THE AMERICAN JOURNAL OF TROPICAL MEDICINE AND HYGIENE

Vol. 20, No. 6
Printed in U.S.A.

EPIDEMIC SHIGA-BACILLUS DYSENTERY IN CENTRAL AMERICA

DERIVATION OF THE EPIDEMIC AND ITS PROGRESSION IN GUATEMALA, 1968–69*

CÉSAR A. MENDIZÁBAL-MORRIS,† LEONARDO J. MATA,‡
EUGENE J. GANGAROSA,§ AND GUILLERMO GUZMÁN¶
Ministry of Public Health, Guatemala, Institute of Nutrition of Central America and Panamá, and Center for Disease Control, Atlanta, Georgia 30333

ABSTRACT: The Central American epidemic of Shiga dysentery began in 1968 or early 1969 in the southwestern part of Guatemala. Epidemiologic evidence indicates that the epidemic extended rapidly eastward to the border of El Salvador and then to the north, encompassing the entire country during 1969. Data obtained from municipal death registers throughout the country revealed high death rates from dysentery in all age groups, particularly in children and aged persons. This indicated the interaction of a virulent organism in a highly susceptible population. Death rates were highest in the more densely populated lowlands, where malnutrition was more common and climatic conditions more severe. Males in all age groups were more severely affected than females. A study of sera obtained in a nationwide survey in 1965 demonstrated that the epidemic derived from a long-standing endemic focus.

(Accepted 22 March 1971)

The other papers in this series describe etiologic and epidemiological characteristics of an unusually severe regional epidemic of bacillary dysentery.[1-3] The etiologic agent was *Shigella dysenteriae* type 1, the classic Shiga bacillus. It caused explosive and widespread outbreaks of illness characterized by diarrhea with 10 to 40 or more bowel movements daily, mucus and blood in stools, tenesmus, dehydration, toxic manifestations, and an excessive number of deaths.

This report deals with the evolution of the outbreak, its magnitude, persons affected, and the progression of the epidemic in Guatemala in 1968–69.

* This research was aided in part by the U.S. Armed Forces Research and Development Command (Grant No. DADA-17-69-G-9283), the Pan American Health Organization, the U.S. Department of Health, Education, and Welfare (NIH Grant AI-05405), and the Guatemalan Public Health Department.

† Present address: Epidemiologist, Pan American Health Organization, Region IV, Lima, Perú.

‡ Present address: Chief, Microbiology Division, Institute of Nutrition of Central America and Panamá (INCAP), Guatemala.

§ Deputy Chief, Bacterial Diseases Branch, Epidemiology Program, Center for Disease Control (CDC), Atlanta, Georgia 30333.

¶ Present address: Epidemiologist, Institute of Nutrition of Central America and Panamá (INCAP), Guatemala.

METHOD OF INVESTIGATION

The data presented in this report were derived from three sources: a survey by questionnaire of the mortality throughout the country; records of dysentery patients seen in Guatemalan hospitals; and serological surveys conducted 3 years before and during the epidemic.

Mortality

A questionnaire was sent to all 325 municipalities of Guatemala in October 1969. The form inquired about deaths reported in the official register. Deaths due to dysentery were to be recorded by age, sex, and month during the period September 1968 through August 1969.

The basis for the use of official registers in Guatemala for analysis of deaths due to gastroenteritis, diarrhea, or dysentery was established by Behar et al.[4] Those workers showed that the causes of death as listed in the official register agreed with the medical diagnosis in health clinics. Nearly all, but certaintly not all, deaths are registered in Guatemala, because burial is not legally possible without an accompanying certificate from the local official registrar.

About a fifth of deaths in rural areas are reported by physicians and certified as to cause. Most deaths are reported by relatives, the police,

or neighbors, and the causes are often indicated in lay terms. Nevertheless, people are familiar with the signs and symptoms of dysentery.

Completed questionnaires were returned by 142 (43.7%) of the 325 municipalities of Guatemala, representing 2,256,645 people or 45.3% of the country's total population. These municipalities were scattered throughout the country so that each of the 22 departments (provinces) were represented.

Information on the elevation above sea level of the reporting municipalities was obtained from official sources.

To validate the data reported in the questionnaires, investigators visited 48 of the responding municipalities and examined their official registers. Agreement was good.

Reported deaths due to dysentery for the period 1960–68 for the country as a whole were determined from official death registers by the Statistics Department of the Guatemalan Government. The population was estimated for each of these years from the 1964 census.

To determine the origin of the outbreak and its direction of spread, we analyzed epidemic curves of individual municipalities. The month of the first definite increase in the number of cases was taken as the time of onset of the epidemic for that particular locality.

Hospital Records

Data regarding dysentery, enteritis, and other forms of diarrheal disease were available from official registers of all state-supported hospitals in Guatemala, except the Roosevelt Hospital, for the years 1968 and 1969. This provided only a small proportion of the morbidity in the population; however, the consistency of attendance to hospitals and health centers and of recording and reporting over long periods provide a sound basis for comparison of different years, with a likely constant error. The records included information on outpatients and hospitalized patients who died or were discharged. Diagnosis of dysentery included bacillary and amebic types. Dysentery associated with liver or other visceral abscesses were excluded from the tabulations.

Serological Surveys

A serological study was carried out in 21 affected communities in 12 of the 22 departments of the country in October and November 1969. Specimens of whole blood were collected in Vacutainer tubes from persons that had experienced dysentery in 1969. Their household contacts and neighbors with no history of illness were also bled. Specimens were transported to the laboratory under refrigeration; sera were separated under sterile conditions and frozen. The Neter-Young[5,6] passive hemagglutination test modified by Cáceres and Mata[7] was used to measure antibodies against *S. dysenteriae* type 1. Agglutination of antigen-adsorbed erythrocytes with a 1:40 serum dilution was considered indicative of infection with the Shiga bacillus. The technique is highly specific for antibodies to the Shiga bacillus and other shigellae, as determined in man and rabbits.

Results were compared with those of a serosurvey conducted in 1965 in 39 municipalities, 22 of which were highland communities and 17 lowland. Communities and survey samples of people bled were taken randomly.

RESULTS

Evolution of the Epidemic

The earliest recorded epidemics occurred in 1968, in southwestern Guatemala near México. From January to March 1969 the focus extended throughout the coastal region and the mountains of the Pacific slope (Fig. 1). The disease spread eastward to the border of El Salvador and then to the north.

Mortality Rates

Deaths due to dysentery are presented by age and sex in Table 1. Rates were significantly highest for the youngest children and the aged, but persons of all ages were affected. Data for children under 1 year of age were not sufficient for us to draw any conclusions regarding attack rates for that group by month of age.

Sex-specific attack rates were highest for males in each age group. The most striking male predominance was in the 15-year to 44-year age group, suggesting greater male exposure or susceptibility, or both.

From September through December 1968, dysentery deaths reported from the municipalities were below 340 per month, but during the period January through March 1969 the monthly average increased to 485. This was followed by a steady month-by-month rise to a peak of 1,100 deaths

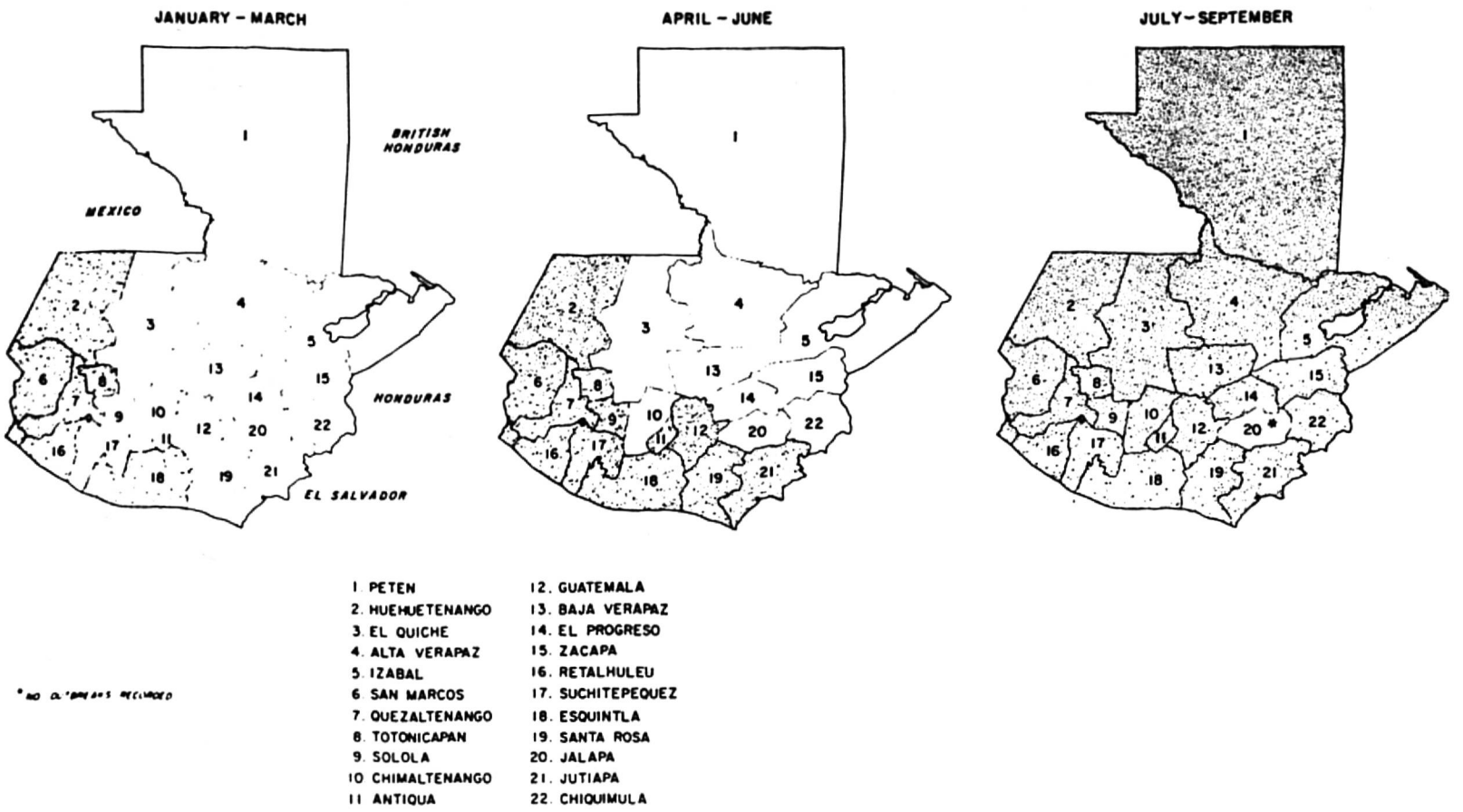


FIGURE 1. Progression of the epidemic of Shiga dysentery, Guatemala, 1969

in July. In August and September there was a slight decline to 997 and 779 deaths, respectively. Surveillance late in 1969 and early in 1970 indicated that the epidemic had not abated.

The death rate due to dysentery for the country was 250 per 100,000 (estimated) for the 1-year period covered by the questionnaire. This rate is fivefold greater than that of the preceding year (1968), and sevenfold greater than that of 1967. The high rate during the epidemic year is in striking contrast to the lower rates calculated in earlier years from the same official registers. (Fig. 2).

A total of 5,640 deaths were recorded from the 142 municipalities during the 1-year period covered by the questionnaire. If this figure is extrapolated to the country as a whole, an estimated 12,500 deaths occurred during that year.

Death rates showed that the highest rates were in certain lowland regions, especially in the departments of El Petén, Baja Verapaz, Quezaltenango, Suchitépequez, Zacapa, Chiquimula, and Jutiapa . The lowest rates were observed in the highlands, particularly in Sacatepéquez.

TABLE 1

Deaths caused by dysentery per 100,000, by age and sex in 142 municipalities, Guatemala, September 1968 through August 1969

Age (*years*)	Sex	Population	Deaths	Rate
0–4	M	208,572	1,450	695.2
	F	206,853	1,171	566.1
		415,425	2,621	630.9
5–14	M	339,164	538	158.6
	F	327,621	411	125.4
		666,785	949	142.3
15–44	M	424,574	560	131.9
	F	448,724	417	92.9
		873,298	977	111.9
45+	M	151,488	616	406.6
	F	149,649	467	312.1
		301,137	1,083	359.6
Total	M	1,123,798	3,164	281.5
	F	1,132,847	2,466	217.7
		2,256,645	5,630	249.5

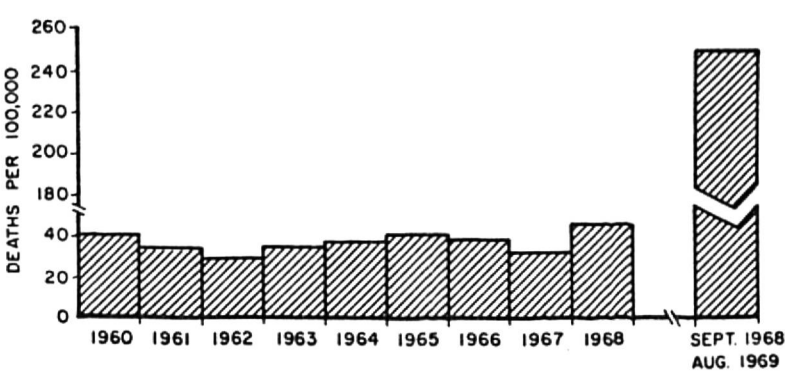
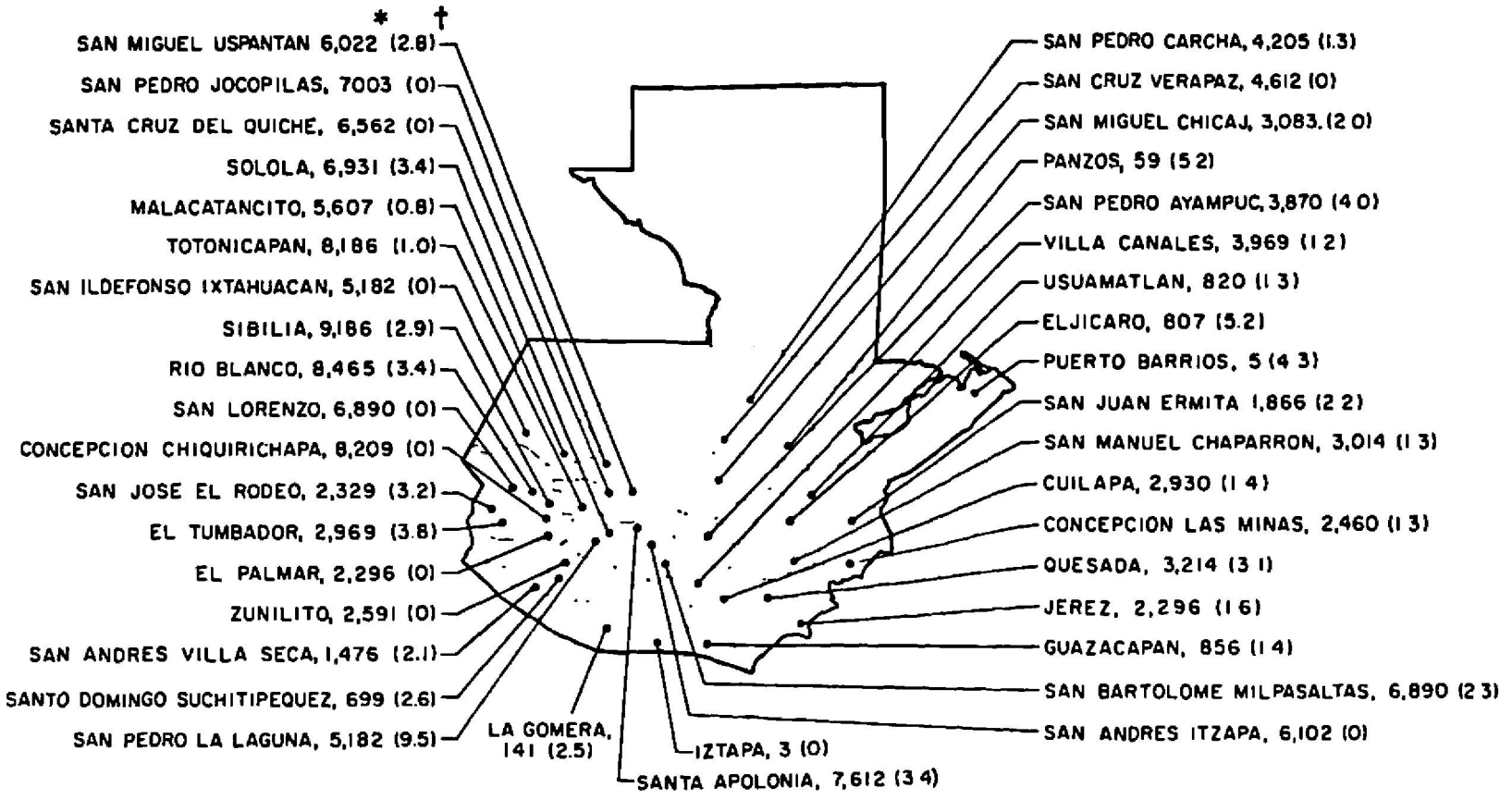


FIGURE 2. Mortality rates for dysentery, Guatemala, 1960–69.



* ELEVATION IN FEET ABOVE SEA LEVEL

† PERCENT OF PERSONS SURVEYED WITH TITERS AGAINST *S. DYSENTERIAE* I ≥ 1:40

FIGURE 3. Distribution of endemic Shiga-bacillus dysentery determined by a serological survey of 2,894 persons of all ages in 39 communities, Guatemala, 1965.

Specific attack rates by altitude above sea level are recorded in Table 2. Rates were significantly greater in the lowlands, and decreased proportionally with increasing altitudes.

Hospital Statistics

The number of outpatient visits and hospital discharges are presented in Table 3. It is evident that there was a significant increase in the number of outpatient visits and hospital discharges of patients with dysentery from 1968 to 1969. The difference was more marked with respect to hospital discharges for patients with dysentery, whether they were discharged alive or dead. On the other hand, there was not a significant increase in the number of outpatients seen for enteritis and other types of nondysenteric diarrheal diseases.

TABLE 2

Deaths caused by dysentery per 100,000, by altitude in 142 municipalities, Guatemala, September 1968 through August 1969

Altitude (*feet*)	Population	Deaths	Rate
Less than 1,000	373,276	1,247	334.1
1,000–2,999	617,595	1,720	278.5
3,000–4,999	518,322	1,240	239.2
5,000 or more	747,452	1,423	190.4
Total	2,256,645	5,630	249.5

TABLE 3

*Dysentery patients attended in government-supported Guatemalan hospitals, 1968–69**

Diagnosis	1968	1969
Outpatients		
Dysentery	7,742	11,816
Other†	65,142	58,153
Hospital discharges		
Alive		
Dysentery	1,596	5,803
Other†	6,851	8,238
Dead		
Dysentery	68	488
Other†	516	777
Total		
Dysentery	1,664	6,291
Other†	9,031	9,015

* From Official Register. Data for Roosevelt Hospital not available.

† Includes enteritis and all other nondysenteric diarrheal diseases.

Serological Findings

A total of 920 serum specimens were collected from 21 affected communities in 12 of the departments of Guatemala. Passive hemagglutinating antibodies to the Shiga bacillus at a dilution of 1:40 or greater were detected in 285 (30.9%). The frequency of significant antibody titers to the Shiga bacillus ranged from 5.5 to 66.7% in the various communities studied in 1969. These results contrast with findings in a nationwide serosurvey conducted 3 years before the appearance of the outbreak (Fig. 3). In that survey, only 1.8% of 2,884 persons examined had antibodies to *S. dysenteriae* type 1 at a titer of 1:40 or greater. In the preepidemic survey, 11 villages had no demonstrable reactors, and 28 had one or more reactors. The prevalence of reactors varied from 0.8 to 9.5%. Prevalence rates were evenly distributed in lowland and highland communities.

DISCUSSION

Field studies conducted in Guatemala have shown that shigellosis is highly endemic in that country, contributing significantly to morbidity and mortality, particularly in childhood.[8-10] After its introduction into a community, a new *Shigella* serotype likely causes infection and illness in the accumulated susceptibles, especially in children. As the number of immunes increases, the outbreak eventually subsides. The magnitude of the outbreak depends in part on the virulence and dosage of the particular strain, on the characteristics of the host (nutritional state, immunity), and on environmental factors (climate, sanitation, and sociocultural pattern). The emergence of a particularly virulent serotype of *S. dysenteriae* type 1 and its dissemination in a highly susceptible population living in an unfavorable sanitary environment provided the conditions required for the most severe epidemic of dysentery since the occurrence of a countrywide epidemic in El Salvador in 1915.[11]

Foci of the Shiga bacillus were known to exist in the area.[1] An extensive serological survey carried out in Central America more than 3 years before the epidemic further attests to the presence of a long-standing and widely disseminated endemic focus. This was revealed by the finding of significant antibody titers to the Shiga bacillus in a small percentage of individuals in more than two-thirds of 39 communities surveyed. This endemic focus probably extended into other Central American countries and into México.

The serological test used in this investigation measures antibody in the IgM component. Detectable titers persist not more than 1 year; their demonstration strongly suggests current or recent infection. Thus, we may conclude that the level of transmission was low throughout the country for years before the epidemic.

The epidemic began in southwestern Guatemala late in 1968 and spread from west to east and then to the north. By the end of the third quarter of 1969, the whole country was affected. Whether the epidemic focus extended into southern México is unclear. However, many of the earliest community outbreaks adjoined or were near the Mexican border.

One characteristic of the outbreak was its high velocity of spread, covering practically all of Guatemala and extending to neighboring countries within 6 months. This could have been due to enhanced virulence of the organism and abundant shedding of the agent. Contamination of the environment was probably effected from persons with active disease, since they are known to be more efficient excretors than are convalescent and asymptomatic carriers.[12] Dissemination was undoubtedly facilitated by the excessive migration of whole families from the highlands to the lowlands as laborers for the coffee harvest at the beginning and end of each year. The high virulence of the Shiga bacillus, due in part to its enterotoxin[13] and the prevailing low level of immunity to this serotype accounted for the numerous cases.

Another important feature of the epidemic was the high case-fatality ratio for untreated patients: 8.4% of village patients[1,2] and 10 to 15% of those hospitalized with acute illness. The application of proper therapy, including such drugs as penicillin and ampicillin, was a major factor in bringing the case-fatality ratio down to 1% in treatment centers during 1970.

Obviously, not all dysentery deaths could be attributed to the Shiga bacillus. However, clinical, bacteriological, serological, and epidemiological evidence obtained in all individual village outbreaks studied in 1969 and 1970 indicates that most dysentery deaths were associated with infection with the Shiga bacillus.

Mortality was important in all age groups but particularly in preschool children and older per-

sons, probably because they tolerate poorly any enteric infection. Malnutrition is more prevalent in these age groups, and the effects of dehydration are more serious. The high attack rates for all age groups confirm observations made in other studies in this epidemic of a highly susceptible population.[2] Males of all ages were affected more often than females, a trend commonly seen in the 1st year of cholera epidemics, another enteric bacterial infection with pandemic propensity.[14] The magnitude and timing of the epidemic were reflected in the increased hospital admissions and subsequent deaths due to dysentery in 1969, which exceeded expectation based on data of the preceding year. Unlike the endemic situation, a large portion of the admissions and deaths were in adults.

Higher death rates were observed in the lowlands than in the highlands. This was probably due to host-related factors, i.e., greater fluid and electrolyte losses and the existence of widespread malnutrition, both of which are most prominent in the tropical lowlands.

Regarding the prognosis of this epidemic, there is every indication that the focus, that by mid-1970 included México and six Central American countries, will continue to expand. The danger of spread is especially serious because of the combination of a highly susceptible population and poor environmental sanitation, which together provide a fertile soil for dissemination. Because no substantive control measures are available that can be practically imposed, the epidemic will persist and expand until the number of susceptible persons decreases to below the critical level needed for epidemic transmission. At that point, the disease will become endemic again, perhaps to reappear at a later date and perpetuate the epidemic cycle. The need for a program to correct the underlying sanitary deficiencies and for an effective immunizing agent to disrupt this cycle are apparent.

SUMMARY

The epidemic of Shiga dysentery in Guatemala derived from a long-standing, countrywide endemic focus. Outbreaks began in 1968 or early in 1969 in the southwestern part of the country adjoining México. The epidemic extended rapidly eastward to the border of El Salvador and then to the north, encompassing the entire country during 1969. The mortality rate was high for all age groups, particularly children and the aged, indicating a virulent organism in a highly susceptible population. Males in all age groups were more severely affected. Death rates were highest in the more densely populated lowlands, where malnutrition was more common and climatic conditions were more severe.

ACKNOWLEDGMENTS

The authors express sincere appreciation to all persons who helped with the collection and analysis of data, particularly Armando Cáceres, Jaime Solórzano, Juan J. Erdemenger, Rafael Godínez, Constantino Albertazzi, Eduardo Arellano, and Marion Landsberger.

PUBLICATIONS CITED

1. Mata, L. J., Gangarosa, E. J., Cáceres, A., Perera, D. R., and Mejicanos, M. L., 1970. Epidemic Shiga bacillus dysentery in Central America. I. Etiologic investigations in Guatemala, 1969. *J. Infect. Dis., 122:* 170–180.
2. Gangarosa, E. J., Perera, D., Mata, L. J., Mendizábal-Morris, C. A., Guzmán, G., and Reller, L. B., 1970. Epidemic Shiga bacillus dysentery in Central America. II. Epidemiologic studies in 1969. *J. Infect. Dis., 122:* 181–190.
3. Reller, L. B., Navarro Rivas, E., Masferrer, R., Bloch, M., and Gangarosa, E. J., 1971. Epidemic Shiga bacillus dysentery in Central America. Evolution of the outbreak in El Salvador, 1969–1970. *Am. J. Trop. Med. & Hyg., 20:* 934–940.
4. Béhar, M., Ascoli, W., and Scrimshaw, N. S., 1958. An investigation into the causes of death in children in four rural communities in Guatemala. *Bull. World Health Organ., 19:* 1093–1102.
5. Neter, E., Westphal, O., Luderitz, O., and Gorzynski, E., 1956. The bacterial hemagglutination test for the demonstration of antibodies to enterobacteriaceae. *Ann. N. Y. Acad. Sci., 66:* 141–156.
6. Young, V. M., Gillem, H. C., Massey, E. D., and Baker, H. J., 1960. A study on the detection and specificity of antibodies to *Shigella flexneri* types using preserved polysaccharide-sensitized human erythrocytes. *Am. J. Pub. Health, 50:* 1866–1872.
7. Cáceres, A., and Mata, L. J., 1970. Hemaglutinación indirecta para la investigación de anticuerpos a enterobacteriáceas. *Rev. Lat. Am. Microbiol.* (México), *12:* 137–144.
8. Beck, M. D., Muñóz, J. A., and Scrimshaw, N. S., 1957. Studies on diarrheal diseases in Central America. I. Preliminary findings on cultural surveys of normal population groups in Guatemala. *Am. J. Trop. Med. & Hyg., 6:* 62–71.

9. Gordon, J. E., Ascoli, W., Pierce, V. Guzmán, M. A., and Mata, L. J., 1965. Studies of diarrheal disease in Central America. VI. An epidemic of diarrhea in a Guatemalan highland village, with a component due to *Shigella dysenteriae* type 1. *Am. J. Trop. Med. & Hyg., 14:* 404–411.
10. Mata, L. J., Fernández, R., and Urrutia, J. J., 1969. Infección del intestino por bacterias enteropatógenas en niños de una aldea rural de Guatemala, durante los primeros tres años de vida. *Rev. Lat. Am. Microbiol., 11:* 102–109.
11. Rivera, A. A., 1916. Apuntes sobre la disentería bacilar epidémica de 1915 en la República de El Salvador, Tesis, Facultad de Medicina y Cirujía, Universidad Nacional, 115 pp.
12. Dale, D. C., and Mata, L. J., 1968. Studies of diarrheal disease in Central America. XI. Intestinal bacterial flora in malnourished children with shigellosis. *Am. J. Trop. Med. & Hyg., 17:* 397–403.
13. Keusch, G. T., Mata, L. J., and Grady, F., 1970. *Shigella* enterotoxin: isolation and characterization. *Bacteriol. Proc.* 70th Meet., p. 99, April 26–May 1, 1970.
14. Dizon, J. J., Alvero, M. G., Joseph, P. R., Tamayo, J. F., Mosley, W. H., and Henderson, D. A., 1965. Studies of cholera El Tor in the Philippines. I. Characteristics of cholera El Tor in Negros Occidental Province, November 1961 to September 1962. *Bull. World Health Organ., 33:* 627–636.