 (1971), p.144.

[9] Maps of housing quality are found in Mabogunje, op.cit., p.304 and in P.O. Sada, Metropolitan Region of Lagos, Nigeria: A Study of the Political Factor in Urban Geography. Indiana University, Ph.D. dissertation, 1968, p.203.

FIGURE 26

SEQUENCE OF CHOLERA ONSET IN LAGOS

The map on the left show the location of the divisions and wards which comprise Metropolitan Lagos. The map also shows the extent of the four regions referred to in Figure 27.

The second map shows the sequence of cholera onset in Lagos. The earliest occurrences were in Mushin, Surulere, and Ward H. Such peripheral areas as Ward G, Apapa and Agege were infected at a late stage. The arrows show the hypothesized pattern of spread.

FIGURE 26

SEQUENCE OF CHOLERA ONSET IN LAGOS

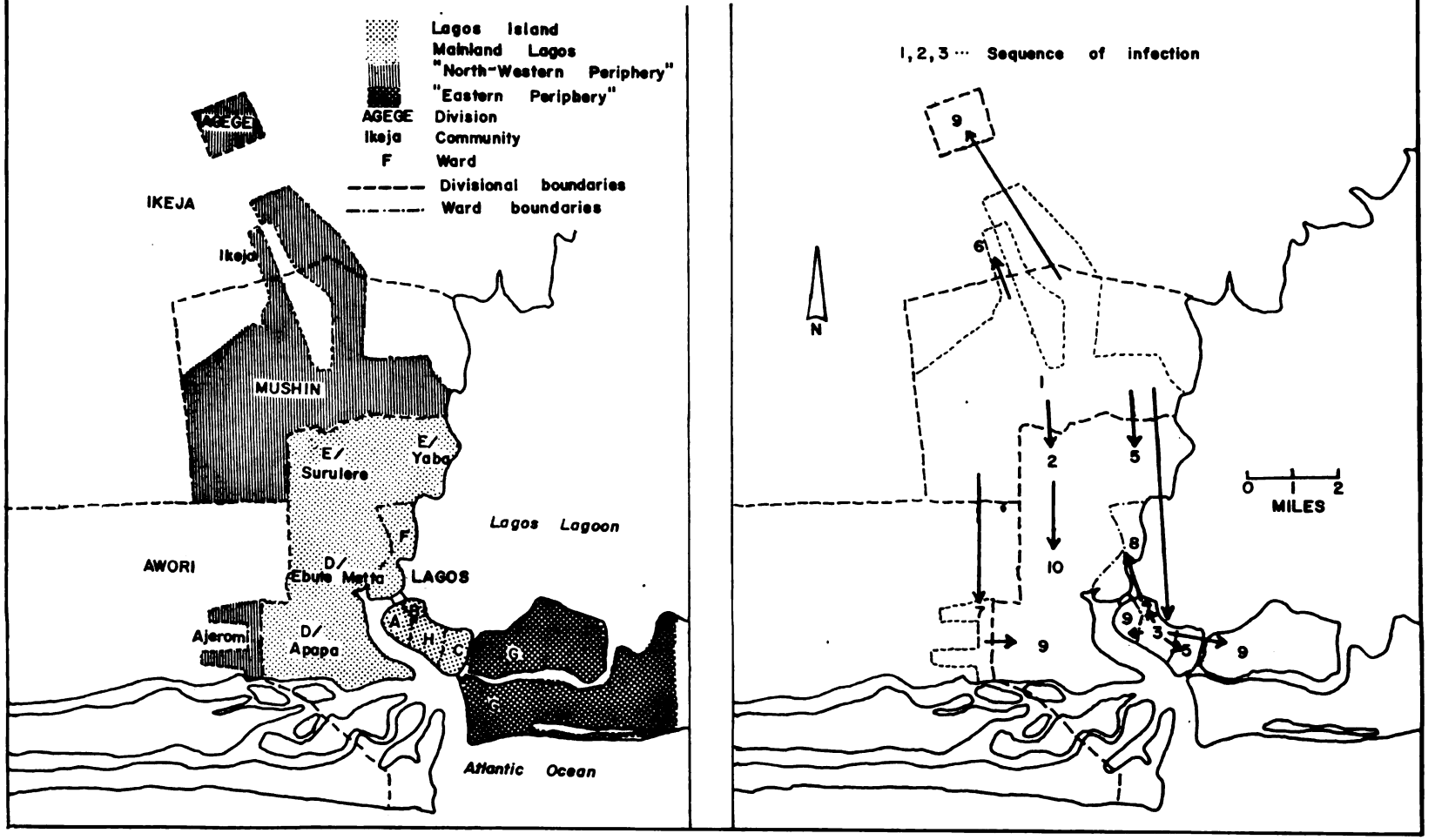

TABLE 5

Cholera morbidity in Lagos State by district or ward during the first sixteen weeks of the 1970-71 outbreak

District/ward	No. of cases in first 16 weeks #	Population(1,000) 1963	Population(1,000) 1971(est.)	Morbidity per 1,000 1963	Morbidity per 1,000 1971 (est.)	Housing quality*
Lagos Island						
A	23	48	50	.48	.46	L
B	64	79	80	.81	.80	L
C	21	53	50	.39	.42	L
H	80	71	70	1.24	1.15	L
Ikoyi-Victoria Is.						
G	62	50	100	1.13	.62	L,H
Mainland						
D/EB	19	82	150	.58	.32	L,M,H
D/Apapa	29					
E/Surulere	52	158	350	.78	.35	M,H
E/Yaba	72					
F	59	58	80	1.01	.74	L,M
Peripheral suburbs						
Mushin	220	312	600	.71	.37	L
Ikeja	56	175	350	.32	.16	M,H
Agege	16	46	100	.35	.16	L
Ajeromi	81	52	150	1.59	.54	L
Non-urban areas						
Awori	16	70	100	.23	.16	
Epe	16	190	250	.22	.16	
Ikorodu	25					
Total	922	1,444	2,500	.64	.37	

After Dada, 1971B.

* L = Low, M = Medium, H = High.
After Sada, 1968, p.203 and Mabogunje, 1968, p.307.

probably changed little since 1963. Most of the growth has been in such peripheral communities as Mushin, Ikeja, Surulere, Agege and Ajeromi District north and west of Central Lagos and to the east in Ikoyi and Victoria Island. Rates based on the 1971 estimates still show the highest morbidity occurring in Central Lagos and Ward F. The incidence in Mushin and Ajeromi is less than expected considering the low standard of living in these suburbs. For example, whether the morbidity estimate is based on 1963 or 1971 population data, the rate for Mushin does not differ significantly from those of the higher class, lower density districts of Mainland Lagos (Wards D and E). The Mushin estimates are approximately half the G ward rate. Ward G includes the most prestigious residential districts of Lagos, but includes such low-class neighborhoods as Ijeh, Maroko and Obalende. Most of Ward G's cases were in these low-class areas.

The distribution of cholera by ward during the first sixteen weeks of the epidemic did not conform to the expected pattern, based on housing conditions, water supply, sanitation and infant mortality. The pattern also conflicted with the results of a stratified random sample survey of 5,467 Lagos residents conducted in search of symptomless carriers. The survey, which was done between mid-February and mid-April, 1971 showed that Mushin and Ward F had the greatest concentration of vibrio carriers.[10]

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[10] U.L.C.M., op.cit., pp.143-144.

Diffusion of cholera within Lagos

Figure 26B shows the sequence of cholera onset in the wards and districts comprising the Lagos Metropolitan Area. Mushin was the first district to report cholera. The adjoining Surulere Ward was next, followed by Ward H in Central Lagos. The pattern of spread shows outward movement from Mushin-Surulere and Central Lagos. The last wards to be infected were Ebute Metta, Apapa, Agege, Ward G and Ward A.

Epidemic curves of the early stages of the Lagos outbreak were studied as a possible source of information on the spread of cholera within the Lagos Metropolitan Area. Epidemic curves were plotted for four Lagos regions, namely, Lagos Island, Mainland Lagos (Apapa, Surulere, Yaba and Ebute Metta), the Eastern Periphery (Ikoyi and Victoria Island) and the Northern and Western Periphery (Mushin, Ikeja, Agege and Ajeromi. Three day running means of cholera morbidity in these regions were plotted for the period December 26, 1970 to March 19, 1971 (Figure 27A). The three day running means have been used to reduce the impact of daily fluctuations.

The pattern of the epidemic curves is a complex one, with multiple peaks in each region. However, the sequence of peaks appears to indicate the source and progression of infection during a thirteen week period.

The first significant peak was on January 7, 1971 in Lagos Island. It was preceded by two smaller peaks on December 29 and January 3 which represent the primary and secondary seeding periods. The first

FIGURE 27

EPIDEMIC CURVES OF CHOLERA IN THE FOUR REGIONS
OF THE LAGOS METROPOLITAN AREA;
DECEMBER 26, 1970-MARCH 19, 1971

The epidemic curves of the four regions of Lagos shown in Figure 26A have been superimposed. They show a pattern of shifting epidemic centers. In period one, cholera peaks in Lagos Island are followed by peaks in other regions. The initial peaking occurs in the North-Western Periphery in period two and in Mainland Lagos in the third period. The inset graph shows the incidence of cholera cases in Lagos State between December, 1970 and September, 1972.

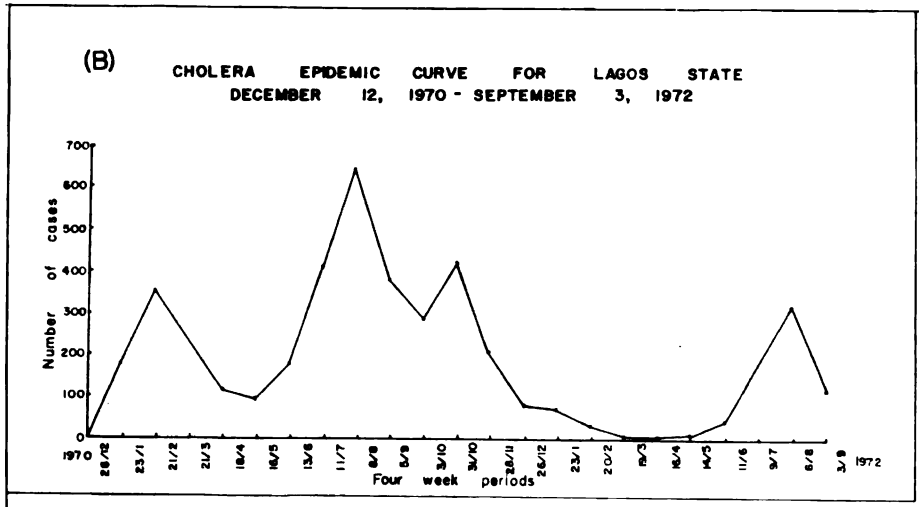
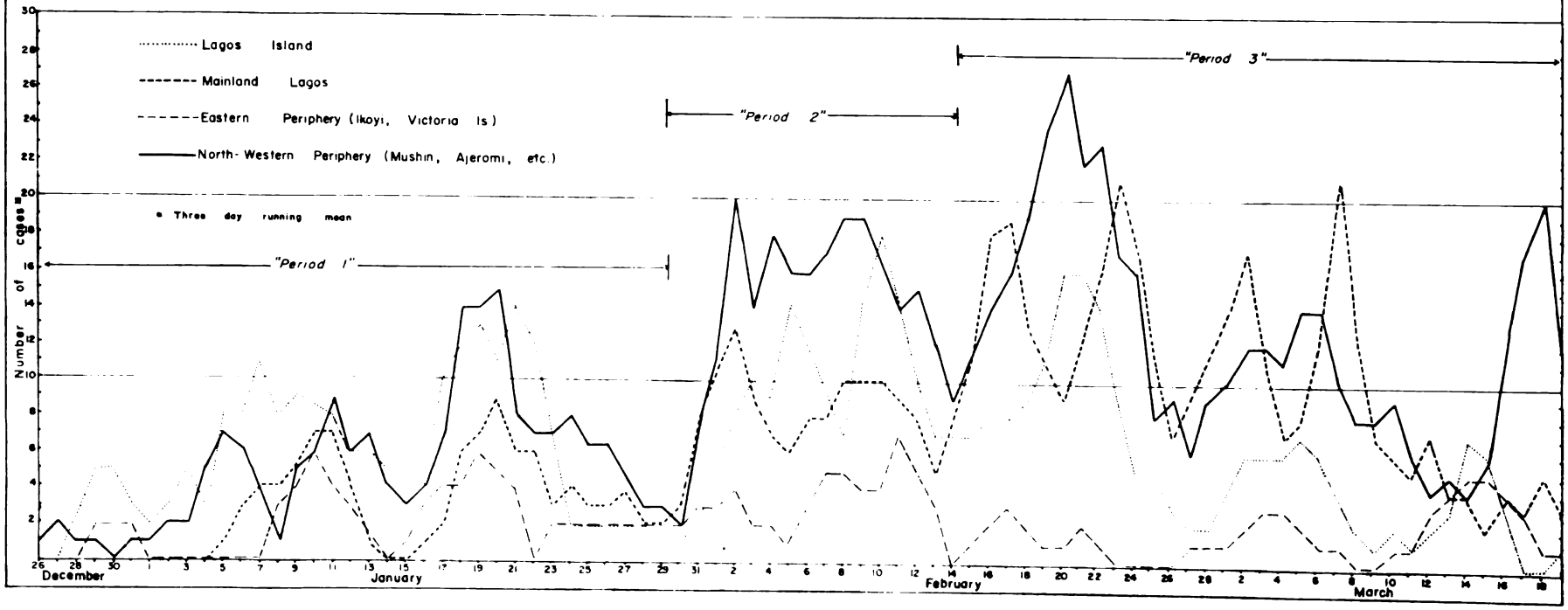


FIGURE 27

EPIDEMIC CURVES OF CHOLERA IN THE FOUR REGIONS OF THE LAGOS METROPOLITAN AREA

DECEMBER 26, 1970 - MARCH 19, 1971



major peak in the North-Western Periphery was on January 20. It was also preceded by two smaller seeding period peaks. Mainland Lagos had two early peaks on January 16 and February 27. They occurred at the same time as peaks in the North-Western Periphery and slightly after peaks on Lagos Island. The first peak in the Eastern Periphery, which is adjacent to only Lagos Island, followed the first major Lagos Island peak. Thus, during January, the earliest peaking of the epidemic was in Lagos Island, followed by peaks in other sections of the Lagos Metropolitan Area. These later peaks are thought to be the result of the transfer of infection from an earlier-infected source, namely, Lagos Island.

At the end of January, there was very low incidence of cholera in all parts of Lagos. However, there was an outbreak at this time just north of the city in the vicinity of the initial cholera cases in Lagos State. The infection may have been introduced to Lagos for a second time, as this second outbreak on the outskirts of the Metropolitan Area was followed by a new rise in cholera incidence within Metropolitan Lagos. During this second epidemic, the sequence of peaking was reversed. It was the North-Western Periphery and Mainland Lagos which peaked first (February 1) followed by Lagos Island on February 5 and February 11. Minor peaks in the Eastern Periphery followed on February 7 and February 11.

The next major peak was in Mainland Lagos on February 17, followed by simultaneous peaks on February 20 in Lagos Island and the North-Western Periphery. February 15-17 therefore appears to be the beginning of a third phase in which Mainland Lagos had become the center of diffusion from which cholera spread to Lagos Island and the North-Western Periphery. Mainland Lagos had four major peaks between February 16 and March 7. Small peaks in Lagos Island and the North-Western Periphery on March 5 followed an epidemic peak in the Lagos Mainland. After about a week of low activity there was a major peak in the North-Western Periphery on March 18. Data inavailability prevented consideration of its impact on adjoining regions.

The sequence of peaks suggests that first Lagos Island, followed by the North-Western Periphery, and then Mainland Lagos acted as source areas for new epidemics in the other regions. It is hypothesized that vibrio contamination was relatively heavy throughout Metropolitan Lagos, and that the introduction of new infection from an adjoining region would be comparable to adding water to a full bucket, i.e. it would trigger an epidemic. The multiple peaks are therefore thought to represent essentially a chain reaction of repeated inter-regional diffusion within the city. It is perhaps significant that Mainland Lagos which is located centrally between Lagos Island and the North-Western Periphery had eight relatively clear peaks between January 6 and March 6, compared to only six in Lagos Island and seven in the North-Western Periphery.

The epidemic curves seemed to indicate the existence of patterns of spread between regions of the city. A single region was selected and the early cases of cholera were mapped to find if similar patterns of diffusion could be found within a city area. Lagos Island was selected because of its physical separation, except for bridges, from the other regions. The early occurrence of cholera on Lagos Island, between December 29, 1970 and February 14, 1971, is shown in Figure 28. The sequence of cases has been divided into cohorts of approximately ten each.[11] The homes of the first ten cohorts have been mapped.

Cholera was initially concentrated in the center of Lagos (period one, Figure 28A). The first case in Lagos Island was within a block of Tinubu Square which is the focal point of the city. During the ensuing second, third and fourth periods cholera moved outward in all directions toward the edge of the Island. After this explosion from the center of the island outward the epidemic wave seemed to implode in periods five and six. The new cases were found ever closer to the center of the Island again. The pattern is less obvious during periods seven through ten. In periods seven and nine, the infection seemed to be relatively scattered toward the edge of the Island, while there appeared to be greater clustering in the center of the Island during periods eight and ten. The areas which had the heaviest incidence of cholera are in the center and north-western part of Lagos Island. These are high density residential areas.

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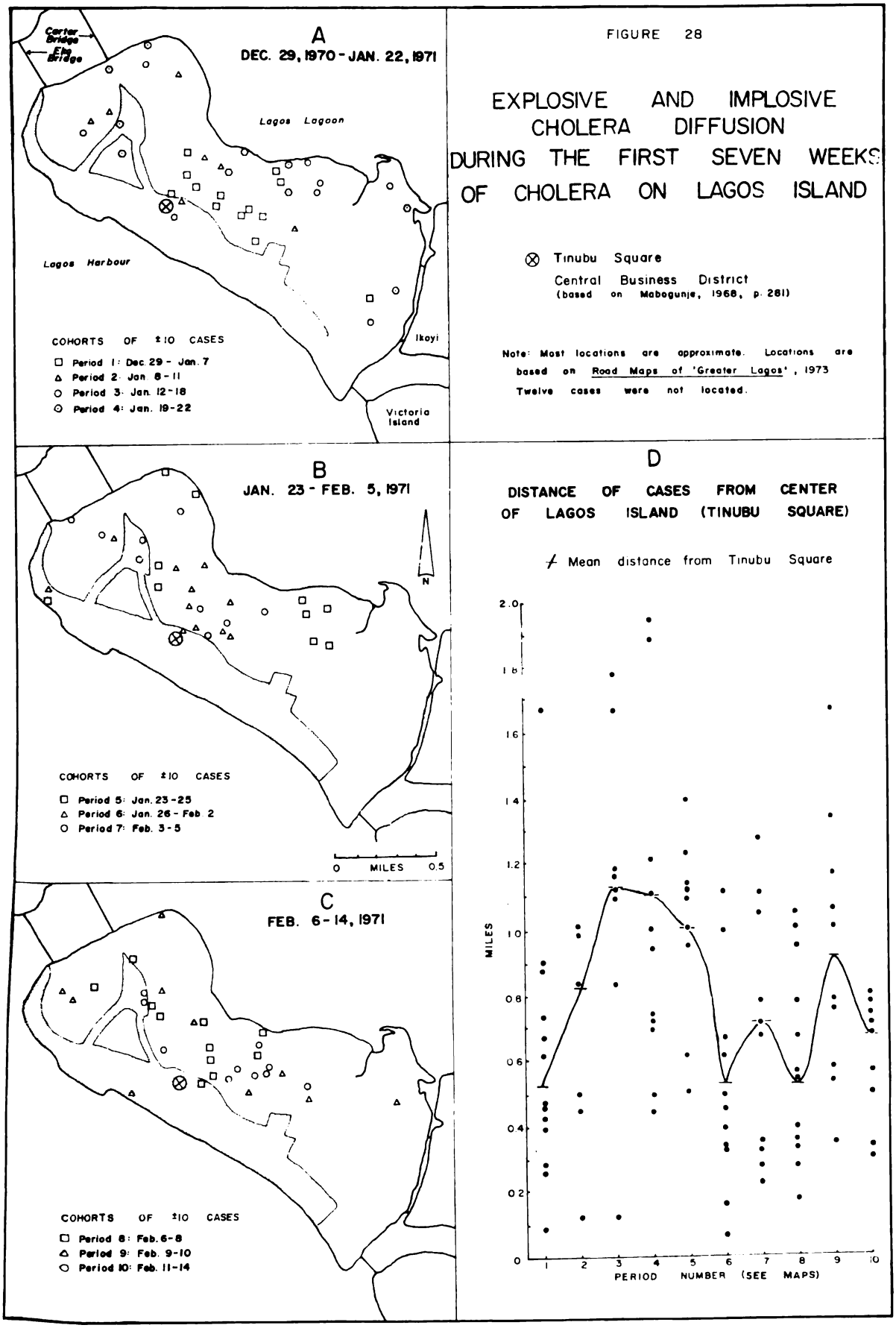
[11] Because all cases reported in one day were considered as a single unit, some groups contain slightly more or less than ten cases.

FIGURE 28

EXPLOSIVE AND IMPLOSIVE CHOLERA DIFFUSION DURING THE FIRST SEVEN WEEKS OF CHOLERA IN LAGOS ISLAND

The locations of ten successive cohorts of ten cases each have been mapped. Beginning in the center of Lagos Island, cholera had spread outward to the edge of the island by periods three and four. The epidemic wave then reversed itself and imploded toward the center of the island (period six). The graph shows the dispersion and mean distance of cases from Tinubu Square which is the focal point of the Lagos Central Business District. The graph clearly shows the explosive and implosive diffusion of cholera in periods one to six and the less spectacular fluctuations characterizing periods seven to ten.


A
DEC. 29, 1970 - JAN. 22, 1971
Carter Bridge
Eko Bridge
Lagos Lagoon
Lagos Harbour
Ikoyi
Victoria Island
COHORTS OF ±10 CASES
Period 1: Dec. 29 - Jan. 7
Period 2: Jan. 8 - 11
Period 3: Jan. 12 - 18
Period 4: Jan. 19 - 22
B
JAN. 23 - FEB. 5, 1971
N
COHORTS OF ±10 CASES
Period 5: Jan. 23 - 25
Period 6: Jan. 26 - Feb. 2
Period 7: Feb. 3 - 5
0 MILES 0.5
C
FEB. 6 - 14, 1971
COHORTS OF ±10 CASES
Period 8: Feb. 6 - 8
Period 9: Feb. 9 - 10
Period 10: Feb. 11 - 14
FIGURE 28
EXPLOSIVE AND IMPLOSIVE CHOLERA DIFFUSION DURING THE FIRST SEVEN WEEKS OF CHOLERA ON LAGOS ISLAND
⊗ Tinubu Square
Central Business District
(based on Mabogunje, 1968, p. 281)
Note: Most locations are approximate. Locations are based on Road Maps of 'Greater Lagos', 1973
Twelve cases were not located.
D
DISTANCE OF CASES FROM CENTER OF LAGOS ISLAND (TINUBU SQUARE)
≠ Mean distance from Tinubu Square
MILES
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0
1 2 3 4 5 6 7 8 9 10
PERIOD NUMBER (SEE MAPS)

The southern and south-eastern portions which have the fewest cases comprise the Central Business District and governmental areas.

The dispersion of cases from the focal point of Lagos Island, Tinubu Square, was measured for each cohort. Figure 28D shows the dispersion of cases and the mean distances of cases from Tinubu Square in each period. This graph confirms that the infection exploded away from the center of the island and after the fourth period began to implode again toward the center.

The explosion-implosion sequence described above is analogous to the behavior of a physical wave which is reflected by an impermeable barrier. The analogy is attractive because the water surrounding Lagos Island is a definite barrier to the spread of the infection. However, the use of a physical wave analogy does not explain why an epidemic wave should appear to behave in such a way. It does not explain why the outward-spreading infection should not "pile up" along the waterfront and cause a large epidemic there rather than receding from the edges of the Island toward the center. This distinctive explosive-implosive pattern of cholera dissemination on Lagos Island therefore remains unexplained.

Neither the regional epidemic curves (Figure 27A) or maps provide evidence about the method of cholera dissemination. The long persistence of the epidemic suggests the existence of a well-established man-environment transmission cycle, as opposed to person to person spread. The large water frontage and grossly inadequate sanitation would facilitate the establishment of such a cycle. The wide dispersion of cases suggests

that carriers were instrumental in disseminating the infection from endemic foci throughout the city. If carriers had not played an important role in diffusion, cases would have tended to be more highly concentrated around endemic foci. The numerous food sellers, whose products are prepared and sold in very unhygienic conditions may also have played an important role in the dissemination of cholera in Lagos.

Age distribution of cholera victims

The age distribution of cholera victims in Lagos State in the first sixteen weeks of the epidemic corresponded to the age patterns found elsewhere in Africa and to the pattern expected for a newly-infected area. The highest incidence was among young adults, with 47.8% of the first 922 cases being among persons aged twenty-one to forty. Normally, adult males have the highest attack rates due to their greater mobility and exposure to the risk of infection.[12] However, adult female cases out-numbered males in Lagos. This may be related to the female domination of market trading among the Yoruba of Lagos. The incidence of cholera among children in newly-infected areas is typically low. In Lagos, the under ten years cohort had only 9.2% of the cases, although they probably account for one quarter to one-third of the total population.

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[12] W.H. Mosley, "Epidemiology of cholera", in Principles and Practice of Cholera Control. Geneva, WHO, 1970, p.25.

TABLE 6

Age-sex distribution of the first 922 cholera cases in Lagos State

Age cohort	Male	Female	Total	Percentage of cases
0-10	56	29	85	9.2
11-20	75	49	124	13.4
21-30	115	139	254	27.3
31-40	87	103	190	20.5
41-50	69	57	126	13.6
51-60	31	37	68	7.4
61-70	17	22	39	4.3
71 & over	6	12	18	2.1
Unknown	10	8	18	2.1
Total	466	456	922	99.9

Source: Dada, op.cit., 1971B, p.138.

Ibadan, Nigeria

Ibadan experienced an explosive epidemic following the introduction of cholera from Lagos on January 3, 1971. The epidemic peaked between its sixth and ninth week. There were over 3,300 cases in this four week period.[13] Treatment facilities were completely overwhelmed.

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[13] J.D. Adeniyi, "Cholera control: problems of beliefs and attitudes", Int. J. Hlth. Education, vol.15, No.4 (1972), p.238.

St. Mary's Hospital, with a total of only eighty beds treated 4,303 cholera victims in eight weeks and admitted up to 256 new patients in a single day.[14] The infection persisted at a lower level for over a year. Infected wells were implicated in the Ibadan cholera epidemic.[15]

A study by Adeniyi[16] showed that poor sanitation and the beliefs and attitudes of the population contributed to the massiveness of the epidemic. An estimated 10% of the population defecates in the streets, streams, and drains, while many people continue to rely on these streams for their water supply. Adeniyi's survey of 250 randomly selected people revealled widespread misinformation about the epidemic. For example, 70% thought that cholera was indigenous to Nigeria, confusing it with a disease known in Yoruba as origbameji (literally two calabashes, i.e. one for vomiting and one for diarrhoea). Because they had successfully treated origbameji at home, people were inclined to use the same traditional treatment for cholera and go to the hospital only when it was too late. Over 18% of Adeniyi's sample believed that cholera was caused by angry Gods or the Yoruba masquerade Olulo. Native medicine was preferred to Western medicine by 14.8%, and 15.2% said they did not believe in vaccination. Many of those preferring native medicine and rejecting vaccination were among the group who attributed cholera to angry Gods or Olulo.

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[14] E.A. Lewis et al., "Cholera in Ibadan", Amer. J. Trop. Med. Hygiene, vol.21 (1972), p.309.

[15] U.L.C.M., op.cit., p.144.

[16] Adeniyi, op.cit., pp.238-245.

A significant implication of the above findings is the difficulty of successfully implementing control measures when a segment of the population is unwilling to participate or cooperate because of their beliefs. The uncooperative group may easily become a persistent reservoir of infectivity.

Zaria, Nigeria

The distribution of cholera in the city of Zaria in 1971 has been studied and mapped by Schram.[17] Most cases were in the Old City from the area surrounding the market. Three-quarters of the victims obtained their water supply wholly or partially from untreated wells or the river. The occupational division of a sample of 200 cholera patients included twenty-one Koranic pupils, twenty-one traders, fifteen tailors, nine foodsellers and butchers, three Koranic school teachers, one water seller and one beggar, but only three students and nineteen farmers and herdsmen.[18]

The concentration of market-associated occupations, as well as the spatial location of cases implicates the market as a source of cholera dissemination. The relatively large numbers of Koranic pupils and foodsellers infected suggests that food may have been an important vehicle of transmission. Koranic students in Northern Nigeria who sustain themselves by begging habitually congregate around the market to get left-over food from foodsellers.

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[17] R. Schram, "The 1971 cholera epidemic in Zaria, Nigeria", Savanna, vol.1,No.2 (1972), pp.213-222.

[18] Schram, op.cit., p.221.

Goudoumé, Ivory Coast

Goudoumé is a lagoon village located a few miles east of Abidjan. It was the site of a bacteriological search for carriers in October, 1970. Of the 506 cultures obtained, 101 were positive, despite the fact that there were only four clinical cases during the entire outbreak.[19] Virtually every resident of Goudoumé must have been a carrier at some point. Examination of wells and the lagoon edge in infected lagoon villages usually showed the presence of vibrio contamination.[20]

The Goudoumé example is probably typical of cholera outbreaks along the West African coast. This was an example of the classical cholera transmission model, with massive contamination of the environment, particularly water supplies. The epidemics in such places tended to be protracted but unspectacular. Among the factors which account for the high risk of establishing a carrier-environment transmission cycle are the location of villages along lagoons or delta channels, the predominance of fishing as an occupation, the use of untreated water from lagoons, rivers and shallow wells, and the suitability of the brackish water found along the coast for vibrio survival.

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19 J. Le Viguelloux and J.C. Doury, "Épidémiologie du choléra moderne", Médecine Tropicale, vol.31, No.1 (1971), p.23.

20 A. Bourgeade, J. Rive et al., "L'épidémiologie du choléra et ses problèmes", Médecine d'Afrique Noire, vol.20, No.3 (1973), pp.177-187.

Louga, Senegal

The holding of gatherings such as markets, funerals, weddings and festivals has been noted in previous chapters as a key element in the inter-regional and long distance diffusion of cholera. Gatherings were equally important for local dissemination of the infection. For example, cholera was introduced to Thiamène, near Louga, Senegal by traders from the Senegal Valley coming to the periodic market.[21] Cholera spread radially from Thiamène to seventeen villages up to fifteen kilometers from Thiamène within five days. Most of the villages were infected by persons who had attended the market, although the actual means of infection, whether person to person or through contact with an infected medium, is unknown.

In the Podor-Matam region of the Senegal Valley local diffusion followed a wedding.[22] A second phase of the diffusion occurred as a result of the gathering of mourners for the funerals of first generation victims.

Akodesséwa-Plage, Togo

Following the introduction of cholera in late November, 1970 by Ghanaian fishermens to Akodessewa-Plage, a fishing village twelve

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21 A. Sy et al., "L'épidémie de choléra dans le département de Louga (région de Diourbel)", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), p.663.

22 A. Carvhallo et al., "L'épidémie de choléra à Podor et Matam", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), pp.655-661.

kilometers from Lomé, stringent measures were taken to control the epidemic.[23] Quarantines, mass vaccination, establishment of treatment centers, prohibition of fishing and batheing in the lagoon or sea, and disinfection of vehicles used to transport cholera victims are some of the measures which were used in an attempt to stop the epidemic.

The containment efforts failed. Despite the deployment of troops to enforce the quarantine, the nightly movement of fishermen into the restricted zones from infected areas in Ghana continued.[24] They also failed to prevent the escape of villagers through the cordon. Fugatives from Akodesséwa-Plage carried cholera to Zolwa which served as a point of diffusion to several other villages. The intense social interaction of the people and their failure to heed warnings against travel, gatherings and ritual treatment of bodies before burial were responsible for the survival and diffusion of cholera in coastal Togo.[25]

The establishment of cholera in Coastal Togo shows the difficulty of implementing control measures to contain the diffusion of the disease. The success of control measures may be affected by the attitudes of the people toward the disease and government-initiated controls. The controls are likely to fail if the understanding and cooperation of the population is obtained, and unless the controls are designed to only minimally disrupt normal activities.

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[23] The control measures are described by E. Amégée et al., "Mésures d'hygiène du milieu au cours de l'épidémie de choléra au Togo", Médecine d'Afrique Noire, vol.20, Nos.8/9 (1973), pp.649-654.

[24] J.J. D'Almeida et al., "L'epidémie de choléra au Togo", Médecine d'Afrique Noire, vol.20, Nos.8/9 (1973), p.642.

[25] D'Almeida, op.cit., p.643.

Goulfey, Northern Cameroun

The cholera epidemic at Goulfey is an example of apparent person to person spread. The outbreak began the day after a large circumcision ceremony attended by 15,000 to 20,000 people from Cameroun, Chad and Nigeria.[26] Between May 7 and May 28, 1971 there were 801 cases and 121 deaths among the 3,500 residents of Goulfey. All wells in the town were chlorinated and all latrines disinfected four or five times daily.

Among the 700 boys who had been circumcized and whose customary post-circumcision isolation coincided with the epidemic, there was not a single case.[27] The absence of infection among the isolated children and the treatment of water sources would seem to rule out spread by contaminated drinking water or food. Person to person spread seems to be indicated. It probably resulted from burials and caring for cholera patients at home.

The attack rate in Goulfey was higher among women than among men.[28] This is somewhat surprising because of the usual confinement of Muslin women and the more active role of men in the conducting of burial ceremonies. However, women might play a more active role than men in the care of the sick at home. This may account for the higher incidence among women.

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[26] B. Coulanges and P. Coulanges, "A propos de l'épidémie de choléra du Sultanat de Goulfey (Nord Cameroun: Mai-Juin, 1971). Considérations sur l'épidémiologie et la proplylaxe", Bull. Soc. Pathol. Exotique, vol.65, No.2 (1972), p.216.

[27] Coulanges and Coulanges, op.cit., pp.222-223.

[28] Coulanges and Coulanges, op.cit., p.221.

Another puzzling aspect of the Goulfey outbreak is the very small number of cases among residents of nearby villages and among guests at the circumcision ceremony. While the infection was imported at the time of the festival, it was only after the festival that widespread dissemination occurred.

Generalizations

The local dissemination of cholera has been shown to be very much a function of local conditions. The following generalizations summarize aspects of the local dissemination of cholera in the seven locations considered in the chapter.

1) Distinct patterns of cholera diffusion may be found in local areas, as well as in inter-regional diffusion. However, the complexity of urban environments and the considerable potential for spread through person to person contact often complicates the analysis of these local diffusion patterns.

2) Markets, funerals and festivals were important factors in local, as well as inter-regional diffusion.

3) The means of dissemination varied from place to place. Infected water supplies were implicated in Goudoumé and Ibadan. Person to person spread apparently occurred in Goulfey. Contaminated food sold by market vendors is the suspected mode of transmission in Zaria.

4) Inadequate sanitation and absence of protected water supplies generally were characteristic of the infected areas.

5) Control measures must be understood and approved by the controlled population if they are to be successful. Control measures should disrupt normal activities as little as possible. Successful implementation of control programs is especially difficult where a part of the population is uncooperative because of their belief systems.

6) The highest incidence of cholera was among young adults. Children had a relatively low attack rate.

CHAPTER 7

SYSTEMS MODELS OF CHOLERA DIFFUSION

The previous chapters dealing with the diffusion of cholera at different scales of analysis from international to intra-urban contain considerable evidence of the complexity of the cholera transmission cycle and diffusion process. Cholera vibrios are adaptable to considerably different environmental conditions and may be spread in a variety of ways. Because of this chameleon-like adaptability, it is generally difficult to discover the basis of survival and transmission during a particular outbreak. A considerable number of detailed studies have been undertaken in recent years in Asian countries, especially India and Bangladesh, which have attempted to gain a better understanding of cholera epidemiology. The investigations seem to have achieved very few significant break-throughs.

As even intensive local studies have generally failed to produce satisfactory explanation of cholera survival and diffusion processes in a particular epidemic or under particular environmental conditions, any attempted general model of the interrelationships of factors governing cholera survival and diffusion is bound to be inadequate, especially when it is based only on secondary sources. Nevertheless, this chapter will focus on the synthesis of factors relevant to the survival and diffusion of cholera El Tor, with particular reference to the experience with cholera in West Africa.

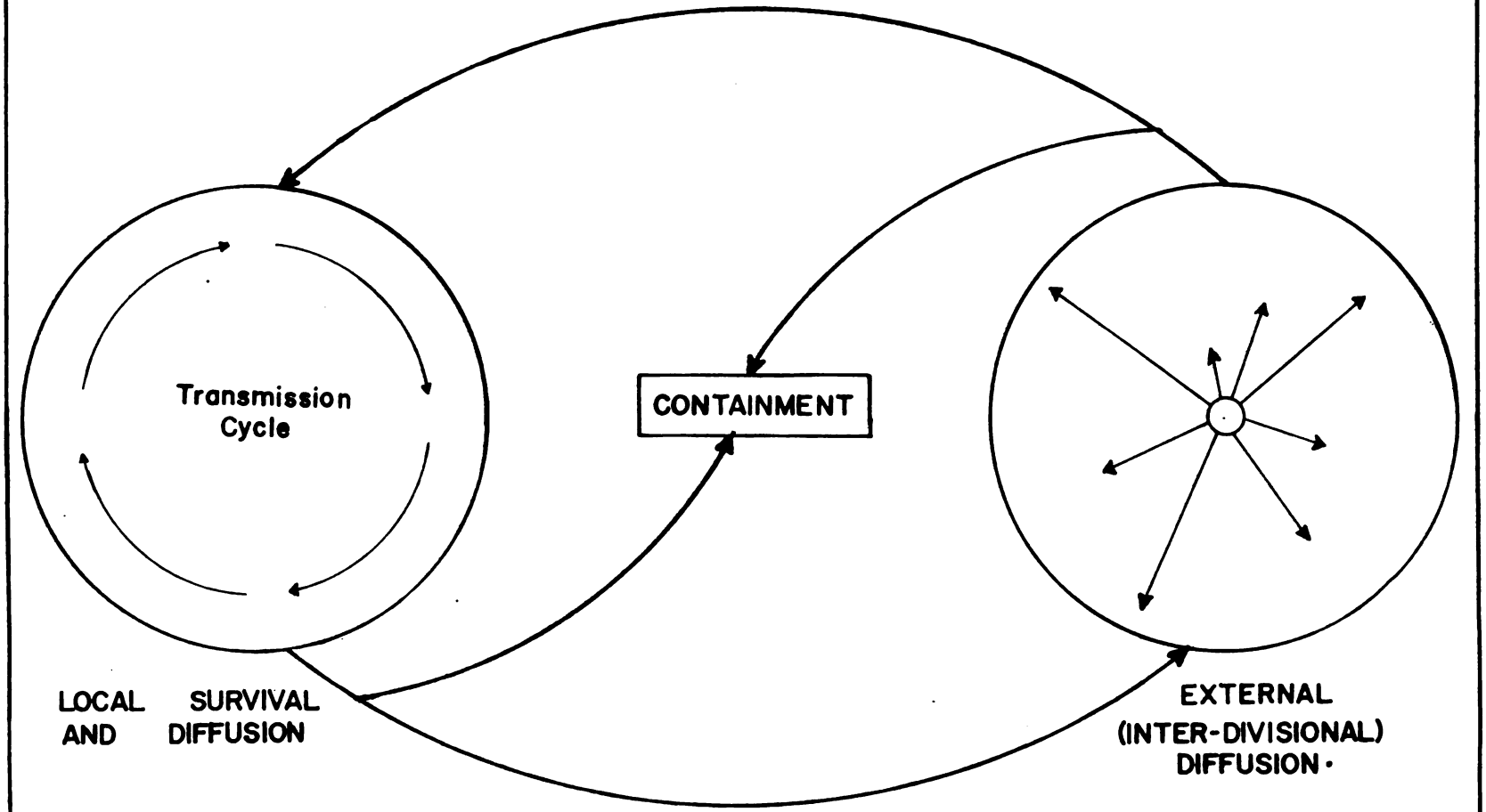
FIGURE 29

LOCAL AND EXTERNAL CHOLERA DIFFUSION

A model of cholera diffusion must include two essential components, namely, local and external diffusion. Figures 30 and 32 show the components of the local and external diffusion subsystems.

FIGURE 29

LOCAL AND EXTERNAL CHOLERA DIFFUSION



Systems modelling techniques will be employed as a method of organizing and classifying the relevant factors and focusing on the interrelations between factors.

There are essentially two facets which must be considered in the diffusion of cholera after its introduction to a group of population clusters or nucleations. One part, which will be referred to as **local** diffusion refers to the survival and diffusion of the infection within a subsection of the territory in question. The subsection could be a small region, a city or a community within a city, depending on the scale of analysis employed. The second aspect of cholera diffusion, external **diffusion**, concerns the spread of the infection from one subsection to another, for example, between two regions or two cities. The possibility of external diffusion is dependent on local survival and diffusion. It is through the establishment of a transmission cycle in a community that the volume of vibrio contamination and number of carriers increases. The probability of a vibrio-positive traveller transferring the infection to a different location varies directly with the amount of vibrio contamination and number of carriers at the travellers' places of origin. External diffusion is the most important way in which the infection comes into contact with new susceptible populations which enables it to survive. In a closed community, the size and duration of an epidemic would be limited by the steady reduction of the cholera-susceptible population.

While the two basic components of the cholera diffusion system are inter-dependent, and although both are diffusion processes,

there are considerable differences in the relevant factors governing external and local diffusion systems. Factors common to both types of diffusion may operate differently at a local level than at an inter-regional level.

External Diffusion

When considering the diffusion of cholera from one city or region to another, we are primarily concerned with the behavior of the subset of interregional travellers who are infected with vibrio cholerae, whether asymptomatic carriers, cholera cases or the bodies of cholera cases being transported for burial. The type of travel, such as labor migration, social visits, nomadic movements or commercial trips will affect the speed, distance and destination of the journey. A population of secondary interest is the subset of transported goods which are cholera-infected. Examples are vibrio-contaminated food, water or goods in the possession of travellers, or food items involved in interregional trade. North African shellfish, for example, have been implicated in the transfer of cholera from North Africa to Italy.[1] Leather water pouches widely used to draw water in West African Savanna may also have facilitated the interregional diffusion of cholera in some cases.

The survival of the vibrio in the travelling carrier until after he reaches his destination is essential if diffusion is to occur.

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[1] Weekly Epidemiological Record, vol.48 (1973), p.358.

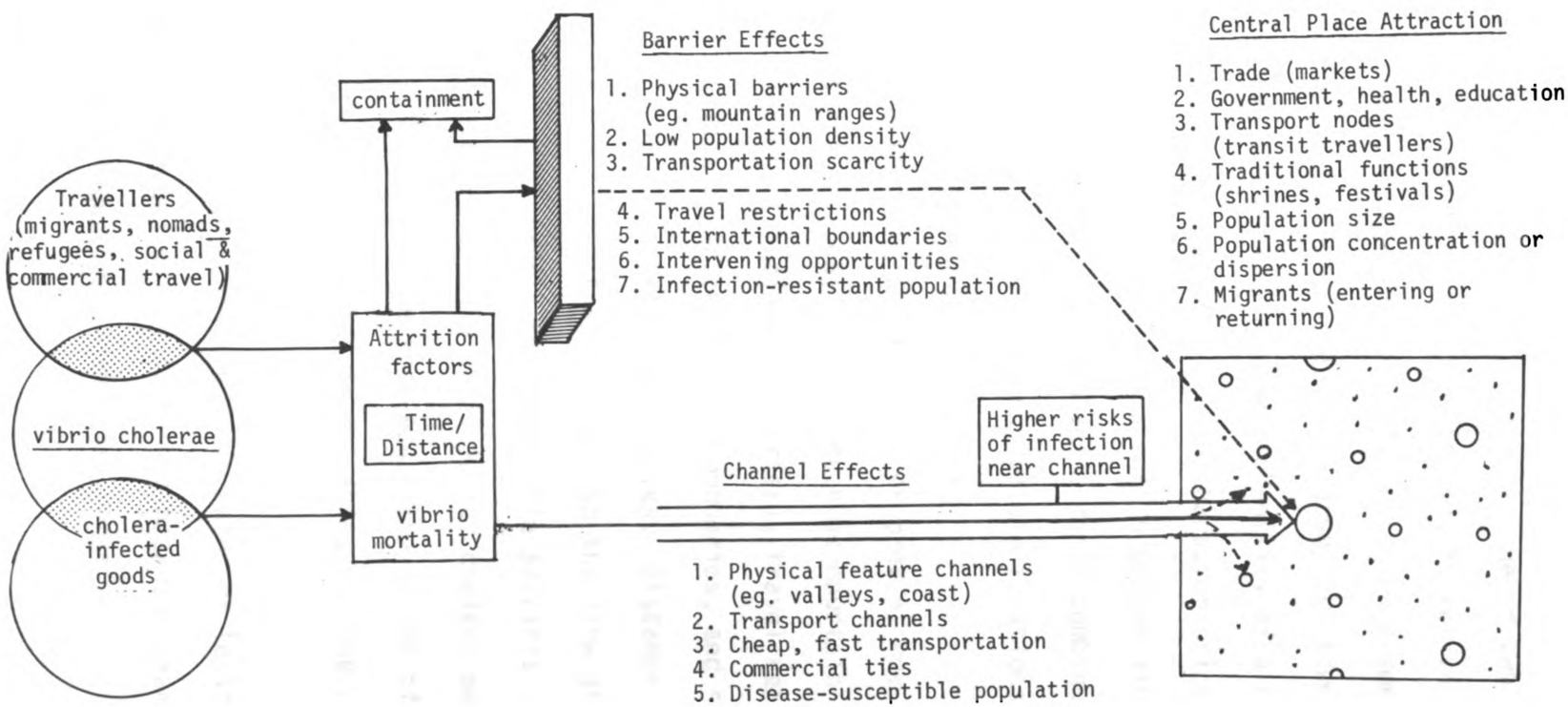
FIGURE 30

EXTERNAL (INTER-DIVISIONAL) CHOLERA
DIFFUSION MODEL

Cholera is spread from one division to another by cholera-infected travellers or goods. Vibrio attrition occurs during the journey, being a function of the rate of vibrio mortality and the time and distance of the journey. Containment may result if these factors are unfavorable. Barrier, channel and central place effects combine to greatly increase the risk of infection of some communities, while reducing the probability of cholera introduction in others.

FIGURE 30

EXTERNAL (INTER-DIVISIONAL) CHOLERA DIFFUSION MODEL



The length of carrier status is short, generally one or two days. Thus, the attrition of the vibrio is a major limiting factor in the spread of cholera, especially with long distance diffusion. Key elements affecting the attrition of vibrios in the course of a journey are time and distance. The longer the journey the greater is the probability of a carrier arriving vibrio-free at the destination. Vibrio characteristics, especially their rate of mortality in the carrier state also govern vibrio attrition.

Barrier, channel and central place effects combine to focus the probability of disease introduction on certain locations, while reducing the probability of infection in others.

Barriers are features which impede or prevent movement. Examples of barriers are physical barriers such as mountain ranges or gorges, immune populations (vaccination barrier), travel restrictions created by travel cordons or international boundaries, and scarcity of transportation routes or transportation vehicles. Distance and intervening opportunities may also act as barriers to the flow of traffic between two locations. The effect of impermeable barriers is to prevent movement and as a result prevent the diffusion of cholera beyond the barrier. Permeable barriers impede and reduce the volume of traffic. Diffusion through a permeable barrier is possible, but the probability is relatively low.

Channels are features which provide paths of least resistance facilitating movement between points along and near the channel. Movement may be facilitated by a reduction in the time, cost or difficulty of the

journey. Channels may be physical features, such as valleys. Transportation routes are man-made channels of particular importance for diffusion. The spatial distribution of the disease-susceptible population also affects the spread of the disease. Places located along physical or man-made channels have a relatively high risk of infection because of the possibility of contamination by persons passing through toward another center further along the channel.

Population nucleations have unequal attraction for travellers. The more important a place, the more traffic it is likely to attract. Among the functions which combine to constitute the central place attraction of cities for travellers are the volume of trade, markets, location of government, health and educational facilities, and traditional functions such as the staging of traditional festivals. Important transportation nodes, such as junction points, railway centers or airports attract transit travellers. Population size and employment opportunities for migrants are other aspects of central place attraction.

Barrier, channel and central place factors are closely interrelated. For example, the Sahara Desert and the Middle Belt in West Africa constituted barriers to cholera diffusion. However, the barrier effect is primarily a result of the lack of central places with substantial attraction in the Sahara Desert and Middle Belt, and the relative scarcity of transport channels.

Three types of barriers, namely, physical barriers, vaccination barriers and travel restrictions affected the diffusion of cholera in

FIGURE 31

MARKET SCENE IN MOKWA (NIGERIA)

The role of markets as foci of cholera diffusion in West Africa is well established. For example, markets were implicated in intra-urban diffusion in Zaria (Nigeria), local rural diffusion at Thiomène (Senegal) and both long distance and regional diffusion at Mopti (Mali). The meeting of cholera carriers and cholera susceptibles from a wide area in the crowded and often unhygienic conditions of the market facilities cholera dissemination.

West Africa. The Atlantic Ocean provided an impermeable physical barrier along the West African Coast, and the Sahara Desert a virtually impermeable barrier to the North. Areas of low population density in the Middle Belt, Southern Cameroun and Central Chad also provided barriers to the infection. Vaccination barriers were used in the vicinity of Bamako[2] and Fort Lamy[3] to protect the urban areas from cholera. Although both cities experienced outbreaks, it appears that the vaccination program was instrumental in reducing the size of the outbreaks and preventing these cities from becoming major diffusion foci. Travel cordons were used in Togo,[4] Mali[5] and Northern Cameroun[6] in trying to contain the infection. These cordons formed permeable barriers which reduced travel but did not prevent further dissemination of the disease. International boundaries were ineffective as cholera barriers in West Africa. Even if legal crossings are controlled, the uncontrollable movements of smugglers and migrating herdsmen and fishermen continue. Smugglers have been implicated in the introduction of cholera in Makary (Northern

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[2] J. Voelckel and G. Causse, "Aperçus prophylactiques", Médecine Tropicale, vol.31, No.6 (1971), p.713.

[3] O. Bono et al., "Installation du choléra aux alentours du Lac Tchad", Bull. Soc. Pathol. Exotique, vol.64, No.4 (1971A), p.389.

[4] J.J. D'Almeida et al., "L'épidémie de choléra au Togo", Médecine d'Afrique Noire, vol.20, Nos.8-9 (1973), p.640.

[5] G. Causse, J. Le Viguelloux and J. Voelckel, "Remarques sur l'orginization practique de la lutte contre le choléra en zone rurale dépouvre de médecin", Bull. Soc. Pathol. Exotique, vol.64, No.5 (1971), p.662.

[6] Causse, Le Viguelloux and Voelckel, op.cit., p.662.

Cameroun)[7] and Tunisia.[8] Nomads were responsible for spreading cholera in East Africa[9] and Mauritania.[10] The role of migratory fishermen in the coastal diffusion phase has been well documented.

Physical and man-made channels were influential in the diffusion of cholera in West Africa. The valleys of the middle Niger and Senegal Rivers were the most prominent physical channels. The coastal fringe, which includes the lagoon networks and inshore areas, also served as a channel for the movement of fishermen in canoes. In addition to providing physical channels facilitating movement, the river valleys and coastal areas tend to have relatively high population densities. This linear concentration of susceptibles also promotes a linear pattern of diffusion. Man-made transportation channels directed the patterns of diffusion in Angola and Moçambique, where diffusion axes tended to follow railway lines, and in Nigeria, where the amount of transportation development was found to be significantly correlated with the spread of cholera.

The regularity with which large cities were infected with cholera in various parts of Africa attests to the importance of urban centrality. The impact of central place attraction was most prominent in Nigeria, where a composite index of urban importance was the most

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7 B. Coulanges and P. Coulanges, "L'épidémie de choléra du sultanat de Goulfey (Nord Cameroun: Mai-Juin, 1971)", Médecine Tropicale, vol.31, No.6 (1971), p.637.

8 H. Félix, "Le choléra Africain", Médecine Tropicale, vol.31, No.6 (1971D), p.620.

9 B. Carteron and J.C. Artus, "Épidémiologie du choléra au Territoire Français des Afars et des Issas", Médecine Tropicale, vol.33, No.3 (1973), pp.235-248.

10 "Cholera and politics", West Africa, No.2881 (Sept.8, 1972), p.1175.

important of six variables correlated with the time of cholera onset. Urban importance was not uniformly important in controlling diffusion patterns. The correlation between urban importance and week of onset in Mali was not statistically significant. In Kenya, cholera penetrated relatively close to such cities as Nairobi and Kisumu, but remained confined to rural areas.

Particular central place functions were instrumental in the diffusion of cholera in various areas. Markets were diffusion foci in several areas, including Mopti, Mali[11] and Thiomène, Senegal.[12] The attraction of thousands of people to a traditional ceremony in the emirate capital of Goulfey[13] brought about the initial cholera outbreak in the vicinity of Lake Chad.

The movement of seasonal labor migrants may also be considered an example of central place attraction because migrants' places of origin and destination are spatially concentrated. Seasonal labor movements have been linked to cholera diffusion in Bangladesh.[14] While there is no conclusive evidence that labor migrants spread cholera in West Africa,

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[11] H. Félix, "Le développement de l'épidémie de choléra en Afrique de l'Ouest", Bull. Soc. Pathol. Exotique, vol.64, No.4(1971C), p.566.

[12] A. Sy et al., "L'épidémie de choléra dans le département de Louga (région de Diourbel)", Bull. Soc. Méd. Afrique Noire Langue Française, vol.17, No.4 (1972), p.663.

[13] Coulanges and Coulanges, op.cit., p.636.

[14] W.M. McCormack, W.H. Mosley et al., "Endemic cholera in rural East Pakistan", Amer. J. Epidemiology, vol.89, No.4 (1969), p.402.

their role must be suspected because of their large numbers and generally poor living conditions. It is suspected that the increased volume of migration associated with the West African drought is partially responsible for the continuing diffusion of cholera in the West African Sahel during 1973 and 1974.

Local Diffusion

The diffusion of vibrio cholerae in a community after its initial introduction may involve the complex interaction of a number of variables. Among the more important of these factors are the characteristics of the vibrio, the suitability of the environment for vibrio survival, the size and location of the susceptible population and use of control strategies. Under ideal conditions, a transmission cycle will be established and the disease will continue to spread until the equilibrium is disturbed through a change in the predisposing environmental conditions or a decline of the susceptible population. The infection will be contained if prevailing conditions are not conducive to its spread.

Figure 32 shows a model of the local transmission of cholera. The various subsections of the model will be described below, together with a number of African examples.

Initial survival

The survival or containment of cholera vibrios after their introduction to a community depends on vibrio characteristics, the form

FIGURE 32

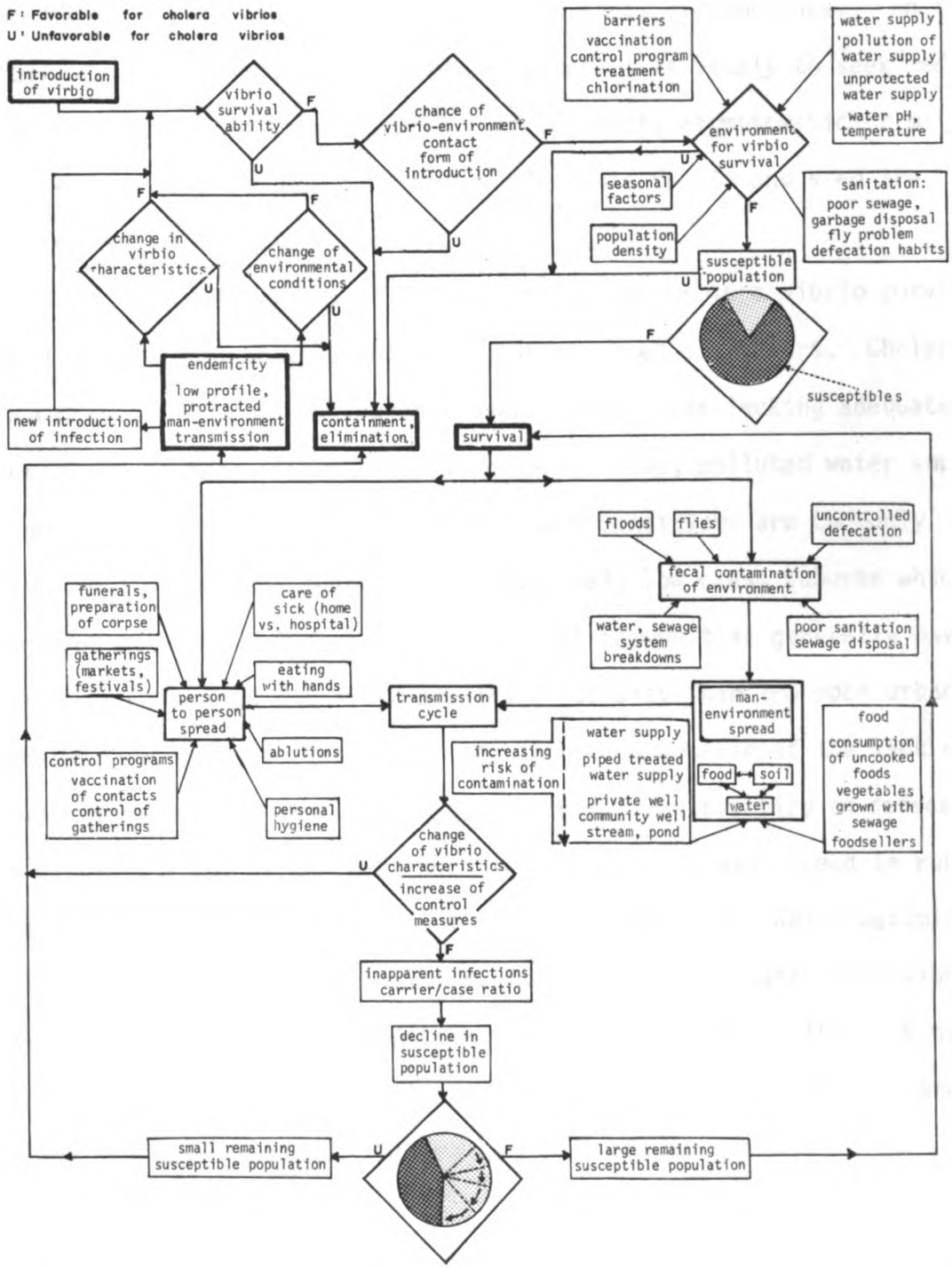
LOCAL SURVIVAL AND DIFFUSION OF CHOLERA

This model shows interrelationships between factors governing the local survival and diffusion of cholera from its initial introduction to its elimination. The diamond-shaped boxes show major factors governing cholera survival and diffusion. These may be favorable or unfavorable for the survival of the vibrio. For example, a large susceptible population and ideal environmental conditions would be favorable, while a very small susceptible population and a suitable environment would be unfavorable.

Unfavorable conditions, as a rule, lead to the containment of the infection. As the epidemic proceeds, the susceptible population decreases, eventually ending the epidemic phase.

FIGURE 32

LOCAL SURVIVAL AND DIFFUSION OF CHOLERA



of introduction, and environmental suitability. The hardiness of the strain of the introduced vibrios influences the duration of their survival and their ability to survive in less than ideal circumstances. An index cholera carrier who develops clinical symptoms is likely to seek medical aid and be confined to a hospital. In contrast, asymptomatic or mildly symptomatic carriers may continue to circulate freely and seed the environment with cholera vibrios.

The suitability of an environment for cholera vibrio survival involves the interrelationship of a large number of factors. Cholera is a disease of poor sanitation. Overcrowded areas lacking adequate sewage and garbage disposal and with unprotected, polluted water supplies experience the highest attack rates. Such conditions are commonly found in the recently constructed, unplanned, low class suburbs which surround many large African cities. Rural communities generally have smaller populations and lower population density than the poor urban suburbs, but are also susceptible to infection because of the lack of sewage disposal and protected water systems. The practice of defecating in gutters, along stream beds and "in the bush" is widespread in rural and low-class urban areas in Africa. Such defecation habits maximize the possibility of the pollution of food or drinking water with vibrios. The use of flush toilets or pit latrines greatly reduces the risk of virbio survival. In upper class, low density neighborhoods with protected sewage and water systems, cholera is not a serious threat.

FIGURE 33

URBAN SLUM HOUSING IN CAPE COAST (GHANA)

This photograph illustrates some typical conditions in the West African urban slum areas which were frequently heavily infected with cholera. These conditions include poor quality housing, crowding, and a probable lack of piped water and sewage systems. The open gutter between the street and houses may provide an ideal survival environment for cholera vibrios if it is used for defecation or the disposal of household refuse.

Various control measures may be undertaken to make the environment less suitable for cholera survival. Construction of pit latrines, chlorination of water supplies, mass vaccination programs, environmental clean-up campaigns and ongoing surveillance to detect and isolate an introduction of cholera vibrios at an early stage are examples of control programs which increase the likelihood of vibrio containment.

Where cholera has been established for a long time, such as India or Bangladesh, it often occurs seasonally. Cholera introduced during the off-season is likely to encounter unfavorable environmental conditions and be contained. Conditions during the cholera season would tend to favor the survival of the infection. It was widely believed at one time that seasonal cholera epidemics were directly related to temperature or humidity.[15] Cholera seasonality is probably related to climate indirectly as a result of such factors as an increase in travel in the dry season or a rise of water levels in ponds, wells, and rivers during the rainy season.

Susceptible population

The potential development of an outbreak of cholera is limited by the size of the cholera-susceptible population. Figure 34 shows that the size of the susceptible population in a community is influenced by a variety of factors. Susceptibility to cholera is relative, rather than

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[15] A.M. Kamal, "Endemicity and epidemicity of cholera", Bull. WHO, vol.28 (1963), p.284.

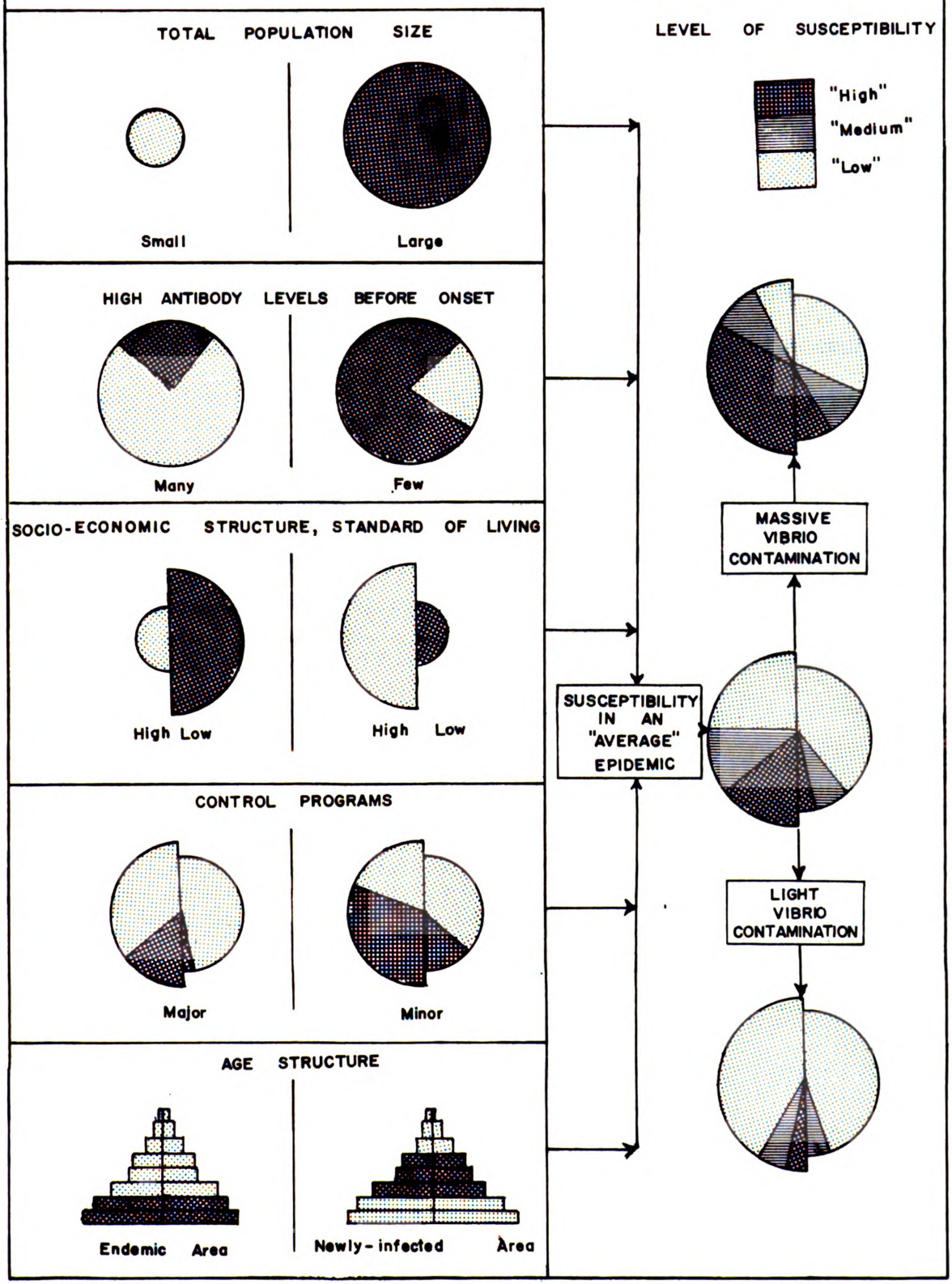
FIGURE 34

SIZE OF CHOLERA-SUSCEPTIBLE POPULATION

The size of the susceptible population is important because it essentially defines the potential size of an epidemic. Such factors as population size, number who are initially cholera resistant, control measures, age structure and the socio-economic structure of the community affect the size of the susceptible population. However, susceptibility cannot be determined in isolation from the extent of vibrio pollution and virulence of the vibrio strains. Where there is exceptionally high vibrio pollution or a particular virulent strain, a considerably larger number will be susceptible than in an "average" epidemic.

FIGURE 34

SIZE OF THE CHOLERA-SUSCEPTIBLE POPULATION



absolute. Given a sufficiently large ingestion of vibrios, no person is immune. In a normal population, the level of susceptibility of the members varies from very low to very high. This susceptibility is a function of such factors as vaccination, recovery from a cholera infection, socio-economic status and age.

Total population size is a basic variable in establishing the limits of the susceptible population. In a village of 1,000 persons, the limit of the local susceptible population is 1,000. The course of an epidemic is likely to be much shorter than in a millionaire city such as Lagos or Ibadan. Within the total community, part of the population may have a degree of immunity as a result of prior exposure to the disease or recent vaccination. The proportion of initial susceptibles in the total population will be less in endemic than in newly-infected areas.

Because cholera is associated with poor living conditions, knowledge of the socio-economic structure is necessary for estimating the population's susceptibility.

Control measures, such as mass vaccination and clean-up campaigns reduce the size of the highly susceptible population. However, vaccination provides only partial protection for less than six months, so even the vaccination of the entire population would not eliminate the threat of cholera. The impact of control measures can be maximized by concentrating on low socio-economic groups who run the greatest risk of infection because of their poor living conditions.

Susceptibility is also a function of age. In endemic areas such as the Indian subcontinent, cholera mainly affects

children[16]. Adults, who generally have acquired high antibody levels as a result of prior exposure to the disease, are less affected. In newly-infected areas, young to middle-aged adult males are infected first and most seriously. Adult males tend to be the most mobile section of the population and therefore have the greatest risk of exposure to infection. There is no change in antibody levels with age in previously uninfected countries.[17]

The various factors displayed in Figure 34 govern the size of the initial highly susceptible population in a community. With the progression of the epidemic the susceptible population declines in size relative to the cholera-resistant population. This results from the increase in cholera antibodies among individuals who have recovered from clinical or sub-clinical cholera infections.

Consideration of the size of the susceptible population is incomplete if the extent of vibrio contamination during an epidemic is not considered. Where there is only light contamination of the environment, only the highly susceptible segment of the population is in danger of being infected. Where there is massive contamination of the environment, virtually the entire population may be considered highly susceptible. The massiveness of vibrio contamination accounts for the

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[16] McCormack, Mosley et al., op.cit., p.403.

[17] W.H. Mosley, A.S. Benenson and R. Barui, "A serological survey for cholera antibodies in rural East Pakistan", Bull. WHO, vol.38 (1968), pp.327-334.

heavy morbidity and mortality in the West African Sahel cholera outbreaks compared to other areas with similar initial susceptibilities.

Transmission cycle

If cholera is introduced to a new community containing a cholera-susceptible population and with conditions favoring its survival in the environment, an ongoing transmission cycle may be established. The conventional transmission cycle involves fecal contamination of the environment and subsequent ingestion of some contaminated element from the environment by a cholera-susceptible person. The cycle is completed by the multiplication of the vibrios in his gut and the return of the vibrios to the external environment in feces. In addition to dissemination through this type of man-environment transmission cycle, cholera may be transferred directly from a carrier's body to the body of a cholera-susceptible person with elements of the environment such as water, food or flies playing no intermediate role. This may be called person to person spread.

Many observers, particularly those from Francophone Africa have emphasized the dominance of person to person spread in the West African cholera epidemic. For example, Félix has claimed that diffusion in Savanna West Africa was exclusively person to person spread.[18] Although person to person spread may have been predominant in certain areas, other examples may be cited from many parts of West Africa which

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[18] Félix, op.cit., 1971C, pp.573-575.

implicate infected water supplies in the dissemination of cholera. Therefore, one type of transmission should not be over-emphasized at the expense of the other. It is probable that man-environment and person to person spread frequently occurred concurrently in a particular outbreak.

Person to person dissemination of cholera was frequently found to be associated with funerals and the care of cholera patients in West Africa. The ritual preparation of a corpse for burial and the collection and disposal of the copious amounts of watery excrement passed by a cholera victim are particularly dangerous activities during a cholera epidemic. Contacts who are unaware of the way in which cholera spreads are unlikely to take the necessary precautions to avoid ingesting vibrios when in close contact with a cholera victim and the victim's environmental surroundings. Person to person spread may also occur in crowds of people such as those found at a market or festival.

Personal hygiene and cultural factors influence the probability of ingesting vibrios. Persons washing infrequently and those who eat with their hands would have a relatively high risk of ingesting vibrios adhering to the hands or another part of the body. It is also possible that the Mushim ablutions performed before all prayers, which involve the washing of various parts of the body such as hands, feet, anus, face and the inside of the mouth could facilitate the transfer of vibrios from the outer surface of the body to the inside of the mouth.

Control programs may be instrumental in reducing the risk of person to person spread. The restriction of gatherings and provision of adequate treatment facilities so patients need not be treated by members of their family at home are examples of control programs which may significantly impair person to person spread.

Man-environment transmission occurs as a consequence of fecal contamination of the environment. This may result from the lack of sewage collection and disposal systems, direct environmental pollution by uncontrolled defecation, floods, or temporary breakdowns of the sewage system. One or more of food, water, soil, or cholera-infected goods may form the environmental link in the transmission cycle. Unprotected domestic water sources, such as streams, lagoons, or community wells are particularly susceptible to contamination. This contamination may occur as a result of direct defecation into the water source disposal of sewage and garbage, and use of an infected container to draw water. In West Africa, areas along lagoons and river flood plains have tended to have persistent, water-borne epidemics.

Food may be contaminated by flies which had come into contact with a cholera-infected medium. The transmission cycle in the Israeli epidemic of 1970 involved consumption of vegetables grown with untreated sewage containing cholera vibrios.[19] Sewage is similarly used for

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[19] J. Cohen et al., "Epidemiological aspects of Cholera El Tor in a non-endemic area", Lancet, July 10, 1971, pp.86-89.

FIGURE 35

A VILLAGE WELL, DOGON DOUTCHE (NIGER)

One or a small number of community wells typically form the water supply for villages in the dry savanna. Because of the crowds of people who gather to draw water and herds of animals brought to the wells the area around wells is frequently unsanitary. Leather devices used to draw water could support the survival of cholera vibrios and be responsible for the transmission of infection from one well to another. Therefore, it is possible for water to play a significant role in cholera diffusion, even in the dry environment of the Sahel Savanna.

growing vegetables in West Africa in the vicinity of Kano, Zaria and other large cities.

The numerous foodsellers found in Africa in markets and along roadsides are an important diffusion hazard. There is little or no supervision of the preparation of this food. Much of it is prepared unhygienically and sold in market places where the risk of having the food contaminated by flies or vibrio-infected people is at a maximum. In Schram's study of cholera in Zaria, the infection was found to be clustered around the market.[20] Relatively large numbers of foodsellers other types of merchants and koranic students who are largely dependent on food vendors' handouts for their subsistence developed cholera in Zaria. This evidence tends to implicate foodsellers as a focus of the dissemination.

Decline of an Epidemic

Cholera epidemics in a community contain the seeds of their own destruction. This results from the growth in the size of the cholera-resistant population and coinciding reduction of the susceptible population. Decreased susceptibility results from the development of vibriocidal antibodies as a consequence of recovery from vibrio infection or from vaccination. Often, the end of an epidemics occurs after only a small proportion of the population has developed clinical symptoms

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[20] R. Schram, "The 1971 cholera epidemic in Zaria, Nigeria", Savanna, vol.1, No.2 (1972), p.216.

FIGURE 36

ROADSIDE FOODSELLERS, PO (UPPER VOLTA)

Markets, transport stops and even residential areas in African towns are frequented by large numbers of mobile or semi-mobile foodsellers. The preparation and sale of this food is generally unregulated. Foodsellers seem to have played an important role in the dissemination of cholera in Zaria (Nigeria). It is probable that they were responsible for spreading cholera in other parts of West Africa as well.

FIGURE 37

FISHERMEN'S CANOES IN FIVE COWRIE CREEK,
LAGOS (NIGERIA)

This photograph illustrates conditions which favored the establishment and long-term survival of cholera in several parts of the West African Coast. The Creek, which is heavily polluted with sewage and refuse, contains brackish water suitable for vibrio survival. Fishermen are highly susceptible to cholera infection because of their close contact with water which may be highly contaminated with cholera vibrios.

of the disease. This apparently premature disappearance may be explained by the typical high ratio of symptomless to symptomatic cases. As an illustration, if a town of 1,000 people had only seven clinical cases, up to 70% of the population could have been infected if a carrier to case ratio of 100:1 is assumed.

In addition to a decrease in the susceptible population, an increase in control measures, a mutational shift of the vibrio or a deterioration of the conditions facilitating survival of vibrios in the environment could lead to a containment of the epidemic before the critical point in the decline of the susceptible population is reached.

As the susceptible population declines beyond a critical limit, or as other factors unfavorable to the continuation of the epidemic become dominant, the epidemic peak passes and the disease again becomes inapparent. The infection may entirely die out, but if environmental conditions are conducive for its survival, a low profile, protracted man-environment transmission cycle may continue. Studies conducted in cholera-endemic areas in the Indian Subcontinent during an inter-epidemic period revealled that there was continuing vibrio survival and circulation, despite the total absence of clinical cases.[21] This protracted, invisible survival may be interrupted by a new introduction of cholera from an outside source, a change in the environmental conditions, or a change in the vibrio characteristics, possibly as a

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[21] R. Sinha et al., "Role of carriers in the epidemiology of cholera in Calcutta", Indian J. Med. Res., vol.56, No.7 (1968), pp.964-978.

result of vibrio mutation. The change in environmental or vibrio characteristics may be either unfavorable for vibrio survival, resulting in its total elimination, or favorable, thus promoting an increase in vibrio contamination and a new epidemic.

CHAPTER 8

SUMMARY AND CONCLUSIONS

The organization of the concluding chapter will be based on the series of study objectives outlined in the first chapter.

Spatial and Temporal Patterns of Cholera Diffusion

After six major cholera pandemics in the nineteenth century, cholera had virtually become confined to the Indian subcontinent after 1920. In 1961, a previously minor cholera biotype known as cholera El Tor spread from its endemic focus in Southern Celebes. This marked the beginning of the seventh cholera pandemic which has affected countries in South and South-East Asia, the Middle East, Southern Europe and Africa.

There have been five cholera epidemic zones in Africa located in North Africa, West Africa, East Africa, Angola and South-Eastern Africa. The diffusion of cholera has followed different patterns in each of these areas. In East Africa, it was mainly confined to nomadic peoples in Southern and Eastern Ethiopia and adjoining countries. The sudden appearance and disappearance of cholera in widely separated parts of North Africa complicates any determination of the means of transmission. The Angolan epidemic has centered on Luanda. There have been four phases namely diffusion from Luanda to other coastal cities, disappearance of cholera for several months, reappearance in an endemic form in Luanda, and renewed diffusion from Luanda to coastal and railway towns. In

Moçambique and Malawi, cholera diffusion axes followed railway lines. The transfer of the disease across the Zimbabwe border is suspected to be a result of liberation troop movements.

There were four types of diffusion in West Africa, namely coastal, riverine, urban hierarchical and radial contact diffusion. From its point of introduction in Guinea during August, 1970, the infection moved along the coast of West Africa, reaching Southern Cameroun in February, 1971. The spread of cholera was in conjunction with the migrations of fishermen. The coastal epidemic was based in rural fishing villages along lagoons, which provided ideal vibrio survival conditions. Coastal cities were frequently infected from nearby fishing villages, but the duration of infection was quite short in these cities. The coastal epidemic was principally water-borne and characterized by relatively low morbidity and mortality.

Riverine diffusion refers to the cholera diffusion axis centered in the Middle Niger Valley. The infection, which was introduced to Mopti by a traveller from Abidjan, quickly spread along the entire length of the Malian and Nigerien portions of the Niger River. A secondary phase consisted of the more gradual penetration of the upland valley periphery and the Senegal Valley. The infection proceeded down the Senegal Valley to the Atlantic Coast. The third phase is the continuing occurrence and diffusion to new areas related to the Sahelian drought. Distance from the point of origin in Mopti and riverine environment are the two variables found to significantly correlated with the occurrence and time of onset of cholera in Mali.

Urban hierarchical diffusion occurred in Nigeria and South-Central Niger. Cholera spread from Lagos to the regional centers of Ibadan, Kano and Port Harcourt, and from these cities to lower ordered cities within their spheres of influence. Diffusion followed the major road and railway lines. Distance from Lagos and urban importance were the two variables most significantly related to cholera occurrence and time of onset.

Radial contact diffusion was observed in the vicinity of Lake Chad in mid-1971. Because of extremely high morbidity and mortality rates, survivors fled from infected vaillages and carried the disease to other villages. The epidemic front moved outward from Lake Chad like a wave. The containment of the epidemic at the beginning of the rainy season is perhaps a result of people remaining at home to farm instead of fleeing.

A study of local cholera transmission in Lagos revealed a complex pattern of epidemic centers shifting between the four major geographical regions of Lagos Metropolitan Area. A peak in the epidemic curve of one of the four regions was generally followed after a few days by peaks in the adjoining regions. These increases in morbidity apparently originated at different times between December, 1970 and March, 1971 in Lagos Island, the North-Western Peripheral Suburbs and in Mainland Lagos. Within Lagos Island, the cholera epidemic began with an explosive outward movement of cases from the center of the island followed by an implosive return to the island's center. This sequence

resembled the pattern of a physical wave reflecting off a barrier. However, the explosive-implosive sequence has not been satisfactorily explained.

Spatial Diffusion Principles

The three major types of spatial diffusion identified by Gould,[1] namely, contact diffusion, hierarchical diffusion, and relocation (long distance) diffusion were observed in connection with the diffusion of cholera in Africa. For example, urban hierarchical diffusion occurred in Nigeria and possibly Angola. The initial importation of cholera by air travellers into Guinea, Angola and Moçambique, as well as the transfer of infection from Abidjan to Mopti are examples of long distance or relocation diffusion. Linear contact diffusion within a channel was observed in the valleys of the Niger and Senegal River and along the West African coast. Unchannelled radial contact diffusion occurred in the Lake Chad vicinity and in Northern Kenya.

The patterns of inter-divisional cholera diffusion observed in West Africa may be compared to those of cholera epidemics in the United States in the nineteenth century which have been described by Pyle.[2] The linear pattern of diffusion along waterways with onset a function

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[1] Gould, Spatial Diffusion, Washington: AAG Commission on College Geography Resource Paper No.4, 1969, p.35.

[2] G.F. Pyle, "The diffusion of cholera in the United States in the Nineteenth Century", Geog. Analysis, vol.1, No.1 (1969), pp.59-75.

of distance from the source of infection, which was found in Mali, is very similar to the 1832 American pattern. The urban hierarchical diffusion system of Nigeria corresponds to Pyle's finding of urban hierarchical diffusion in the United States in 1866. Pyle attributed the change in diffusion pattern in the United States to development of an integrated transportation system and a significant increase in urbanization between 1832 and 1866.[3] Parallel differences exist in Nigeria and Mali in the 1970's. Nigeria had twenty-five cities in 1963 exceeding 100,000 population, while the second largest city in Mali has only 32,000 people. Central and Eastern Mali's main transportation artery is the seasonally-navigable Niger River. Nigeria has a rather well developed road and railway network.

The relatively simple spatial concepts of permeable and impermeable barriers, channels and central place attraction were found to be useful in explaining diffusion patterns occurring in various areas. The most prominent type of barriers were physical barriers. The Atlantic Ocean, and Sahara Desert were barriers defining the Northern Southern and Western boundaries of the cholera-susceptible areas. Relatively unpopulated areas with poor transportation networks were barriers to diffusion in the Middle Belt, Central Chad and Southern Cameroun. Vaccination barriers and travel cordons were barriers of local significance.

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[3] Pyle, op.cit., p.71.

The channels most significant for diffusion were physical and transportation channels. River valley and coastal physical channels were most prominent in areas with relatively poor transportation and where population density was greatest along the corridor. Roads and railways frequently were important diffusion axes.

Central place attraction and the resulting large volume of travellers assured the infection of most important urban centers within the cholera-infected areas. Specific urban functions such as markets, transport functions and staging of traditional festivals were frequently found to be responsible for the introduction and dissemination of cholera into otherwise insignificant villages such as Goulfey (Cameroun) and Thiomène (Senegal).

Barrier, channel and central place effects are useful in explaining the patterns of inter-divisional diffusion of cholera, but are of little value in analyzing local diffusion, especially in large and environmentally complex urban areas. It is possible that a "probability of contact" model, perhaps using Markov Chain Analysis or the susceptible population - immune population models developed by mathematical epidemiologists could be successfully adapted for the analysis of cholera diffusion in local areas.

Model of Cholera Survival and Diffusion

The model of cholera survival and diffusion is composed of two parts, namely, local survival and diffusion and external or inter-

divisional diffusion. The effects of distance, barriers, channels and central place attraction, which are the main elements of external diffusion have been described in the previous sections.

The cholera transmission cycle and its relationship to particular environmental conditions in a community must be considered if local survival and diffusion processes are to be understood. General systems models are particularly useful for this purpose because they focus attention on the selection of relevant factors and a consideration of their interrelationships. The main elements in the local survival and diffusion system are the survival ability of the vibrio strain, form of introduction, environment for vibrio survival, size of the susceptible population and predisposing factors for the development of man-environment or person to person transmission cycles. The decline of an epidemic may result from changes in vibrio characteristics, the successful implementation of control measures or a substantial decline in the cholera-susceptible population.

Various facets of the standard of living, in particular environmental sanitation, pollution and protection of water sources, and population density are the major determinants of the suitability of an environment for cholera survival. Cholera primarily affects poor people living in crowded, unsanitary conditions. Programs such as cleanup campaigns, chlorination and vaccination may be used to reduce the favorability for survival.

The absolute and relative size of the cholera-susceptible population is a function of total population size, the number with initially high cholera antibody levels, the socio-economic structure of the community, the extent of control programs and age structure. However, the number of susceptibles in a population cannot be determined without knowledge of the extent of virbio contamination in a particular epidemic.

The risk of person to person spread is greatest where there is close contact between a vibrio carrier and susceptibles. Funerals, home care of cholera patients, and attendance at markets have promoted person to person dissemination in Africa. Personal hygiene and such practices as eating with hands or performing ablutions are suspected to have been involved in person to person cholera transmission.

The development of a man-environment transmission cycle stems from unsatisfactory disposal of sewage or uncontrolled defecation, leading to the contamination of the environment with vibrio-bearing feces. Establishment of a transmission cycle through pollution of water sources is the most common form, but soil, food or other goods may also become contaminated.

Although the dominance of person to person spread has been emphasized by several observers of cholera in West Africa, both types of transmission were found. Simultaneous man-environment and person to person spread probably occurred in many communities.

A variety of control programs were used to combat cholera in Africa. Mass vaccination programs were most prominent. It has been

claimed that vaccination cannot be justified when its cost is compared to the cost of treating cholera cases.[4] However, the scarcity of treatment facilities in rural Africa, the excessive mortality experienced in areas such as rural Mali and Chad where treatment was unavailable, compared to the small outbreaks in vaccination protected cities show that vaccination is a valuable and perhaps essential cholera control method in Africa. Other controls such as the prohibition of travel and market attendance were only partially successful. Belief systems and attitudes which were incompatable with the functioning of organized control programs created a problem in various areas, including Ibadan and Coastal Togo. The ultimate control program involves the elimination of environments suitable for vibrio survival through improved sanitation and housing and provision of protected water supplies. However, this would require huge sums of money and take decades to achieve.

Endemicity

Cholera appears to have disappeared from East Africa, where there have been no reported outbreaks since 1972. Reports of cases have continued from the other four African epidemic zones during 1973 and 1974. Twelve West African countries reported cholera in 1973 and the first half of 1974. These reports have been relatively infrequent

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[4] D. Barua, "Cholera vaccination as a tool for cholera control", Bull. Soc. Pathol. Exotique, vol.64 (1971), pp.652-659.

and scattered, suggesting a significant under-reporting of cases or that there are many small, scattered pockets of endemic cholera in West Africa.

The areas which have reported cases most frequently include Coastal Ghana, the Douala-Victoria area in Cameroun, Montserrado County (Monrovia), Liberia and the Lagos-Ibadan region in Nigeria. These locations all include areas of lagoon coast. Rainy season cholera epidemics, followed by the apparent disappearance of the infection have occurred in the Senegal Valley in 1971, 1972 and 1973. The continuing presence of cholera in the Sahel is related to drought conditions.

Cholera has persisted for four years, so permanent endemicity in Africa must be considered to be a definite possibility. If cholera continues to survive in Africa, new epidemic outbursts comparable to those of 1970-1971 are probable. With time, antibody levels in the population decline and number of children with no antibodies due to their never having been exposed to cholera increases. As the level of susceptibility of the population rises, the risk of a new epidemic grows. However, the apparent elimination of cholera from East Africa and such West African countries as Sierra Leone, Ivory Coast and Togo, which would seem to be favorable locations for cholera survival shows that permanent endemicity is not inevitable.

APPENDIX 1

MAP CODE FOR WEEK OF CHOLERA ONSET*

Week	Dates	Week	Dates
1	Aug.15-21, 1970	27	Feb.14-20, 1971
2	Aug.22-28	28	Feb.21-27
3	Aug.29-Sept.4	29	Feb.28-Mar.6
4	Sept.5-11	30	Mar.7-13
5	Sept.12-18	31	Mar.14-20
6	Sept.19-25	32	Mar.21-27
7	Sept.26-Oct.1	33	Mar.28-Apr.3
8	Oct.2-8	34	Apr.4-10
9	Oct.9-15	35	Apr.11-17
10	Oct.16-22	36	Apr.18-24
11	Oct.23-29	37	Apr.25-May 1
12	Oct.30-Nov.7	38	May 2-8
13	Nov.8-14	39	May 9-15
14	Nov.15-21	40	May 16-22
15	Nov.22-28	41	May 23-29
16	Nov.29-Dec.5	42	May 30-June 5
17	Dec.6-12	43	June 6-12
18	Dec.13-19	44	June 13-19
19	Dec.20-26	45	June 20-26
20	Dec.27-Jan.2, 1971	46	June 27-July 3
21	Jan.3-9	47	July 4-10
22	Jan.10-16	48	July 11-17
23	Jan.17-23	49	July 18-24
24	Jan.24-30	50	July 25-31
25	Jan.31-Feb.6	51	Aug.1-7
26	Feb.7-13	52	Aug.8-14

* Week 1 coincides with the first confirmed outbreaks in Africa (Guinea and Libya).

APPENDIX 1 (cont'd)

Week	Dates	Week	Dates
53	Aug.15-21, 1971	81	Feb.28-Mar.5, 1972
54	Aug.22-28	82	Mar.6-12
55	Aug.29-Sept.4	83	Mar.13-19
56	Sept.5-11	84	Mar.20-26
57	Sept.12-18	85	Mar.27-Apr.2
58	Sept.19-25	86	Apr.3-9
59	Sept.26-Oct.2	87	Apr.10-16
60	Oct.3-9	88	Apr.17-23
61	Oct.10-16	89	Apr.24-30
62	Oct.17-23	90	May 1-7
63	Oct.24-30	91	May 8-14
64	Oct.31-Nov.6	92	May 15-21
65	Nov.7-13	93	May 22-28
66	Nov.14-20	94	May 29-June 4
67	Nov.21-27	95	June 5-11
68	Nov.28-Dec.4	96	June 12-18
69	Dec.5-11	97	June 19-25
70	Dec.12-18	98	June 26-July 2
71	Dec.19-25	99	July 3-9
72	Dec.26-Jan.1, 1972	100	July 10-16
73	Jan.2-8	101	July 17-23
74	Jan.9-15	102	July 24-30
75	Jan.16-22	103	July 31-Aug.6
76	Jan.23-29	104	Aug.7-13
77	Jan.30-Feb.6	105	Aug.14-20
78	Feb.7-13	106	Aug.21-27
79	Feb.14-20	107	Aug.28-Sept.3
80	Feb.21-27	108	Sept.4-10

APPENDIX 1 (cont'd)

Week	Dates	Week	Dates
109	Sept.11-17, 1972	137	Mar.26-Apr.1, 1973
110	Sept.18-24	138	Apr.2-8
111	Sept.25-Oct.1	139	Apr.9-15
112	Oct.2-8	140	Apr.16-22
113	Oct.9-15	141	Apr.23-29
114	Oct.16-22	142	Apr.30-May 6
115	Oct.23-29	143	May 7-13
116	Oct.30-Nov.5	144	May 14-20
117	Nov.6-12	145	May 21-27
118	Nov.13-19	146	May 28-June 3
119	Nov.20-26	147	June 4-10
120	Nov.27-Dec.3	148	June 11-17
121	Dec.4-10	149	June 18-24
122	Dec.11-17	150	June 25-July 1
123	Dec.18-24	151	July 2-8
124	Dec.25-31	152	July 9- 15
125	Jan.1-7, 1973	153	July 16-22
126	Jan.8-14	154	July 23-29
127	Jan.15-21	155	July 30-Aug.5
128	Jan.22-28	156	Aug.6-12
129	Jan.29-Feb.4	157	Aug.13-19
130	Feb.5-11	158	Aug.20-26
131	Feb.12-18	159	Aug.29-Sept.2
132	Feb.19-25	160	Sept.3-9
133	Feb.26-Mar.4	161	Sept.10-16
134	Mar.5-11	162	Sept.17-23
135	Mar.12-18	163	Sept.24-30
136	Mar.19-25	164	Oct.1-7

APPENDIX 1 (cont'd)

Week	Dates	Week	Dates
165	Oct.8-14, 1973	184	Feb.18-24
166	Oct.15-21	185	Feb.25-Mar.3
167	Oct.22-28	186	Mar.4-10
168	Oct.29-Nov.4	187	Mar.11-17
169	Nov.5-11	188	Mar.18-24
170	Nov.12-18	189	Mar.25-31
171	Nov.19-25	190	Apr.1-7
172	Nov.26-Dec.2	191	Apr.8-14
173	Dec.3-9	192	Apr.15-21
174	Dec.10-16	193	Apr.22-28
175	Dec.17-23	194	Apr.29-May 5
176	Dec.24-30	195	May 6-12
177	Dec.31-Jan.6, 1974	196	May 13-19
178	Jan.7-13	197	May 20-26
179	Jan.14-20	198	May 27-June 2
180	Jan.21-27	199	June 3-9
181	Jan.28-Feb.3	200	June 10-16
182	Feb.4-10		
183	Feb.11-17		

APPENDIX 2

MAP PLACE NAME CODE

Code	Division
	Cameroun
K5	Kribi
L5	Lomet Djerem
M5	Méfou
	Dahomey
A1	Atlantique
M1	Mono
O1	Ouémé
Z1	Zou
	Ghana
A2	Ashanti
B2	Brong-Ahafo
C1	Central
E1	Eastern
V1	Volta
W1	Western
	Ivory Coast
A6	Aboisso
A4	Adiaké
A8	Alépé
B3	Bingerville
D1	Dabou
G2	Grand Bassam
	Liberia
B4	Bong
C2	Cape Mount
G3	Grand Bassa
G4	Grand Gedeh
M2	Maryland
M3	Montserrado
N1	Nimba
	Mali
B6	Banamba
B10	Bankass
D5	Djenné
K7	Kadiolo
K8	Kangaba
M6	Macina
T3	Touminian
Y3	Yorosso

Code	Division
	Nigeria
A11	Aba
A12	Abakaliki
A14	Aboh
A16	Auchi
B18	Badagry
B25	Brass
D10	Degema
E4	Eket
E5	Egbado
I7	Ikot Ekpene
I8	Ikom
I13	Ilesha
K15	Kabba
N7	Ngwa (Umushia)
O5	Owerri
O6	Okene
O10	Opobo
O12	Oshun
O15	Okitipupa
U3	Ughelli
W3	Western Ijaw
W4	Warri
	Sierra Leone
E4	Eastern
N4	Northern
S4	Southern
	Togo
A5	Akposso
A7	Atakpame
A10	Anecho
L6	Lama-Kara
M9	Maritime
N6	Nuatja
P6	Palimé
S9	Sokodé
T6	Tabligbo
T7	Tsévié
V7	Vogan

APPENDIX 2 (cont'd)

Code	Division	Code	Division
	Upper Volta		
B31	Boulsa	K34	Koupéla
B32	Barsalago	M30	Manga
B33	Bogandé	033	Orodara
B34	Boromo	P30	Pissila
D30	Djibo	P31	Po
D32	Dédougou	S30	Saponé
D33	Diébougou	S31	Seguénéga
D34	Diapaga	T30	Titao
K30	Kaya	T31	Tiébéle
K31	Kombissiri	T34	Tenkodogo
K32	Kongoussi	Y30	Yako
K33	Koudougou		

APPENDIX 3

SUMMARY OF CHOLERA ONSET, MORBIDITY, MORTALITY

AND DURATION OF INFECTION: AUGUST, 1970 - JULY, 1974.

Primary Source: Weekly Epidemiological Record, 1970-1974, Weekly reports of cholera.

Secondary Sources: Where more complete data are available from other sources, these have been listed. When the total of cases differs from the number reported in the annual Weekly Epidemiological Record summary of cholera incidence, both figures are listed, the latter in brackets. The 1974 time period varies, as some countries report less promptly.

S Suspected cases, but none reported in the Weekly Epidemiological Record.

R Reported outbreak; no data available.

M Months

\+ Incomplete data

#1 Footnote. See the last page of the table.

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
ALGERIA #1						
Annaba	65		2//	10/0/1		
Aurès	58		27//	1/0/1	10/0/1	
Algiers	54		292//			
Constantine	58		130//			
El Asnam	53		129//		5/0/1	
Maghnia	48		1/1/1			
Mostaganem	52		300//		10/1/1	

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Oasis	59		9//			
Oran	52		193//			
Sétif	56		78//	6/0/1		
Tittéri	103			10/0/1		
Tiaret	53		16//			
Tizi-Ouzou	56		75//			
Tlemcen	49		81//			
Sidi Bel-Abbès	51				6/0/1	
Gazouet	111				2/0/1	
Sebdon	111				1/0/1	
Beni Sef	50		1/0/1		5/0/1	
TOTAL			1,332/#1 (109)	27/0	39/1	
ANGOLA						
Ambriz	193					1/0/1
Benguela	72			128+/10/12		9/2/2
Cacuso	176					6/0/5
Calulo	77			5/0/2		
Carmona	177					
Cazengo	180					1/0/1
Dondo	179					5/0/4
Duque de Braganca	176					6/1/2
Golungo Alto	188					8/0/4
Gabela	75			18/2/2		
Libolo	75			1/1/1		
Lobito	76			17/0/9		5/0/2
Luanda	71		1/0/1	71/7/9	268/9/46	538/7/19
Moçamedes	179					19/1/7

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Nova Redonda	75			8/2/3		4/1/3
Porto Alexandre	185					36/1/9
Porto Amboin	76			5/1/2		
Sa de Bandeira	193					1/0/1
TOTAL			1/0/1	189/23	268/9	
CAMEROUN						
Wouri	28		363/33/15	252/12/44		4/0/3
(Douala)	27		75/11/1			
Mungo	28		92/27/4	51/6/19		
Kumba	29		22/9 /4	22/4/5		2/0/1
Victoria	29		157/4/21	36/2/9	8/0/2	
Méfou	29		5/0/2		1/0/1	
N'Kam	29		37/2/2	8/1/5		
Sanaga-Maritime	29		47/3/7	7/3/5		
Haut N'Kam	31		1/0/1	1/0/1		
Kribi	30		1/0/1			
Bamoun	36		1/1/1	1/0/1		
Lom et Djerem	68		2/1/1			
Nyong et Kelle	35		6/2/2			
Margui-Wandala	35		1/0/1			
Logone-Chari	40		1024/178/10	3/0/1		
Diamaré	40		2/0/2			
Mayo Danai	45		272/9/10			
TOTAL			2,167/280	379/28 (362	9/0	6/0

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
CENTRAL AFRICAN REPUBLIC			S(June) #2			
CHAD						
Chari-Baguirmi	42		6203/1939/18	5/1/3		
Fort Lamy	38		22/5/2			
Kanem	47		48/16/2			
Lac	42		897/281/8			4/1/1
(Ile Kindjeria)	190					52/16/1
Mayo-Kebbi	44		717/92/23			
Ouaddi	46		4/3/1			
Tandjilé	47		27/1/8			
TOTAL			8225/2337	5/1		56/17
DAHOMEY #3						
Atlantique	19	80/28/1	853/139/37	75/5/29		
Borgou	22		271/25/7	44/6/4		
Mono	17	95/15/4	312/37/20	22/6/4		
Ouémé	19	71/17/1	655/68/42	40/3/15		
Zou	24		27/0			
TOTAL		246/50	2108/267	181/20		

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
EGYPT #4						
Alexandria		S				
Cairo		S				
ETHIOPIA						
Harar	13	270/22/1	S			
Shoa	13	433/44/1	S			
Wollo	13	147/6/1	S			
Eritrea		S				S #5
Gemo-Gofa			S			
Kefa			S			
Arusi		S	S			
Tigre		S				
Sidamo		S	S			
TOTAL		850/72				
F.T.A.I. (Djibouti)						
Ali Subieh	12	1/0/1	R			
Bal Balah	13	1/1/1	14/5/4			
Dikkil	47		22/4/1			
Djibouti	13	4/2/2	282/60/10	1/0/1		
Obock	48		1/0/1			
Todjourah	48		1/0/1			
Daoudouya	76			8/2/2		
Guifou	45		R			
Randa			R			
Hol-Hol	43		R			
TOTAL		6/3	320/69 (440)	9/2		

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
GAMBIA						
Bathurst	55		3/1/1			
GHANA						
Ashanti	22		640/47/45	34/1/11		
Brong-Ahafo	21		125/15/13			
Central	17	1680/27/5	4841/240/45	116/9/21	397/30/18	20/1/7
Eastern	16	156/9/5	2349/133/51	165/6/26	1/0/1	5/1/1
(Accra)	18	306/14/5	1013/38/43	7/6/3	4/0/1	4/0/3
Northern	21		19/3/4			
Volta	18	455/20/3	1131/35/45	11/0/3		
Western	15	289/3/5	2525/101/44	200/30/5	251/6/24	
Upper						
TOTAL		2,886/73 (3,557)	12,623/609 (13,057)	534/21 (619)	653/36	98/2
GUINEA						
Conakry & vicinity	1	2000/60				
IVORY COAST						
Abengown	11	*#7	4/0/1M			
Abidjan	11	837/38	203+/3+/3M			
Adiaki	22		218/0+/4M			
Agboville	11	#7	R			
Bingerville	10	1/1	R			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Dabou	20		64/0+/4M			
Dimbroko	31		20/0+/2M			
Grand Lahon	35		5/0/1M			
Jacqueville	9		R			
Tiassalé	27		12/5/3M			
Aboisso	11	#7	#7			
Bouaké	11	#6				
Adzopé	11	#7				
Grand Bassam		S				
Alépé		S	R			
TOTAL		(828)	(668)			
KENYA						
Garissa	25		R	1/0/1		
Hola	22		R			
Kitui	33		R			
Mandera	22		6+/0/2			
Marsabit	66		32/6/5	2/0/2		
N.Horr	57		R			
Tana R.	81			1/0/1		
Trans-Nzoia (Kitale)	80			R		
Turkana	32		51/17/8	9/0/2		
Wajir	22		3+/0/2	9/0/2		
W. Pokot	80			18/0/3		
TOTAL			92/23 (301)	40/0		

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
LIBERIA						
Bong Co.	20		6/0/3M			
Cape Mount	20		69/20/3M	6/4/2M		
Grand Bassa	20		16/6/3M			
Grand Gedeh	20		40/10/1M			
Maryland	51		35/8/2M			
Montserrado	7		22/0/2M	65/2/1M	1067/18/9M	112/2/3M
(Monrovia)	7	30/0/1		75/3/2M		
Nimba	51		6/0/2M			
TOTAL		30/0 (121)	194/44 (606)	146/9 (947)	1067/18	112/2
LIBYA						
Tobruk	1	21/0/1				
Tripoli	1	7/0/1				
TOTAL		28/0				
MALAWI						
SOUTHERN REGION						
Blantyre	176				9/0/1	98/0/4
Chikwawa	167				152/14/10	23/0+/4
Chirandzulu	176				3/0/1	61/0+/8
Kasupe	178				3/0/1	48/0+/4
Mangoshe	180					11/0/4

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Mlange	176				9/0/1	71/0+/7
Nsanje	167				180/0+/10	7/0/2
Mwanza	180					22/0/5
Thyole	176				7/0/1	12/0/6
Zomba	176				2/0/1	28/4/8
CENTRAL REGION						
Dedza	179					18/0+/3
Dowa	190					18/0/4
Lilongwe	180					53/0+/4
Nkota Kotu	196					3/0/2
Ncheu	176				8/0/1	88/0+/6
Salima	176				1/0/1	37/0+/4
NORTHERN REGION						
Karonga	181					24/0/5
Nkhata Bay	181					17/0/5
TOTAL					271/14+	622/0+
MALI						
Bamako	17	115/1/3				
Banamba	37		2/1/1			
Dioila	56		13/2/3			
Kolokani	49		2/2/2			
Koulikoro	16	63/0+/3	72/19/6			
Nara	38		1129/332/13			
Koulouba	20		39/0/7			
GAO						
Gao	16	210/24+/4	7/3/4			
Ansongo	20		3/0/1			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Bourem	28		1/1/1			
Diré	15	298/10+/5#8	117/38/15	6/1/1	6/1/1	
Goundam	15	620/23+/4#8	89/26/5	138+/16/3	138+/16/3	
Menaka	32		48/18/4			
Tombouctou	16	86/9/3				
KAYES						
Kayes	42		142/15/8			
Bafoulabé	38		268/16/4			
Nioro	62		1/0/1			
MOPTI						
Mopti	14	895/71+/6	270/27/37		25/1/3	130/21/1
Bandiagara	25		139/37/8			
Bankass	40		75/3/4			
Djenné	16	68/0+/2	38/11/2		16/3/2	
Douzentza	16	354/0+/3	47/15/4			
Koro	25		391/39/7			
Niafunké	15	583/16+/3	89/35/3			
Tenenkou	15	404/104+/5	71/21/6			
SEGOU						
Ségou	15	236/1+/5	246/85/21		39/11/3	8/0/1
Macina	15	114/8+/5	96/24/10			
Niono	17	13/2/2	337/107/11		4/1/2	
San	42		428/8/9	2/0/1		
Touminian	50		16/10/3			
SIKASSO						
Koutiala	35		23/15/3			
TOTAL		2665/269	3866/437 (4,792)	2/0	228+/33+	138/21

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
MAURITANIA						
1st REGION						
Abdel Bagrou	43		301/12/5			
Amourj	43		63/9/4			
Boustiella	50		152/3/4			
Djiguéni	50		364/7/9	32/0/1		
Néma	50		278/9/7	2/0/1		
Timbédora	51		66/1/5	3/1/2		
4th REGION						
Kaedi	50		80+/1+/10		1/0/1	
5th REGION						
Boghé	52		94/20/9	2/0/1		
M'Bagne	53		1+/0/2			
Moudjeria	190				51/7/2	
6th REGION				24/11/2		
Nouakchott	104			3/0/1	3/0/1	
Rosso	104		S	8/0/1	84/11/4	
3rd REGION (Sélibaby)			S			
TOTAL			1398+/62+ (1139)	74/12 (148)	139/18	
MOROCCO #9						
Fès	109			3/0/2		
Kenitra	104			1/0/1		
Nador	46		56/0/1	2/0/1		
Oujda	105			1/0/1		
TOTAL			56/0	7/0		

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
MOÇAMBIQUE						
INHAMBANA						
Govuro	174				6/1/1	1/0/1
MANICA & SOFALA						
Baruè	170				23/0/1	
Beira	162				236/14/15	280/56/18
Cheringoma	165				9/1/3	1/0/1
Chimoio	165				19/1/6	55/4/9
Manica	164				2/0/1	45/11/5
Marromeu	171				3/0/2	3/3/1
Sena	171				7/0/1	7/0/1
Vila Fontes	182					7/1/3
LORENCO MARQUES						
Lorenço Marques	179					176/21/17
Manhica	183					5/0/4
TETE						96/6/13
Mutarara	164				179/21/6	45+/6/10
Tete	170				113/26/7	
ZAMBEZIA						
Ile	172				32/3/3	
Mopeia	168				5/1/2	8/1/5
Morrumbala	166				54/8/8	
Quelimane	168				103/17/6	52/4/11
TOTAL					795/92	785+/112

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
NIGER REPUBLIC						
AGADES						
Bilma	45		4/0/1			
DIFFA						
Diffa	44		30/22/2			47/26/2
N'Guigmi	42		89/34/3			
DOSSO						
Dogon-Doutchi	27		4/1/1		55/13/2	
Gaya	23		3/1/1			
Birni N'Gouré	50		1/0/1			
Birni N'Konni	50		R/-/1			
MARADI						
Dakoro	47		141/48/4			
Maradi	33		222/62/7			52/16/4
Mayahi	37		1133/336/10			
Tessaoua	36		2213/619/10			
Takalgos	183					21+/10+/2
NIAMEY						
Filingué	21		1/0/1			
Niamey	21		850/95/23		3/0/1	4+/0/3
Ouallam	21		159/37/14			
Say	32		113/24/9			
Téra	21	R	1677/239/31		95/9/8	
Tillabery	21	R	1188/120/33		15/7/2	
ZINDER						
Gouré	37		80/64/1			
Magaria	82			51/12/2		
Matameye	40		23/2/1			
Mirria	41		76/22/2			107+/10+/2
Tanout	37		925/457/8			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Zinder	37		152/73/4			
TAHOUA						
Bouza	47		84/44/5			
TOTAL		(16)	(9255)	51/12	168/29	304/58
NIGERIA						
BENUE PLATEAU						
Benue Prov.	30		54/12/3			
Gboko	34		R			
Makurdi	35		R			
Wukari	30		28+/15/4			
Plateau Prov.	36		R			
Idoma	30		R			
Jos	36		16/5/4			
EAST CENTRAL			61/1/4	28/2/5	5/0/1	
Aba	29		27/0/9	4+/0/2		
Abakaliki	73		R	R		
Enugu	28		7/5/1			
Ngwa	29		R			
Onitsha	29		R			
Orlu	45		1/1/1			
Owerrix	32		6+/2/4			
Ukwa						
KANO					S	
Kano	28		2299/926/4+			
Northern	30		S			
KWARA			149/15/9			
Ilorin	30		265/27/17			
Kabba	49		R			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Idah	32		R			
Kotonkarfi	32		R			
Okene	33		R			
Borgu	33		R			
LAGOS #10		15/4/1	3541/219/12M	1175/38/12M	192/3/7	
Badagry	22		R	R		
Epe	23		R	R		
Ikeja	20	R	R	R		
Ikorodu	23		R	R		
Lagos	20	R	R	R		
MIDWEST #11			758/39/	7/3/3	4/0/1	4/0/2
Agbor in			3+/0/3			
Aboh	30		R			
Asaba	29		R			
Benin	29		R			
Ika			R			
Owan			R			
Ughelli	30		R			
Urhobo			R			
Urhobo E.			R			
W. Ijaw	25		R			
Warri	26		R			
Sapele			R			
Auchi	30		R			
NORTH CENTRAL			1062/82/22	4/0/1	3/0/3	11/0/4
Funtua			R			
Kaduna	30		213+/33/13			
Katsina	29		39+/2+/19			
Zaria	29		300+/9+/21			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
NORTH EAST			0+/7/1			116/11/5
Adamawa	46					
Azare			S			
Bauchi	38		R			12/3/1
Bornu	38		R			32/2/1
(Maiduguri)	38		44/0/7			
Bedde			S			
Bama			S			
NORTHWEST			322+/818/8			
Niger Prov.			R/3			
Abuja	46		R			
Bida	28		R			
Kontagora	46		R			
Minna	46		R			
Sokoto Prov.	29		R/4			
Argurgu	44		R			
Gusau			R			
Kebbi	44		R			
Sokoto	29		8+/2/3			
Yauri			R			
RIVERS				1/0/1		
Ahoada			S			
Brass	27		7/0/1			
Degema	60		S			
Ogoni	14			3/0/1		
Port Harcourt	27		R			
SOUTH EAST			1625/165/6		135/4/3	
Abak	33		R/2			
Calabar	29		R/5			
Eket	29		R/4			
Ikot Ekpene	29		R/4			
Itu	32		R/2			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Obubra	46		R/2			
Opobo	32		R/2			
Uyo	29		R/7			
WESTERN			1048/281/26	150/2/5	744/27/29	7/4/4
Abeokuta	28		R/17		R	
(Egba)	28		R			
(Egbado)	28		R			
Ekiti	32		R/10			
Ibadan	22		782+/119/33	966/23/13		
Ife	25		R/19			
Ijebu	25		R/22			
(Shagamu)	25		R			
(Ijebu Ode)	28		R			
Ilesha	28		R/21			
Ondo	30		R/15			
Oshun	29		R/18			
Owo	28		R			
Oyo	30		R/15			
Otikpupa	26		R			
TOTAL		15/4	(22,747)	(3,740)	1,083/34	182/20
RHODESIA						
Darwin	168				47/6/7	257/26/10
Mtoko	170				60/0/6	
Chipinga	174				1/0/1	165/8/14
Melsetter	182					12/2/5
Chiredzi	192					7/1/4
TOTAL					108/6	441/37

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
SENEGAL						
CAP VERTE	55		7/1/3	17/2/4	73/3/8	
DIOURBEL	64		1/0/1	106/7/1	1788/103/21	
Louga	64		81/14/6	54/6/6		
FLEUVE					288/52/16	
Dangana	54		3/2/1	129/16/11		
Matam	54		12/2/3			
Podor	53		150/35/4	14/4/1		
St. Louis	55		R	9/0/2		
ORIENTAL						
Bakel	50		12/7/2			
SINÉSALOUM	153				55/8/11	
THIÈS	108			R	13/2/2	
M'Bour	108			138/0+		
TOTAL			266/61	462+/35	2217/168	
SIERRA LEONE						
EASTERN	19		8/0/4			
NORTHERN	5	51+/4/9	171/33/25			
SOUTHERN	15	4/0/1	40/5/11			
WESTERN	10	9/9/5	20/3/6			
(Freetown)	8	38/15/7	8/2/6			
TOTAL		102+/28 (293)	247/43 (211)			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
SOMALIA						
Belet Uen		5/0/2				
Hargeisa			1+/0/1			
Mogadiscio		R	70/11/23			
BENADIR						
Balad Town			13/1/7			
Coriole Town			27/7/1			
Goihar Town			12/1/6			
TOTAL		5/0 (43)	122/20 (79)			
SUDAN #12			S			
TANZANIA						
Kyella (Mwanza)	203					15/8/2
TOGO						
MARITIME	16					
Anécho	18	19/2/3	63/4/22			28/3/9
Vogan			R			
Tabligbo	22		8/0/2			
PLATEAU						
Nuatja	31		4/0/1			
Palimé	27		17/0/2			
Atakpamé	56		26/0/3	11/0/1		
Akposso			R			
LOME			14/0/5			
Lomé	16	23/1/7	27/0/23			8/0/2

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
Tsévié	21		33/15/7			
SOKODÉ	52		R	5/0/2		
LAMA-KARA	57		4/0/2			
TOTAL		42/3	198/19	16/0		36/3
TUNISIA					S #14	
Djerba	3	S				
Gabès	153			2/0/1		
Gafsa	153			2/0/1		
Cap Bon #13	1	22/0				
TOTAL		39/0 #13		4/0		
UGANDA						
KARAMOJA			471/10/9			
Lapoko	42					
Maroto	43					
Amudat	43					
TOTAL			471/10 (757)			

APPENDIX 3 (Continued)

Division	Week of Onset	cases/deaths/weeks of infection 1970	1971	1972	1973	1974
UPPER VOLTA						
Bobo Dioulasso	27		1/0/1			
Boulsa	153				2/2/2	
Djibo	34	R	343/71/13		31/10/3	
Dori	19		731/217/20		486/138/9	
Fada N'Gourma	166					
Kaya	161				6/1/3	
Kombissiri	155				3/0/3	
Kongoussi	162				1/0/1	
Koudougou	155				7/0/5	
Koupéla	166				32/3/3	
Nouna	25		161/71/8	1/0/1		
Ouagadougou	40		1/0/1		297/23/22	
Ouagouiya	18		5/4/4		10/1/5	
Oudlan	40		213/64/7		82/25/8	
Pissila	161				2/0/2	
Saponé	159				2/0/2	
Séguenéga	160				1/0/1	
Titao	34		141/53/3			
Tiébélé	174				1/0/1	
Tougan	22		137/40/19			
Yako	168				1/0/1	
TOTAL		(25)	1588/520 (1761)	1/0	948/203	

FOOTNOTES

1. 1971 Algerian data from Mered, 1971.

2. Briefly noted by Félix, 1971C. No details given.

3. The following data are reported by Comité centrale de lutte contre le choléra (1971) for 1970 and up to February 13, 1971: Mono (1970): 1,018/95, (1971): 1,222/203; Atlantique (1970); 79:21, (1971): 1,794/401; Ouémé (1970): 30/5, (1971): 1,455/208.

4. No official report. According to the New York Herald Tribune, August 1, 1970 there were 1,500 victims in Alexandria and several in Cairo.

5. Outbreak at Shep mentioned in The London Free Press, London, Ontario, September 24, 1974, p. 33.

6. Reported in Félix, 1971C.

7. Reported in Bourgeade, Rive, et. al., 1973.

8. Félix, 1971A reports the occurrence of at least 5,000 cases and 2,000 deaths in Dire and Goundam in 1970 among sedentary farmers, plus an unknown number among fishermen and nomads.

9. The Moroccan epidemic was almost certainly more serious (space, time, number of victims) than reported.

10. Lagos State 1970-1972 data from B. Dada, 1972.

11. Mid-Western State 1971 data from Amu, 1971.

12. Reported by Félix, 1971B and 1971C.

13. According to Ben Rachid et. al., 1971, the 1970 Cap Bon outbreak included 22 patients at Kéliéba, 13 at Boukrim, 2 at La Goulette, and 1 at Kamara and Menzel Teminé.

14. Imported cases in Europe in August - September, 1973 had come from Tunisia.

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