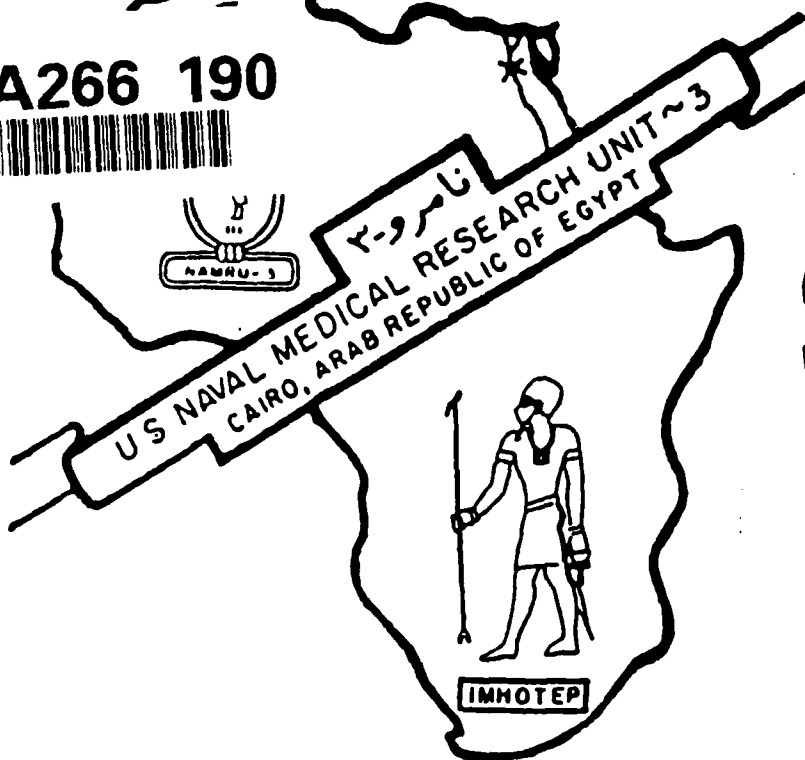


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BY

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## The Endemic Infectious Diseases of Somalia

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*Haddaan la kala roonan roob ma do'o. [If no one is merciful, the rain will not come.]*

—Somali proverb

Somalia has recently been suffering death rates that are among the highest ever recorded in a famine-affected population [1]. The combination of some of the greatest poverty in the world, a severe drought, tribal fighting, and political anarchy have led to this desperate situation. In December 1992, the United Nations initiated Operation Restore Hope (ORH) in Somalia. With a mission to restore order and allow safe delivery of intensive relief aid, nearly 26,000 troops will be deployed in the largest military humanitarian relief effort conducted in recent history. The participants in ORH are from 20 countries; most heavily represented is the United States.

Endemic infectious diseases of the region—especially diarrheal disease, measles, malaria, and acute respiratory illnesses—have caused most of the mortality among the malnourished Somali people. The participants in ORH as well as members of numerous other relief agencies will be exposed daily to the pathogens causing these endemic diseases. They are at risk not only of acquiring acute diseases in Somalia but also of importing these diseases back into their countries of origin. In addition, increasing numbers of Somalis will be seeking asylum or refugee status in countries outside the endemic area. The United Nations has estimated that one of every six Somalis is now a refugee, with 100,000 in European countries and 65,000 in Yemen. The rapid increase in these numbers is illustrated by the local Somali population in San Diego, California, which grew from 30 in 1990 to more than 1,000 by the end of 1992.

Because information on the endemic diseases of this area is scarce, often anecdotal or unpublished, and scattered

through journals with minimal circulation in the United States, it seems valuable to provide an in-depth review of the most significant of these conditions. When considering the current state of medical knowledge of Somalia, we would do well to remember what Cahill said when he surveyed the situation in 1971: "In a review of [maps showing] the world distribution of diseases, Somalia had the dubious distinction of having 'no data available' for 22 of the 23 maps, a record unequalled by any other country in Africa" [2]. This situation certainly holds for the last 2 years of political anarchy.

In this review we will focus primarily on the endemic infectious diseases of Somalia. However, because the nomadic population commonly migrates across nearby international borders with adjoining countries and may introduce diseases from these countries into Somalia, we will also mention the endemic diseases of other countries in the Horn of Africa and make extrapolations based on these data. For the purposes of this paper, we will define the Horn of Africa as including Somalia, Djibouti (former French Somaliland), the Ogaden region of Ethiopia, and the northeastern region of Kenya. Currently, an estimated 1 million ethnic Somalis live in Ethiopia, Kenya, and Djibouti.

### Background Information

Somalia occupies the easternmost tip of the continent of Africa and is bordered by the Gulf of Aden to the north and the Indian Ocean to the east and south (figure 1). The country has a total area of 246,000 square miles (about the same as that of Texas). The terrain is mostly flat in central and southern Somalia, with rugged highlands in the north. The climate is hot year-round, with irregular rainfall and droughts that may recur in 2 of every 5 years [3]. There are four seasons. From January to March, the weather is hot and dry; this is considered the harshest time of the year for nomadic groups. The major rainy season—from April through May—is erratic and undependable. Another dry season follows in June through September, and the year ends with light scattered rains from October through December.

Somalia has a population estimated at 7,700,000. Around 30% of Somalis are nomadic shepherds who migrate during the rainy season across many hundreds of miles, often crossing international borders to lead their flocks to their traditional pastures. When the dry season returns, they migrate

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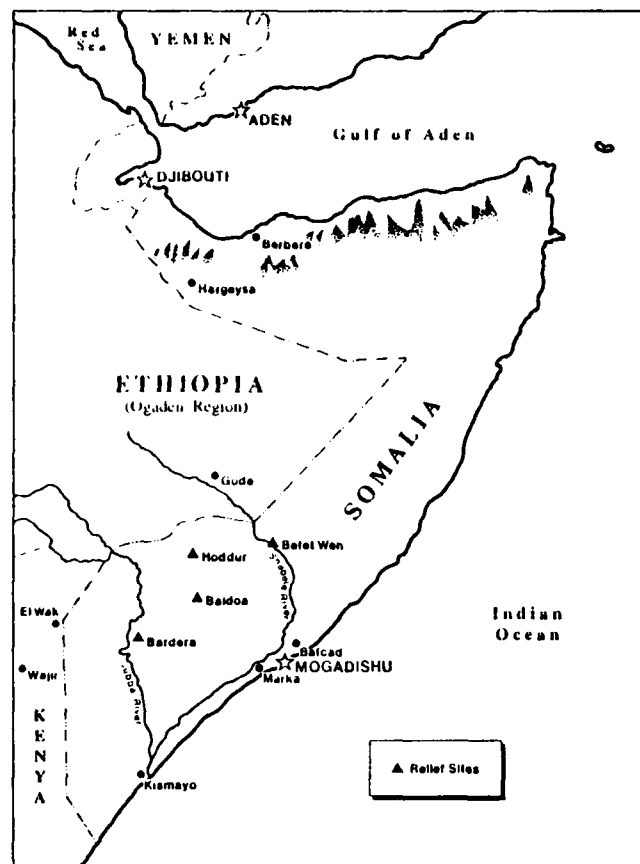


Figure 1. The Horn of Africa.

back to their home wells. Even during good years, caloric intake (obtained almost exclusively from camel's milk among men) may drop to 50% of the daily requirement during the dry season [3]. One of the major reasons that the last case of smallpox in the world occurred in Somalia was the continual movement of the nomads and the reintroduction of the disease into settled populations. This case was transmitted from a 6-year-old nomadic girl to a resident of Marka in October 1977 [4].

Another 20% of Somalis are seminomadic, spending a portion of the year farming, and about 30% are settled farmers, who are also subject to the capriciousness of the rains in this semiarid environment. Under optimal conditions, the dry-land farmers between the Jubba and Shebele rivers can expect at least 2 years without crops, one with a poor crop, and two with good crops during a 5-year period. Those in the floodplains of the Shebele River can expect three good crops, one fair crop, and one poor crop. However, during droughts, when the river fails to flood, they are left with little alternative other than to return to the nomadic life.

The country's population is 98% ethnic Somali. The Somalis are a tall slender race with aquiline features whose language is related to the Galla and Afar of Ethiopia. Sir

Richard F. Burton, the noted explorer and linguist who explored Somalia in 1854, aptly described the Somalis as "a fierce race of Republicans, the Irish of Africa," referring to their sense of humor and their great respect for poetry, oral traditions, age, and religion [5]. The love of oral traditions is not surprising for a people whose language had no written form until 1972. The Somalis possess a strong cultural unity, and at least 90% are Sunni Muslims. However, deep divisions exist among competing clan-families, clans, and lineages. The history of Somalia is a long and repetitive story of conflicts, cleavages, and temporary alliances among these groups. This intense rivalry among lineage groups is best described by a Somali saying that describes their concept of loyalty as "with the tribe against the state, with the clan against the tribe, with my family against the clan, and with my own brother against the rest" [5a]. Most of the current anarchy is due to clan rivalries.

Somalia achieved its independence on 1 July 1960, with the uniting of the Trust Territory of Somalia (formerly Italian Somalia) in the south and the British Protectorate of Somaliland in the north. (The latter has recently declared independence, but this status has not yet been recognized by other nations.) Major General Mohammed Siad Barre seized power in a bloodless coup in 1969 and ruled the country until he was overthrown in January 1991. His overthrow marked the beginning of the period of political anarchy and clan rivalry that continues today.

Before the onset of this interval of social disruption, the health system in Somalia was poorly funded, with only 1.6% of the development budget allocated for health services. The average life expectancy was 47 years, and the infant mortality was 145 per 1,000 live births. According to the 1980-1985 National Health Plan, there were 70 district hospitals outside the capital city of Mogadishu, and 50% of these hospitals were staffed only by nurses [6]. Of the larger regional hospitals, most were staffed by a single physician. Of the fewer than 400 doctors in the entire country, 60% practiced in Mogadishu, where only 20% of the population resided. Preventive-medicine efforts were almost nonexistent: only 30% of the nonurban population had access to safe water, and 60% of preschool children were malnourished even in nondrought periods.

Somalia has almost no natural resources and thus faces major development problems. There are no railroads, and there are only 3,500 miles of all-weather roads. The per capita annual income was estimated in 1985 to be \$300, with a 30% annual inflation rate. Fewer than 1% of Somalis own automobiles.

#### Traditional Practices

Certain traditional Somali practices may result in medical complications that need to be treated both among the Somali populace and among refugees living in other countries. Fore-

most among these practices is female circumcision, of which there are three types: infibulation, clitoridectomy, and Sunna (excision of the prepuce of the clitoris). Currently, 100% of Somali women undergo some form of circumcision, and 88% undergo the most severe form, infibulation, which involves total excision of the clitoris and labia minora with fusion of the inner aspects of the labia majora. The prevalence of the custom led anthropologists to refer to the area as "the land of the sewn women" [7]. When interviewed in a recent survey, most Somali women justified the procedure on religious grounds, although the subject is not mentioned in the Koran. All of the women wanted their daughters to be circumcised, and those views did not vary with educational level or socioeconomic status [8]. The World Health Organization (WHO) has identified female circumcision as the most dangerous traditional custom—in terms of health—that exists today [9]. In Africa, there are an estimated 80 million circumcised women [8].

Among the nomads, the procedure is a private family ceremony performed by the women; no men are allowed to be present. The 4- to 8-year-old girls are held by female relatives and friends while the clitoris and labia are excised without anesthetic. The wound is covered with myrrh, a resinous product of a local tree. Thorns from the acacia bush are used to close the wound, and the legs are bound from hip to ankle for 2 weeks. The end result is a fleshy closure that extends from the pubis to the anus with a small inferior opening.

A number of complications of this procedure have been reported. During one 12-month period, 118 women were admitted to Benadir Maternity Hospital in Mogadishu with complications of female circumcision [10]. Among these women, 47% had dermoid cysts (diameter, 2–15 cm) at the site of the clitoral excision, 36% had vaginal stenosis, 4% had associated hematocolpometra, and 9% had extensive abscess formation at the excision site. These complications resulted in a total of 1,967 occupied bed-days. The numerous immediate complications of the procedure, including local infection, hemorrhage, and urinary retention, were not encompassed by the figures reported, nor were obstetric complications, which include a requirement of two episiotomies for every delivery: one anterior incision splitting the infibulation scar and one mediolateral incision. The incidence of perineal tears is also much higher among circumcised women. In fact, repair of these tears is the most frequently performed operation at Benadir Maternity Hospital [10].

Elimination of female circumcision has proven difficult. There is no law in Somalia that would prohibit the procedure, even in public health facilities. In the Sudan, a law forbidding infibulation resulted in the subjection of many young girls to hastily performed procedures before the law was enacted. Persistent educational efforts will be required to alter the strong traditional beliefs that perpetuate female circumcision, an ancient practice that currently is almost universally accepted by both male and female Somalis.

### Traditional Healers

In a country like Somalia that has a poorly developed medical system, traditional healers assume an important position in the provision of health care. These traditional practitioners are highly respected and most commonly prescribe herbal medicines, religious acts, or traditional dancing as treatments. The Somali Ministry of Health has granted certificates to some traditional healers, giving them a certain legitimacy and recognition within the national health care system. It is interesting that the level of satisfaction with the health care provided may be higher among Somalis than among patients in the United States. Only 12% of surveyed Somali patients considered their health care expensive, only 3% had adverse reactions to the prescribed treatments, and no patient felt that these treatments were "unscientific" [11].

In Somalia and Djibouti, traditional treatment for long-lasting upper respiratory symptoms in children commonly includes uvulectomy, epiglottectomy, and removal of a deciduous tooth, usually a molar. Local abscesses and hemorrhage are the most frequent complications of these procedures. Another traditional maneuver that is widely used in both children and adults is skin burning for pain relief. Scars of skin burns are extremely common and reveal to physicians part of the medical history of their patients. Other traditional practices include the chewing of sticks for oral hygiene and the use of a wide variety of plants, including the popular purgative seeds of *Croton macrostachys*.

### Diarrheal Disease

Diarrheal disease is likely to be the most common medical problem among the native Somali population as well as the foreign military personnel and civilian humanitarians. As in many developing countries, diarrheal disease in Somalia is a leading cause of morbidity and mortality among children under 5 years of age [12] and among displaced persons [13]. In famine-affected populations, it has been responsible for 28%–40% of all deaths. Diarrhea was the cause of 35% of all deaths among Somali refugees in the Liboi camp in Kenya in 1991 [14].

For military personnel involved in foreign operations, diarrhea has always been a serious cause of morbidity. In World War II, attack rates of diarrhea and dysentery in the Middle East were 497 cases per 1,000 men during the month of August 1942. Especially hard hit were new arrivals to the area, whose illnesses sometimes rendered their units incapable of effective action. During Operation Desert Shield/Storm (ODSS), large outbreaks of diarrhea occurred, with attack rates exceeding 10% of the force strength each week in some units. It was estimated that during the first month of ODSS, at least 50% of all troops were affected. These outbreaks were directly linked to the ingestion of unapproved lettuce procured from areas in which human waste is used as

fertilizer. The epidemic rates began to decrease only after the use of these food sources was stopped on 25 September 1990 [15].

Diarrheal disease may cause even more morbidity among the civilian relief workers and journalists than among the military, as the former groups are more likely to use local food, water, and sanitation facilities.

#### Etiology and Antimicrobial Sensitivity Patterns in Diarrheal Disease

As would be expected, a broad spectrum of pathogens are responsible for diarrhea in Somalia. In a large-scale study conducted in Mogadishu in 1983-1984, at least one enteric pathogenic species was found in 61% of hospitalized children with severe diarrheal disease [12]. The etiologic agent most frequently identified was rotavirus (25% of children); the other pathogens detected were enterotoxigenic *Escherichia coli* (ETEC, 11%), *Shigella* species (9%), *Giardia lamblia* trophozoites (8%), *Campylobacter jejuni* (8%), and *Vibrio cholerae* non-O1 (6%). In a countrywide survey conducted in nearby Djibouti in February 1989, ETEC was the most common isolate from diarrheal stools (11%); also frequently recovered were enteroadherent *E. coli* (EAEC, 10.6%) and enteropathogenic *E. coli* (EPEC, 7.7%). *Shigella* species, *C. jejuni*-*C. coli*, *Salmonella* species, and *Aeromonas* species were found in 7.7%, 3.3%, 2.9%, and 3.3% of stool samples, respectively [16].

Strains of ETEC in Somalia have been shown to be different from those in Europe [17]; thus relief workers and troops will most likely be immunologically naive with respect to the local strains. This situation made ETEC the most common cause of diarrhea during ODSS (28% of all cases) [18]. In Somalia in the mid-1980s, the O4 strain of ETEC played an important role in the local epidemiology of diarrheal disease [19]. In a recent study from Djibouti [16], isolates of ETEC, EAEC, and EPEC were all highly resistant to commonly used antimicrobial agents; resistance to ampicillin, doxycycline, sulfisoxazole, tetracycline, and trimethoprim-sulfamethoxazole (TMP-SMZ) was found. However, all strains were sensitive to the quinolone norfloxacin, and all except *Campylobacter* species were sensitive to ceftriaxone.

Bacillary dysentery caused by *Shigella* species is also common in Somalia and can be expected to be a serious problem in displaced populations and among relief workers and journalists. During ODSS, *Shigella* was almost as common as ETEC, being isolated from 26% of patients with diarrhea [18]. Severe epidemics due to *Shigella dysenteriae* type 1 were reported in Somalia in the 1960s [20] and 1970s [21]. A strong seasonal prevalence of shigellosis is noted early in the spring rainy season, with recurrent annual outbreaks. During the dry season in January and February, fewer than 5% of 63 isolates from individuals with acute diarrhea in Mogadishu were *Shigella*. This figure increased to more than 15% of 144

isolates during the rainy season in May and June. There is also a variation from year to year in the cause of outbreaks of dysentery. In the rainy season of 1983, 18 (31%) of 59 isolates were *S. dysenteriae* A1; in the following rainy-season outbreak, 23 (42%) of 55 isolates were *Shigella flexneri* B4. In 1984, only 11% of isolates were *S. dysenteriae* A1 (A. Bourgeois, personal communication).

As in the Middle East, *Shigella* isolates in Somalia are exhibiting increasing resistance. A strain isolated in 1976 was resistant to ampicillin, chloramphenicol, streptomycin, sulfonamides, and tetracyclines. The eventual spread of this strain to Zaire and other Central African countries was demonstrated by the presence of an identical resistance plasmid [22, 23]. The strain was later noted to have acquired an additional plasmid conferring resistance to trimethoprim and hence to TMP-SMZ [24].

*Shigella* isolates from Mogadishu in 1984 were resistant to ampicillin (87%), tetracycline (96%), and TMP-SMZ (74%). No resistance to ciprofloxacin was detected (A. Bourgeois and E. C. Oldfield, unpublished data).

By 1989, a high level of shigella resistance to both ampicillin (69%) and TMP-SMZ (69%) was reported in nearby Djibouti [16]. All isolates remained sensitive to norfloxacin and ceftriaxone. *Shigella* species isolated during the 1991-1992 winter season at the French Military Hospital in Djibouti were highly resistant to amoxicillin, amoxicillin/clavulanic acid, tetracycline, and TMP-SMZ but retained full sensitivity to ciprofloxacin (R. Bercion, personal communication).

Early reports from ORH suggest that dysentery has been a major cause of morbidity among both military personnel and civilian humanitarians as well as among reporters (J. Burans and M. Wallace, personal communication). The most common isolates have been *S. flexneri* and *Shigella boydii*; in addition, *S. dysenteriae* was isolated from a French soldier who had been in Djibouti. A number of isolates have been resistant to all commonly used antibiotics except the quinolones; such resistance has been especially frequent in *S. flexneri*.

*Campylobacter* and *Salmonella* species have not been major causes of diarrheal disease in Somalia. In Djibouti, these species represented 3.3% and 2.9% of isolates, respectively, during one study in 1989; the *Salmonella* isolates remained relatively sensitive to antibiotics, with no resistance to chloramphenicol and only 11% and 22% of strains resistant to TMP-SMZ and ampicillin, respectively [16]. *Aeromonas* species can be a significant cause of diarrhea, especially in the rainy season, when these organisms are isolated from up to 20% of all patients with acute diarrhea who are seen at primary health units and hospitals in Mogadishu. During the dry season, species of *Aeromonas*, like those of *Shigella*, are less common, with an isolation rate of <5% (A. Bourgeois, personal communication).

Cholera is endemic in the Horn of Africa [25]. The seventh cholera pandemic, caused by *V. cholerae* El Tor,

reached this region in the early 1970s and was most prevalent in Somalia. No recrudescence of the disease was observed until March 1985, when a cholera epidemic was reported in a refugee camp in the Hiran region, north of Mogadishu; this epidemic eventually spread to the rest of the country. Numerous strains of *V. cholerae* O1, all of the E1 Tor biotype, were isolated from patients, contacts, and water sources. Only the isolates from the Hiran region belonged to the Inaba serotype, whereas the strains involved in almost all of the other cholera outbreaks in Somalia were of the Ogawa serotype. The latter strains were resistant to ampicillin, kanamycin, streptomycin, sulfonamides, and tetracycline but were susceptible to chloramphenicol, nalidixic acid, and trimethoprim [26]. A source of concern were the cases of cholera reported in May 1992 in neighboring Kenya [27] and in July 1992 among Somali refugees in Yemen [28].

### Prevention of Diarrheal Disease

Major efforts should be made to develop diarrhea control programs both in the military and in camps for displaced persons. In refugee populations, the provision of an adequate amount of water is more beneficial overall (and also easier) than the provision of smaller amounts of microbiologically pure water [14]. This is particularly true with regard to the prevention of shigellosis. The minimal volume of water recommended is 20 l/d per person. Convenience as well as availability must be taken into account. If the water source is located >150 meters from an individual's dwelling, its use by that individual for hygienic purposes will be markedly decreased. This decrease will be especially pronounced in famine-affected populations. Sanitation in refugee camps is critical. Family or personal latrines are preferable to larger facilities. Soap should be made readily available for personal hygiene.

For military personnel serving in ORH, preventive measures will be strongly emphasized, especially in light of the aforementioned epidemic early in ODSS. The preventive-medicine officer for the U.S. Marines was quoted in the *Los Angeles Times* (14 December 1992) as saying: "We are launching a world-class public health effort with no holds barred." The provision of safe water and packaged food such as MREs (meals, ready to eat), effective sanitation, disease surveillance, and strong support from field commanders will be stressed in ORH.

### Treatment of Diarrheal Diseases

The mainstay of the treatment of diarrhea is rehydration. In all cases except those that are most severe and those that are associated with vomiting or altered mental status, oral rehydration is preferred. Oral rehydration has proven effective for diarrhea regardless of the etiologic agent or the age of the patient. For initial rehydration, the American Academy of Pediatrics recommends solutions with 70–90 mEq of so-

dium/L; WHO oral rehydration solution packets (available in the United States from Jianas Brothers, Kansas City, MO) and Rehydrate (Ross Laboratories, Columbus, OH) have 90 and 75 mEq of sodium/L, respectively [29]. For maintenance, the American Academy of Pediatrics recommends fluids with 40–65 mEq of sodium/L; Pedialyte (Ross) and Ricelyte (Mead-Johnson, Evansville, IN) have 45 and 50 mEq of sodium/L, respectively. If fluids with >60 mEq/L are used for maintenance, the patient should receive other fluids that are low in sodium to prevent hypernatremia.

Although MREs cannot be considered masterpieces of the culinary arts, they effectively provide the salts and sucrose necessary to make oral rehydration solutions in the field. MREs contain a salt packet with 4.0 g of NaCl and a beverage base powder with 28 g of sugar. The MRE cocoa beverage, in addition to 30 g of sugar, contains 0.85 g of KCl equivalent [30]. The combination of one salt packet, one beverage base powder, and two cocoa beverage packets in a canteen full of water (1 qt) provides a balanced salt solution similar to the WHO oral rehydration solution, except that it does not contain trisodium citrate, which replaced sodium bicarbonate in the WHO solution to provide a longer shelf life.

For cases of diarrhea requiring antibiotic therapy, empirical use of a quinolone is appropriate. The standard regimen is ciprofloxacin (500 mg twice daily) or norfloxacin (400 mg twice daily) administered for 3–5 days. A number of studies [31–35], including one conducted in Somalia [35], have shown the efficacy of single-dose therapy for shigellosis. On the basis of these studies, a reasonable alternative is to treat acute diarrheal disease with a single dose of ciprofloxacin (750–1,000 mg) or norfloxacin (800 mg). One exception to this rule involves situations in which the incidence of disease due to *S. dysenteriae* type 1 is high, as it was in Mogadishu in 1983. Against this particular species, single-dose therapy fails significantly more often than a full 5-day course [36]. When diarrhea persists, an evaluation for giardiasis, amebic dysentery, and antibiotic-associated colitis should be undertaken. If adequate diagnostic facilities are not available, empirical therapy with metronidazole should be considered.

Some physicians prefer a combination of loperamide plus an effective antimicrobial agent. In a study by Ericsson et al., the administration of TMP-SMZ and loperamide reduced the duration of diarrhea to just 1 hour from the 28 hours documented among patients given TMP-SMZ alone [37]. More recent data have shown that loperamide—when used in combination with an effective antibiotic—not only is safe in the treatment of shigellosis but also decreases the total number of unformed stools and the duration of diarrhea [38].

### Malaria

Malaria is a potentially serious problem for the malnourished Somalis and for persons involved in the humanitarian

relief effort—both civilian and military. The impact of malaria on malnourished populations has been related to the local endemicity as well as to the level of immunity in the exposed group. In a recent study in Malawi, where malaria is highly endemic, malaria was responsible for 18% of all deaths among refugees and for 25% of deaths among children <5 years of age [14]. In the Gedo region of Somalia (near the borders with Kenya and Ethiopia), where there is lower endemicity, 2% of all deaths among Ethiopian refugees were attributed to malaria [14].

In Somalia, malaria is considered moderately endemic, with year-round transmission whose frequency increases during each rainy season. All areas are considered at risk with the possible exception of Mogadishu, which—as of 1986, at least—was thought to be risk free. More than 90% of cases are due to *Plasmodium falciparum*, only 5% to *Plasmodium malariae*, and only 3% to *Plasmodium vivax*. The principal vectors of malaria are the *Anopheles gambiae* complex and *Anopheles funestus*; the former consists of domestic mosquitoes that breed in small pools, puddles, and man-made containers, and the latter species breeds in swamps, rivers, and on the margins of streams. Both species feed and rest inside houses and other types of dwellings.

Three factors, however, arouse concern that the impact of malaria during the current crisis may be much more serious than expected. First, the endemicity of malaria is apparently increasing throughout the Horn of Africa, and focal epidemics are being reported with increased frequency. For example, Djibouti was considered free of malaria until 1973, when scattered outbreaks were reported in its southern portion [39]. Since 1988, regular seasonal epidemics of *P. falciparum* malaria (starting in November and peaking in April or May) have become important [40]. Beginning in 1990, cases of malaria began to be documented throughout the year, even during the summer dry season. Population migrations have clearly contributed to this increasing prevalence [41]. An epidemic of malaria reported in December 1988 from the area of southern Somalia around Balad, 30 miles north of Mogadishu, was followed by an outbreak of what was probably malaria (with several hundred deaths) in April 1989 in the Berbera area of northern Somalia [42]. Increasing endemicity of malaria is also suggested by an increase in the number of officially reported cases from about 2,000 in 1984 to a high of 16,000 in 1988.

The second factor is the increasing prevalence of chloroquine-resistant *P. falciparum* in the Horn of Africa. There are three types of resistance to chloroquine. RI denotes a level of resistance in which there is clearance of the asexual malarial parasites from the blood smear within 7 days of beginning a standard therapeutic regimen of chloroquine (25 mg of base/kg over 3 days); this initial clearance is followed by a recrudescence of parasitemia within 28 days. An RII response is defined as a marked reduction (>75%) but persistence of parasitemia on day 7 after the beginning of therapy. An RIII response occurs when there is a <25% decrease or even an

increase in the level of parasitemia within the first 48 hours of treatment. Reports of chloroquine-resistant strains in neighboring Kenya began to appear in the 1970s [43]. As recently as 1986, a study in the Mogadishu area showed that the standard therapeutic dose of chloroquine resulted in the clearance of parasitemia by day 3 in all cases [44]. However, in similar studies conducted in 1987, 1988, and 1989, RII/III chloroquine resistance was noted and confirmed by in vitro tests in 17%, 19%, and 30% of cases, respectively. Fortunately, sensitivity testing revealed no evidence of resistance to mefloquine [45]. In the previously mentioned epidemic in Balad, isolates from 31 of 36 patients given a standard chloroquine regimen showed evidence of resistance (5 at RI and 26 at RII/III). High degrees of in vitro resistance were confirmed in 33 of 37 isolates. The presence of detectable blood levels of chloroquine before treatment in 78% of patients confirms the lack of value of chloroquine prophylaxis in this area. All isolates retained sensitivity in vitro to pyrimethamine/sulfadoxine. Spread of chloroquine-resistant *P. falciparum* throughout the Horn of Africa has been confirmed by reports from areas of Ethiopia along the border with Kenya and Somalia [46] as well as by a 1990 in vivo study from Djibouti (E. Fox, personal communication) in which RII/III resistance was noted.

The third factor is that the above-average precipitation in the fall and early winter of 1992 (the second and usually minor rainy season) may enhance breeding of the anopheline vectors—and, consequently, the impact of malaria. Although the rain is welcome in that it may help alleviate the ongoing drought, it may contribute to an increase not only in malaria but also in other vector-borne diseases as well as diarrhea.

The acute impact of malaria on military and civilian humanitarian workers should be minimal if there is strict adherence to antimalaria regimens, especially the terminal 4 weeks of prophylaxis after departure from the endemic area. However, a high level of discipline is often difficult to achieve under field conditions. An estimated 70% of returning troops failed to complete their recommended terminal prophylaxis during the Vietnam War [47]. Because most U.S. troops would be expected to go on leave soon after returning from Somalia, there is a real chance that cases of chloroquine-resistant *P. falciparum* malaria will be seen throughout the United States.

Physicians caring for Somalis who are granted asylum or refugee status in other countries should be aware of the risk of imported malaria. The importation of this disease was recently noted to be a problem in Israel, where more than 1,000 cases were diagnosed in Ethiopian immigrants [48], with more than 20% of cases due to chloroquine-resistant *P. falciparum* [49].

As of mid-January 1993, 30 cases of malaria have been diagnosed among U.S. troops in Somalia; 25 cases have been due to *P. falciparum*, 4 to *P. vivax*, and 1 to *P. malariae*.

Some of these patients had never left the Mogadishu area (M. Wallace, personal communication).

### Treatment of Malaria

As in most parts of Africa, first-line treatment of malaria among Somalis continues to be chloroquine, which is both inexpensive and readily available. The use of chloroquine is restricted to semi-immune people with uncomplicated malaria whose second visit to the clinic within a few days can be ensured. Data from East Africa shows that amodiaquine remains a useful alternative agent in ~75% of cases [50]. The usual regimen for adults is an initial dose of 600 mg of base followed by either 400 mg of base at 24 and 48 hours or 300 mg of base at 6, 24, and 48 hours.

There are three quinine sulfate-based oral regimens that would be preferable to both chloroquine and amodiaquine, especially for the treatment of nonimmune populations. These regimens are quinine sulfate (650 mg three times daily) plus (1) tetracycline (250 mg four times daily for 7 days); (2) pyrimethamine (25 mg) plus sulfadoxine (500 mg) (a combination marketed as Fansidar; Hoffman-LaRoche, Nutley, NJ) three tablets at once on the last day of quinine administration; or (3) clindamycin (900 mg three times daily for 3 days) [51].

One of these quinine-based combinations would be the regimen of choice for military or civilian humanitarian workers who are receiving mefloquine for malaria prophylaxis. It has been suggested that quinine be used with caution by persons taking mefloquine for prophylaxis in light of the potential for induction of cardiac dysrhythmias. Concern about this reaction arose because therapeutic doses of the above drugs used in combination to treat acute cases of malaria were noted to be toxic. In persons whose prophylaxis fails, blood levels of mefloquine will presumably be low because of noncompliance or poor absorption. In this situation, for nonimmune persons, the risk of progressing to complicated malaria would far outweigh the theoretical risk of dysrhythmias due to drug interactions.

Halofantrine, which is effective against more than 90% of *P. falciparum* strains, has recently been introduced into Kenya and Djibouti and may be available in Somalia. The recommended schedule is three doses of 500 mg each, given at 6-hour intervals and repeated after 7 days. Two other drug combinations, pyrimethamine/sulfadoxine and pyrimethamine/sulfalene, are similarly efficacious and can be administered as a single dose [50]. Mefloquine (1,250 mg as a single dose or, as recommended outside the United States, a 750-mg dose followed 6–8 hours later by a 500-mg dose) is highly effective [52]. With large single doses of mefloquine, a number of adverse reactions can be expected, including ataxia, toxic psychoses, seizures, and gastrointestinal disturbances. Mefloquine should not be used in combination with quinine or quinidine for the treatment of clinical malaria.

For complicated cases (those including altered mental status, hypotension, or >5% parasitemia), an initial course of intravenous quinine is the treatment of choice. The recommended dose of quinine hydrochloride is a loading dose of 20 mg of salt/kg in 10 ml. of 5% dextrose/kg over 4 hours followed by 10 mg of salt/kg over 2–4 hours every 8 hours (maximum, 1,800 mg/d) until oral medications can be tolerated [51]. Oral quinine sulfate should be given to complete a 3-day course of therapy in combination with tetracycline, pyrimethamine sulfadoxine, or clindamycin, as previously described.

An alternative therapy for complicated malaria is intravenous quinidine gluconate, with a loading dose of 10 mg/kg (maximum, 600 mg) in normal saline administered slowly over 1 hour and followed by a continuous infusion of 0.02 mg/(kg·min) for a maximum of 3 days. An oral regimen should be instituted as soon as possible [51]. If available, continuous electrocardiographic monitoring should be performed during the infusion of either quinine or quinidine.

### Malaria Prophylaxis

Although ongoing research may ultimately yield an effective vaccine for malaria [53], prophylaxis at present is fraught with problems because of increasing drug resistance. A combination of proguanil (200 mg daily) and chloroquine (600 mg weekly) is considered appropriate for malaria chemoprophylaxis in the indigenous semi-immune population of the Horn of Africa. A number of other alternatives are preferable for prophylaxis in nonimmune persons involved in relief efforts in this area. Mefloquine (250 mg weekly from 1 week before arrival in the endemic area through 4 weeks after departure) is highly effective. Although this drug is usually well tolerated, the full extent of neurological and psychiatric impairment with which it can be associated needs to be closely monitored. Another option is doxycycline (100 mg daily during the individual's stay in the malarious area and for 28 days after departure). Doxycycline is inexpensive and efficacious; however, the need for daily administration will hamper compliance, and the drug may enhance the recipient's photosensitivity [54] and cause pill-induced esophagitis.

The low incidence of malaria due to *P. vivax* and *Plasmodium ovale* makes terminal primaquine prophylaxis for relapse of the latent hepatic phase (hypnozoite) of questionable value. Final recommendations will be based on the observed incidence of disease caused by these species among military personnel.

In addition to medications, the avoidance of mosquito bites is critical to the prevention of malaria. After dusk, outside activities should be minimized and *N,N*-diethylmetatoluamide (DEET)-containing repellents should be applied to exposed skin. The military currently uses a long-acting (12-hour) formulation containing 35% DEET in cream. Per-

methrin-impregnated jackets and mosquito netting should also be used. Interior mosquito control can be accomplished with D-phenothrin 2% insecticide spray. The standard aerosol should be sprayed 10 seconds for every 1,000 cubic feet of space. Space sprays have minimal residual effect. In spaces where reentry of mosquitoes is a problem, interior residual sprays should be used on potential mosquito resting areas. In addition, mosquito breeding sites should be eliminated through application of larvicide. Adult mosquitoes should be controlled by ground ultra-low-volume aerosols and mists. These agents should be used when mosquitoes are active.

After returning from malaria-endemic areas, military personnel and civilian relief workers should not donate blood for 3 years if they have received prophylaxis for malaria or if they have developed an acute case of malaria. If they have received no prophylaxis, they should wait for 6 months before donating blood. These restrictions apply to donors at military blood banks as well as at civilian agencies.

### Measles and Other Vaccine-Preventable Diseases

Somalia has historically been characterized by poor vaccine coverage, especially among nomads, who make up a large proportion of the currently displaced population. Despite the efforts begun in 1979 through the WHO Expanded Programme on Immunization, a survey conducted in Mogadishu and Hargeysa in 1984 found that only 19% of children were fully immunized [55].

This poor vaccination coverage has led to very high rates of vaccine-preventable diseases, even in cities and during times of relative stability. In the National Health Plan for 1980-1985, 180,000 cases of measles, with 9,010 deaths were reported; an estimated 90% of 1-year-old cohorts had been infected. In addition, 159,470 cases of pertussis, with 1,590 deaths, were reported; an estimated 80% of birth cohorts were affected. The populace also suffered a reported 9,970 cases of tetanus neonatorum, with mortality of >90%; an estimated 5% of all live-born infants were affected [6]. In Somalia, tetanus neonatorum is called *todoba-ma-gaarto*—"the disease that kills in the first 7 days" [56]—and accounts for the custom of waiting to name children until the seventh day of life. Given the social disruption of the last 2 years, recent vaccine coverage has probably been near zero except as provided by relief agencies.

The lack of vaccine coverage should make measles a devastating problem among displaced children. In the Gedo region of Somalia in 1980, measles was the leading cause of mortality, responsible for 47% of all deaths. In the Wad Kowh camp in the Sudan in 1985, measles accounted for 53% of all deaths, with 30 of every 1,000 children <5 years of age dying of the disease each month [14].

Of critical importance to displaced populations will be a campaign of mass measles vaccination; this effort should be assigned a priority second only to that given to the provision

of adequate food [57]. Among refugees in Somalia in 1980, a measles immunization program was highly effective, resulting in the coverage of 86%-97% of the population [58].

Current recommendations are for the use of standard Schwarz vaccine and the immunization of all children 6 months to 5 years old. Those children vaccinated between 6 and 9 months of age should receive an additional dose once they reach 9 months of age [14]. Fever, respiratory tract infection, or diarrhea is not a contraindication to vaccination. In camps for displaced persons, isolation of patients with measles is impractical and is not recommended.

Because measles rapidly depletes stores of vitamin A and may have sequelae such as corneal xerosis, ulceration, and blindness [59], all children with measles should receive 200,000 IU of oral vitamin A [14]. This treatment should be part of a routine program of vitamin A supplementation, which, even under more normal conditions, has been shown to decrease childhood mortality in randomized community trials [60].

### Acute Respiratory Disease

Acute respiratory tract infection (ARI) is a major cause of morbidity and mortality in developing countries. Its impact is greatest among the crowded, the undernourished, and the youngest age groups. ARI has been a leading cause of death among displaced persons. In studies in Somalia (1980), Sudan (1985), Ethiopia (1989), and Malawi (1990), ARI was associated with 6%-13% of cause-specific deaths among displaced persons [14]. In a study of preschool-aged children admitted to hospitals in Mogadishu in 1971-1973, bronchopneumonia was associated with 14%-25% of admissions annually and with case-fatality rates of 9%-15%. Between 47% and 68% of admissions among children ≤1 year of age were due to pneumonia; the corresponding case-fatality rate was 10%-24% [61].

The etiologic agents of ARI among the Somali people are likely to be similar to those causing ARI in the developed world except that *Mycobacterium tuberculosis*, *Coxiella burnetii*, *Bordetella pertussis* [14], and *Corynebacterium diphtheriae* may be more prevalent. Respiratory syncytial virus, adenoviruses, influenza-parainfluenza group viruses, *Mycoplasma pneumoniae*, *Streptococcus pyogenes*, *Streptococcus pneumoniae*, and *Haemophilus influenzae* type b will probably be common pathogens in ARI.

Foreign personnel providing relief in Somalia are likely to acquire ARI. ARI is common among military populations, particularly during and after large mobilizations [62, 63]. American soldiers participating in ODSS had a high incidence of mild ARI of undetermined etiology [64]. British soldiers in ODSS also had high attack rates of pneumonia [65]. It is important to note that the etiology of pneumonia among military populations often differs from that among healthy U.S. adults. Military personnel have historically had

higher rates of pneumonia due to *M. pneumoniae* [66, 67], *Chlamydia pneumoniae* [68], and viruses [69, 70]. Fortunately, most military personnel assigned to participate in ORH will have received vaccinations to prevent influenza, pertussis, diphtheria, and infection with adenovirus types 4 and 7.

### Meningitis

*Neisseria meningitidis* is the agent most likely to cause meningitis among the Somali people. Meningococcal meningitis is associated with high attack rates and high case-fatality rates among displaced persons. For example, an outbreak in 1980 among displaced persons in Thailand resulted in attack rates of 0.13% and case-fatality rates of 28% over a 4-month period. The malnutrition and crowding of much of the displaced Somali population are significant risk factors for such epidemics [71].

Although Somalia lies below and to the east of the traditional sub-Saharan meningococcal belt, recent epidemics in adjacent African nations suggest that Somalia too is at risk. During 1989 an epidemic of group A meningococcal disease occurred in Nairobi; the 3,800 cases of clinical disease represented 250 cases/100,000 population. The meningococcal strain involved was closely related to isolates from epidemics in Saudi Arabia (1987) and Ethiopia (1989). The Nairobi epidemic was unusual in that attack rates were high among adults—a pattern suggesting lack of exposure in the past. The epidemic occurred mainly in the city's slums [72].

Should patients present to medical clinics in Somalia with confirmed meningococcal meningitis (particularly that due to organisms of group A or C), relief workers will need to institute public health interventions, possibly including isolation of patients, increased surveillance for additional cases, chemoprophylaxis for close contacts of patients, and vaccination. Experience among high-risk populations in the African meningococcal belt has resulted in the adoption of a guideline calling for widespread vaccination whenever average disease rates exceed 15 cases/100,000 person-weeks for 2 consecutive weeks [71]. As displaced persons are difficult to count, this guideline may not be practical. A simpler rule of thumb for detecting an outbreak of meningitis is a doubling of the number of cases from one week to the next for 3 consecutive weeks [14].

The optimal treatment for meningococcal disease consists of high doses of intravenous penicillin every 4 hours. However, in an epidemic setting, medical resources may be overwhelmed; thus alternative therapy has been recommended: an intramuscular injection of an oily suspension of chloramphenicol. The patient's condition should improve in 24–48 hours [71]. A second dose of chloramphenicol may be necessary in 25% of cases.

Close contacts of patients with meningococcal meningitis should receive chemoprophylaxis. As many strains of *N.*

*meningitidis* in tropical Africa are resistant to sulfonamides, chemoprophylaxis with rifampin is indicated. The adult dose of rifampin is 600 mg twice daily for 2 days. Children >1 month of age should receive 10 mg/kg twice daily for 2 days; children <1 month of age should receive 5 mg/kg twice daily for 2 days. Pregnant women should not receive rifampin. Ceftriaxone and ciprofloxacin are reasonable but expensive alternative agents. Mass chemoprophylaxis is usually discouraged because it has not proven effective for control of epidemics in populations of refugees and displaced persons [14].

Most U.S. military personnel sent to Somalia will have received a quadrivalent meningococcal vaccine protective against organisms of groups A, C, Y, and W-135. Meningococcal vaccination is prudent for relief workers scheduled to visit Somalia.

### Schistosomiasis

More than 200 million people in the world are infected with schistosomes, and almost 93% of these people live in Africa [73]. Schistosomiasis is a major health and economic problem in Somalia and neighboring regions, with an estimated one-half of the population at risk for infection and a prevalence of 36% in the entire population [73].

*Schistosoma haematobium* is endemic in southern Somalia [2, 74–76], southeastern Ethiopia [77], and northeastern Kenya [78]. In Somalia, essentially all reported cases of schistosomiasis are due to *S. haematobium*. Infection with *Schistosoma mansoni* has been reported in the mid-highland area of Ethiopia (Harar) bordering northern Somalia [74]; although it has also been reported sporadically in persons living in Somalia, these cases are thought to have been acquired outside Somalia [2]. In a survey during 1950 and 1951, no foci of schistosomiasis were found in what is now Djibouti or in northern Somalia. In southern Somalia, the geographic area of highest schistosomiasis endemicity is the land bordering and irrigated by the Shebele and Jubba rivers. Surveys of egg counts in urine specimens in this region demonstrate that as many as 89% of the inhabitants are infected with *S. haematobium* [79]. In the Shebele river valley of Ethiopia on the border with Somalia, 35%–73% of all children 11–15 years old were excreting eggs of *S. haematobium* in the 1970s [77]. An extensive survey conducted by the Chinese Investigation Team for Schistosomiasis Control found a prevalence of *S. haematobium* of 63.4% (512 of 808) in the Shebele river valley and 59.4% (256 of 431) in the Jubba river valley, with rates of 78.7% in Kismayo and 38.1% in Bardera [80]. Schistosomiasis is strongly associated with water development, and any plans to increase irrigation or resettle displaced nomadic populations as farmers will only increase the prevalence and morbidity of this disease. For example, Arfaa found a prevalence of *S. haematobium* of 27%–58% in an area where water development was only be-

ing planned but documented a prevalence of 58%–75% where irrigation was actively being used [76].

Military and civilian humanitarians working in Somalia will be at risk of acquiring infection with *S. haematobium* if they are exposed to freshwater. The military personnel most likely to be exposed are members of construction battalions, especially if they are involved with projects in or around the Shebele and Jubba rivers. During World War II, more than 1,200 cases of schistosomiasis were acquired during the invasion of Leyte in the Philippines. Hardest hit were the combat engineers, with infection rates of 50%–85% [81]. Additional concern has been raised by a short review of seven outbreaks of acute schistosomiasis among U.S. travelers exposed to infested freshwater in Africa; infection rates were 55%–100% in these outbreaks [82].

Clinical manifestations of *S. haematobium* infection are diverse and correspond to the life cycle of the schistosome. When exposed to infested freshwater, human skin is penetrated by cercariae. This event may result in a mild cutaneous erythema of the area or punctate lesions at the site of entry. However, more commonly, the skin lesions will go unnoticed or will be associated only with a prickling sensation as the water evaporates. In persons who have been previously sensitized, the rash may be much more prominent, with a pruritic papular eruption that persists for 7–10 days and heals without scarring.

After penetrating the skin, the cercariae lose their tails and become schistosomulae, which migrate through the lung to the liver. For persons with heavy infestations, eosinophilic pneumonitis may occur at this stage. During the 1- to 4-week period of the parasite's maturation to its adult form in the liver, the infected person is asymptomatic. Male and female pairs of *S. haematobium* adults next migrate through the vascular system to the venous plexus of the bladder. The adult worm pairs spend their 3- to 7-year life-span copulating and laying eggs within the venules of the bladder plexus. Because they acquire a coating of human antigens during their migration through the lungs, they evoke no immune inflammatory response despite their intravascular location [83, 84]. Thus this species is a veritable "stealth" trematode.

The immunologic reaction to the eggs is the primary cause of both acute and chronic clinical symptoms and pathology in schistosomiasis. In acute infection due to *S. mansoni* or *Schistosoma japonicum*, the onset of egg deposition may produce an acute systemic illness known as Katayama syndrome. However, this syndrome is rare in patients infected with *S. haematobium* and would not be expected to be seen in Somalia.

Ectopic syndromes may occur if worm pairs undergo aberrant migration and begin egg deposition outside the vesical plexus. Of most concern would be cerebral schistosomiasis. The most common manifestation with *S. haematobium* is transverse myelitis. This syndrome was well reported when two American students developed dorsal myelitis after the

*mansoni* after participating in a work/study program in Kenya [85]. The clinical presentations also include radiculitis and symptoms suggestive of a spinal cord tumor. More rarely, *S. haematobium* infection may present as a cerebral syndrome manifested as focal epilepsy or as an expanding mass lesion [86].

Once egg deposition in the bladder wall begins, terminal hematuria with associated frequency and suprapubic pain may occur. The most common bladder lesions appear as patches on the mucosal surface; these lesions are located in the trigone in 65% of patients [87]. Polypoid patches are lobulated and usually sessile, with a red granular mucosal surface with focal erosions. These early lesions contain few calcified eggs. The veins underlying the patches usually contain a single pair of adult worms. The second type of lesion, active sandy patches, are finely granular gray to tan areas with a rough sandy surface. In these older lesions, more than half of the eggs are calcified. Last are the residual sandy patches, in which 70%–80% of the eggs are calcified and adult worms are no longer found submucosally. Chronic egg deposition can lead to ulceration, most commonly in the midline of the posterior wall of the bladder. Bladder polyps composed of eggs surrounded by an inflammatory reaction are also common. As the infection becomes more chronic, muscle layers are replaced with fibrous tissue and the eggs calcify. This process leads to loss of bladder elasticity and eventually to a calcified contracted bladder.

The ureters are also commonly involved, especially the ureterovesical junction and the lower ureter [88]. All of the lesions that are found in the bladder can also develop in the ureter, leading to destruction and obliteration of both the inner and outer longitudinal layer of muscle. Eventually, the muscle is replaced with a fibrotic mass that calcifies. Ureteral obstruction is a serious and common occurrence in chronic *S. haematobium* infection. The obstruction is due to concentric sandy patches in 70% of cases, to obstructive polyps in 25% of cases, and to calculi in the remainder. In 95% of cases, obstruction is incomplete. Hydroureter is common and can be attributable to either of two mechanisms: stenosis with proximal hydrostatic dilatation or replacement of the muscular layers with fibrous tissue and resultant passive dilatation.

Ureteral obstruction often leads to bilateral hydronephrosis, which can be severe. However, in most series of cases, renal function has been well maintained, even with advanced changes. A reduction in rates of creatinine clearance is uncommon, but a reduction in maximal urinary concentrating ability is frequent [89]. The seminal vesicles, urethra, prostate, and other pelvic organs can also be involved.

The chronic complications of urinary schistosomiasis may be found commonly among the infected Somali population. The future development of chronic changes should be extremely rare in relief workers because of the limited life-span of the adult worms, the lack of reinfection, and the low intensity of infection.

An association between infection with *S. haematobium* and cancer of the bladder has been postulated since the turn of the century. In Egypt, cancer of the bladder is the most prevalent cancer in men; a study published in 1981 found that 700 of 2,500 new cancer patients on the Cairo Cancer Institute Registry had bladder cancer [90]. The incidence of schistosomal bladder cancer peaks during the third to the fifth decades of life, while cases are infrequent during the sixth and seventh decades. This pattern is quite different from that in the West, where bladder cancer occurs only rarely in the young or middle-aged. The average age at diagnosis of bladder cancer is 60 years in the United States versus 41 years in Egypt [90].

The histopathology of schistosomal bladder cancer also differs from that of the nonschistosomal disease. In the schistosomal form, 77% of the lesions are squamous cell and only 21% are transitional cell; conversely, in nonschistosomal bladder cancer, 94% of lesions are transitional cell. The prevalence of schistosome-associated bladder cancer in Somalia is unknown.

Another complication of *S. haematobium* infection is chronic urinary excretion of *Salmonella typhi* and *Salmonella paratyphi*, along with chronic intermittent salmonella bacteremia [91]. Successful management must include antibiotic therapy for the salmonella infection combined with eradication of the schistosomes [92, 93].

### Diagnosis of Schistosomiasis

Urinary schistosomiasis is diagnosed by detection of the terminal spined ova of *S. haematobium* in urine or in urogenital tissue (in the latter case via cystoscopic biopsy). Urine samples should be obtained between noon and 3 PM—the period of peak egg excretion—and should be examined for eggs after centrifugation or simple sedimentation. Serological tests have proven less than satisfactory; however, they may be of some value in evaluating ectopic syndromes, such as transverse myelitis, in which total egg burden may be low and urine examination may be unrevealing.

The radiological features of urinary schistosomiasis are distinctive, with ureteral calcifications considered almost pathognomonic. In most series of cases from Egypt, all symptomatic patients have had abnormal urograms and more than 60% of chronically infected patients have had obstructive uropathy [94]. Postvoid retention of urine is the most common finding, when the ureters are involved, vesicoureteral reflux is noted in 25% of studies. Stones are particularly common and are found mostly in the pelvic portion of the ureter. These stones may be huge (weighing up to 55 g); they are radiopaque 98% of the time and bilateral in 48% of cases. One patient had 104 stones removed from his lower ureters [94].

### Treatment and Prevention of Schistosomiasis

The current drug of choice for the treatment of infection with any of the four species of schistosomes is praziquantel. The standard dose for *S. haematobium* infection is 40 mg/kg divided into two doses and given in 1 day [51]. Cure rates of up to 95% can be expected, and the drug is well tolerated with only minimal toxicity (headache, dizziness, and/or abdominal discomfort). Numerous studies have shown that treatment can reverse many radiological and pathological changes, especially bladder polyps, thickening of the bladder wall, and hydronephrosis [89, 95, 96]. Chronic severe hydronephrosis may be irreversible.

The best personal method of preventing schistosomiasis is the avoidance of contact with schistosome-infested freshwater. U.S. Navy and Army researchers have recently tested a topical niclosamide lotion that may prove effective in preventing cercarial penetration of the skin [97].

### Hepatitis

The Horn of Africa is hyperendemic for hepatitis viruses. Infection with hepatitis A virus (HAV), which is spread by the fecal-oral route in conditions of poor sanitation and overcrowding, is endemic in all parts of the world; however, some of the highest prevalence figures are found in the Horn of Africa. The prevalence of antibody to HAV is virtually universal in individuals >15 years of age in Somalia (95%), Ethiopia (84%), and Djibouti (98.5%) [98–100]. The prevalence of HAV antibody increases rapidly with age, with the steepest rise during the first 4 years of life. Infection during early childhood is inapparent and protective for life.

Immune serum globulin (ISG) is highly effective in the prevention of hepatitis A. Studies have shown that ISG can prevent 80%–90% of cases if given before or up to 2 weeks after exposure [101]. All military personnel and civilian humanitarians should receive ISG (0.06 mL/kg) before traveling to Somalia. The dose should be repeated every 3–5 months. Because fewer than 25,000 U.S. troops are expected to be involved in ORH (whereas 500,000 troops were involved in ODSS), the impact of such prophylaxis on civilian supplies of ISG should not be serious. The highly effective hepatitis A vaccine recently marketed in Europe will provide a more convenient and effective method of prevention in the future [102].

The reported prevalence of markers of infection due to hepatitis B virus (HBV) was 68% for Somali adults in 1991 [99]. In Mogadishu, the prevalence of HBV markers was 89% among men seeking treatment for sexually transmitted diseases (STDs) and 66% in a control group of men [103]. The reported rate of chronic carriage of hepatitis B surface antigen (HBsAg) was 11%–12% in the general population [104].

105] and ranged from 3%-4% among children <14 years of age in Mogadishu to 23% among nomadic men [106]. An additional study in Mogadishu found 4% of neonates, 20% of prostitutes, 22% of educated women, and 37% of pregnant women positive for HBsAg [107]. A number of studies have revealed associations between hepatitis B and other diseases endemic to the region, including ancylostomiasis, urinary schistosomiasis, and lepromatous leprosy [104, 108-111]. It is interesting that the traditional use of the stimulant *khat* is suspected as a risk factor [100].

Data on the prevalence of hepatitis C in the Horn of Africa are limited. However, in a study of sera collected in 1987, the prevalence was 2.7% among persons at high risk for STDs and 1.5% in the general population (E. Fox, personal communication).

The newly identified hepatitis E virus (HEV), which is responsible for most cases of enterically transmitted non-A, non-B hepatitis, has been detected in the Horn of Africa. A large outbreak of hepatitis E occurring from January 1985 to September 1986 at four refugee camps in Somalia resulted in more than 2,000 cases and 87 deaths. Intensive investigation of the outbreak at the Tug Wajale B refugee camp in northwestern Somalia demonstrated the presence in stool of virus-like particles later identified as HEV [112]. Characteristic epidemiological features of hepatitis E include an incubation period of 40 days (as opposed to 30 days for hepatitis A and 60-180 days for hepatitis B), common occurrence among adults, and high mortality among pregnant women. Large outbreaks of acute viral hepatitis among adults in areas where the population is immune to hepatitis A should suggest the presence of hepatitis E. The signs and symptoms of hepatitis E are similar to those of other forms of viral hepatitis. The majority of nonpregnant patients recover completely, and there is no evidence of chronic liver disease as a long-term sequela.

In addition to causing epidemics, HEV may commonly be responsible for sporadic cases of hepatitis. In a recent study in Ethiopia, serum antibodies to HEV were found in 33% of patients with acute sporadic hepatitis (versus only 7% of healthy controls); IgM antibodies to HAV and markers of acute infection with HBV (HBsAg and IgM antibody to hepatitis B core antigen) were found in 8% and 20% of patients, respectively. For 39% of patients, the cause of the acute sporadic hepatitis was unidentified [113]. An analysis of acute and convalescent-phase sera revealed that 62.5% of 40 adult Djiboutian patients diagnosed with acute hepatitis after April 1992 were positive for IgM antibodies to HEV (G. R. Rodier, unpublished data). These data emphasize the prominent role of HEV in acute sporadic hepatitis among adults living in the Horn of Africa.

Hepatitis E remains a threat to civilian relief workers since no vaccine has yet been produced and the effect (if any) of ISG against this virus remains unknown.

## Sexually Transmitted Diseases

STDs are highly endemic in underdeveloped countries where resources for treatment, prevention, and education are often unavailable to the general population.

Syphilis is highly prevalent in Somalia. During the late 1980s and early 1990s in Mogadishu, the proportion of individuals with a positive *Treponema pallidum* hemagglutination assay ranged from 3% in a control group and among pregnant women [103, 114] to 28.1%-69% among prostitutes [114-117]; 26% and 47%, respectively, of these groups of women had active syphilis [114, 117]. In a follow-up study, the 6-month incidence of syphilis among prostitutes was 12.5% [117]. In three separate surveys, 4.4% [115], 10% [101], and 12.6% [116] of men seeking treatment for STDs had a positive *T. pallidum* hemagglutination assay.

Yaws, an endemic nonvenereal treponematoses, can cause false-positive results in serological tests for syphilis. However, clinical data and results of surveys among children in the region do not support a significant prevalence of yaws [118].

Few reliable data exist about chancroid in Somalia, primarily because of the relative difficulty of isolating the infectious agent, *Haemophilus ducreyi*. However, physicians and nurses working in the region often suspect a diagnosis of chancroid on clinical grounds, and infection with *H. ducreyi* is believed to be a common cause of genital ulceration (F. Marrot, personal communication). The relative importance of chancroid and syphilis in the region is unknown.

Infection with herpes simplex virus is almost universal in Somalia. The prevalence of antibodies to this virus is 100% among prostitutes and male attendees at STD clinics [103, 107]; however, the test used in published studies does not differentiate antibodies to type 1 virus from those to type 2 virus.

Gonorrhea has a high prevalence in Somalia. Although one recent report from Mogadishu stated that only 7.7% of gonococcal isolates were penicillinase producers [115], reports from nearby countries in the Horn of Africa suggest that rates of resistance have the potential to be much higher. Among gonococcal isolates in Djibouti and Ethiopia, 16% and 66%, respectively, were reported to be penicillinase producers [119, 120]. More recent data (from 1992) document a prevalence of penicillinase-producing strains of 50% among 160 isolates of *Neisseria gonorrhoeae* collected in Djibouti (R. Bercion, personal communication). Although 24% of these gonococcal isolates from Djibouti were resistant to spectinomycin, no resistance to the standard therapeutic agent ceftriaxone has been reported from the Horn of Africa. Of interest is that sensitivity to vancomycin, which is used in modified Thayer-Martin agar to select for the growth of *N. gonorrhoeae*, has been identified in strains from Djibouti and has been implicated as a cause of decreased isolation of the organism [121].

The AIDS pandemic reached the Horn of Africa in the mid-1980s, primarily affecting Ethiopia [122-124]. As of April 1988, Ethiopia had reported 37 cases of AIDS and Kenya had notified WHO of a total of 2,097 cases, whereas Djibouti and Somalia apparently remained free of the disease. Djibouti reported its first case of AIDS in November 1988 [125], and Somalia reported its first four cases in July 1989 [126]. By late December 1991, Ethiopia, Djibouti, and Somalia had declared 1,534, 104, and 13 cases of AIDS, respectively [127].

In two serological surveys performed in February 1987 and in February and March 1989 in Mogadishu and south of the capital city (i.e., in Marka and Kismayo) among prostitutes and male attendees at STD clinics, researchers found that all sera were negative for antibodies to types 1 and 2 human immunodeficiency virus (HIV-1 and HIV-2) [115, 116]. In 1990, only one of 155 prostitutes tested in Mogadishu was found to be positive for HIV-1 [117]. In another survey in May 1990, the seroprevalence of HIV-1 was 3% among prostitutes in Mogadishu [128]. No data from northern Somalia are available.

The strains of HIV-1 present in the Horn of Africa seem to be distinct from those isolated in Central and West Africa [129, 130]. This difference could be important in determining the type of HIV vaccine to be employed in the future. Although HIV-1 is predominant in the region, the presence of HIV-2 has also been documented [131].

The rapid spread of HIV in Djibouti may be a harbinger of the future for Somalia. This possibility is especially likely because of the traditional population movements between Hargeysa in northern Somalia and Djibouti. The situation is compounded by the high degree of mobility of prostitutes in Djibouti; the fact that these prostitutes are primarily Ethiopian again emphasizes the potential for regional spread of HIV [132, 133]. In the first quarter of 1992, the prevalence of HIV was 51.3% among street prostitutes, 21.7% among bar hostesses, and 11.8% among male attendees at STD clinics (Djibouti Ministry of Health, unpublished data).

Tuberculosis has been reported to be the most frequent opportunistic infection among AIDS patients from Ethiopia [134], whereas no Kaposi's sarcoma has been observed. Tuberculosis and chronic diarrhea are the most common opportunistic infections reported in AIDS patients from Djibouti.

Prevention of dissemination of HIV-1 in Somalia will be difficult because of the high rates of genital ulcer disease that have been associated with increased transmission [135-138]. This problem is compounded by poor access to treatment for syphilis, chancroid, and herpes infection. Control of the spread of HIV-1 will be further impaired by political unrest, resettlement of displaced populations, poor diagnostic capabilities, and infrequent use of condoms.

### Arboviral Diseases

More than 500 arthropod-borne viruses have been identified worldwide. The diseases they cause are primarily zoo-

notic, and about 100 of these viruses have been responsible for disease in humans. A number of arboviruses are endemic in the Horn of Africa; however, specific data are lacking for most of them in Somalia.

Dengue virus is most likely to cause outbreaks among humanitarian relief workers and the local population. Dengue has been well documented as a problem for the military, with outbreaks among personnel in the Philippines and Thailand [139], and as an important imported disease. One hundred two cases of suspected dengue were imported into the United States in 1990 [140].

In May 1983, three expatriates who were living in Mogadishu and had developed a clinical syndrome typical of dengue were found to have a specific rise in titer in the dengue type 2 IgM capture antibody assay [141]. Further evidence of dengue activity in northeastern Africa came from the investigation of an outbreak of a febrile disease that occurred each winter in Sudan and was thought by local physicians to be antibiotic-resistant malaria [142]. The most common diagnosis was acute dengue fever; there were 17 isolates of dengue type 2 and one of dengue type 1.

The potential for outbreaks of dengue in the area was confirmed by serological evidence in outbreaks of febrile disease in the Dam refugee camp near Hargeysa in 1985-1987 [143]. In addition, a serosurvey conducted during 1989 in Berbera found that 54% of persons tested had antibodies suggestive of prior dengue infection. An outbreak of a dengue-like illness occurred during the winter of 1991-1992 in Djibouti, with an estimated 10,000 cases. During this outbreak, dengue type 2 was isolated for the first time in the Horn of Africa (G. R. Rodier and D. Gubler, unpublished data).

The potential for outbreaks in Somalia is emphasized by the presence of the vector, *Aedes aegypti*, in the area. The Mogadishu environment was found to be permissive of transmission of dengue during a survey that revealed *A. aegypti* house indices of >80%; the mosquitoes were found predominantly in water storage jars, which are ubiquitous throughout Somalia (J. Zimmerman, personal communication). Djibouti was considered free of the vector at the time of a 1969 survey, but entomological surveys conducted in October 1992 confirmed the reintroduction and significant presence of *A. aegypti* in the capital city (S. Cope and G.R. Rodier, unpublished data).

As of mid-January 1993, 10 cases of dengue have been diagnosed in participants in ORH, as confirmed by IgM capture ELISA, and another 15 clinically suspected cases are awaiting serological confirmation (M. Wallace, personal communication).

Important differences exist between the anopheline vectors of malaria and *A. aegypti*, vector of both yellow fever and dengue. *Aedes* is a genus of urban domestic mosquitoes. The female is a day-biter with a silent flight and unnoticed bite, breeding in and around dwellings in household water containers. Thus it is imperative that mosquito repellent be

applied both at night (for *Anopheles*) and during the day (for *Aedes*).

No vaccine for dengue is available, although extensive research has been conducted on attenuated strains of the virus and, more recently, on subunit vaccine preparations [144, 145]. Current investigations are being directed at identification of the epitopes that are most important for protection [146, 147]. Difficulties persist because of the lack of an appropriate animal model in which to test the attenuated vaccines and concern about antibody-dependent enhancement of viral growth [148]. The lack of a vaccine makes personal protection and vector control critical components of a prevention program.

Yellow fever virus has been found in the Horn of Africa. According to international quarantine regulations, Somalia lies within the Central African yellow fever belt. Despite a severe epidemic in the Omo and Didessa valleys of Ethiopia (with more than 100,000 cases and 30,000 deaths), no cases of yellow fever have been documented in Somalia. However, as has been mentioned, the vector *A. aegypti* is present in the country, and some seroepidemiological evidence of infection (i.e., a seropositivity rate of 6%) was found during the 1960s in the Giohar district of Somalia, north of Mogadishu [149]. The threat of yellow fever is minimal and should be nonexistent for persons vaccinated with the attenuated 17D strain. The vaccine is 99.9% effective, and protection may persist for 30–35 years, although revaccination every 10 years is required by international health regulations [150].

The information on other arboviral activity in Somalia is spotty and inconclusive. Some evidence for endemic activity of West Nile virus has been obtained in serosurveys in the Giohar district of Somalia as well as in neighboring countries [2, 151]. Moreover, serological evidence of prior infection with West Nile virus was found in a survey in Berbera in 1989, where 34% of persons tested were seropositive; in contrast, a survey in 1982 of individuals living at a higher elevation in Hargeysa found only one seropositive person among 28 tested [42, 143]. *Culex* species are the vectors of West Nile virus, while migratory birds serve as important animal reservoirs. In their annual migration from central Asia to East Africa, birds cross the Bab-el-Mandib strait between Djibouti and Yemen, providing an entry for arboviruses into Africa.

A serosurvey for arboviruses was conducted in 1983 among 187 lifelong residents of four regions of Somalia (Hargeysa, Mogadishu, Baidoa, and Kismayo); the representation of the four regions was almost equal. The tests used were hemagglutination inhibition and indirect fluorescence antibody, and a wide range of seroprevalences was detected [152]. The prevalence of flaviviruses was highest (82%–88%) at the lower elevations in the south of Somalia, while in sera from the north the prevalence was 19%. Of the serum samples from Kismayo, 43% contained antibodies to chikungunya virus and 12% were positive for Rift Valley fever virus. No sera from Hargeysa in the north were positive for these

two viruses; this low seroprevalence of flaviviruses was confirmed in a 1987 survey [143]. Titers of antibody to Sindbis virus were elevated in only two of the 187 sera tested, and both of these samples also contained elevated titers of antibody to other alphaviruses.

Limited serosurveys for hemorrhagic fever viruses (those causing Lassa, Marburg, Ebola, Hantaan, and Crimean-Congo hemorrhagic fevers) have yielded negative results in Somalia and Djibouti [143, 152, 153]. Hantaan virus has been demonstrated in rats trapped in the port area of Djibouti; however, no clinical cases of Hantaan fever have been reported [154].

Arboviral diseases bear many similarities to one another and cannot be differentiated on clinical grounds alone. The classic dengue syndrome seen in adults infected for the first time includes an abrupt onset of fevers, severe myalgias, arthralgias, frontal headache, and retroorbital pain. The fevers are often biphasic; the first febrile phase of 1–6 days is followed by an afebrile period of 1–3 days, which is in turn followed by a recurrence. This basic clinical syndrome is also typical of West Nile fever, chikungunya fever, malaria paroxysms, and leptospirosis. The presence of cough (described in 40%–60% of cases in some series) can result in the confusion of arboviral disease with influenza. Altered gustatory sensation (often described as a metallic taste) is suggestive of dengue, while a sore throat may develop in West Nile and chikungunya fevers. Most suggestive of chikungunya infection are severe arthralgias and frank arthritis affecting most prominently the small joints of the hands, wrists, feet, and ankles [155]. The onset of disabling arthritis is often so abrupt that the patient can remember the exact time; this predominance of joint pain led to adoption of the Swahili term *chikungunya*, which means "that which bends up." Residual arthralgias may persist for weeks to months after recovery from the febrile episode. It is interesting that an association between the presence of the HLA-B27 haplotype and chronic arthritis has been noted. Rarely, patients with Sindbis fever may also have arthralgias. Dengue hemorrhagic fever and shock syndrome probably will not be seen among relief workers as this manifestation occurs primarily in children.

A rash is common with dengue, West Nile, chikungunya, and Sindbis infections, usually developing towards the end of the febrile period. In dengue, the rash is generally maculopapular and may be preceded by more generalized flushing. It may also have a petechial or purpuric component. In West Nile fever, the rash is predominantly truncal (as it is in chikungunya fever) and can also be found on the extensor surface of the extremities. Sindbis fever has a more distinctive rash that is found over the buttocks, legs, palms, and soles. The lesions may be both papular and vesicular. They are pruritic and occur in crops that may last up to 10 days, leaving a brownish discoloration after they resolve. An enanthem, with vesicles or small ulcerated lesions in the oropharynx and an associated sore throat, has been described.

It should be emphasized that the specific diagnosis of a suspected arbovirus infection on serological grounds alone is extremely difficult since there are numerous cross-reactions within groups (either flaviviruses or alphaviruses). This same cross-reactivity creates problems in the interpretation of serosurveys. Although IgM antibody assays and plaque neutralization techniques are more specific than the classic hemagglutination inhibition method, isolation of the virus remains the gold standard for diagnosis.

The impact of arboviral disease on ORH will depend on vector, reservoir, and virus populations in the area. Despite the threat of arboviral diseases in ODSS, there was minimal evidence of arboviral activity. In a study of 32 ODSS participants with febrile illness and 102 asymptomatic participants, all were seronegative for Sindbis, dengue, and sandfly fevers. A single case of West Nile fever was documented [156].

### Tuberculosis

An estimated one-third of the world's population is infected with *Mycobacterium tuberculosis*. In the past decade, an average of 2.5 million to 3.2 million cases have been reported each year to WHO. In 1990 alone, an estimated 8 million people developed tuberculosis and 2.6 million to 2.9 million died of the disease. The risk of acquiring tuberculosis in the Horn of Africa remains one of the highest on the African continent.

In Somalia, the National Health Plan of 1980-1985 noted that an estimated one in three children from 5 through 9 years of age was infected with *M. tuberculosis* and that 1% of the population had active disease due to this organism [6]. Tuberculosis is a serious problem among refugees in the region. A survey conducted among Somali refugees from the Ogaden region of Ethiopia revealed a prevalence of sputum smear positivity of 2%-3% [157]. Before that survey, no microscopy or radiography facilities were available in the camps and no active case-finding had been undertaken. Diagnosis was based on a history of chronic cough unresponsive to tetracycline treatment as well as hemoptysis, night sweats, and weight loss. The standard therapeutic regimen was 3 months of daily administration of streptomycin, isoniazid, and thiacetazone followed by a 9-month maintenance regimen of isoniazid plus thiacetazone [157]. Except in Djibouti, pyrazinamide has been considered to be too expensive for tuberculosis control programs in the Horn of Africa.

Tuberculosis should be considered a serious threat to relief workers who are in close contact with the native population. This infection can also be expected to be a problem among Somalis who seek asylum in other countries. An examination of Ethiopian refugees in the United States revealed that 72% had a positive tuberculin skin test, 3.4% had abnormal chest radiographs, and 0.4% had active tuberculosis [158]. Surveys documented active disease in 1.9%-9% of Ethiopian immigrants to Israel [48, 159, 160]. In Djibouti in 1990, 16% of all

new cases of tuberculosis were imported from Somalia and 25% from Ethiopia [161].

Multidrug-resistant tuberculosis is being reported increasingly from many areas of the world; the Horn of Africa is no exception. In tuberculosis control programs among refugees in Somalia, 50% of the patients who began a course of treatment were lost to follow-up in the first year. In 63% of cases, the reason for default was related to social factors or to the patient's feeling well [156]. In Djibouti, 25.6% of patients were lost to follow-up before they had completed 6 months of therapy. The consequences of this poor compliance are relapse and the emergence of drug resistance.

Although data are not available for Somalia, recent assessments of the drug sensitivity of *M. tuberculosis* in Djibouti suggest a serious emerging problem for the Horn of Africa. In 1990-1992, 36% of *M. tuberculosis* isolates from suspected instances of treatment failure displayed combined resistance to isoniazid, rifampin, streptomycin, and ethambutol; 78% of these isolates were resistant to at least two of the four drugs. Most recently, isolates resistant to eight antibiotics have been found; however, these strains have remained sensitive to pyrazinamide (G. R. Rodier, unpublished data).

In the United States, recent outbreaks of multidrug-resistant tuberculosis have been associated with HIV infection [162, 163]. In Djibouti, where HIV has only recently begun to spread, 10% of all cases of tuberculosis in adults in 1992 were associated with HIV. The introduction of HIV into a population with a baseline high prevalence of tuberculosis can lead to a logarithmic increase in rates of tuberculous disease [164].

The combination of a disorganized national tuberculosis control program, migration of populations, poor compliance, famine, crowding in camps for displaced persons, multidrug resistance, and increasing HIV prevalence make the outlook for the future grim.

### Leishmaniasis

Leishmaniasis comprises a variety of clinical manifestations of infection with protozoa of the genus *Leishmania*. The leishmanial diseases of humans are commonly divided into those producing mainly cutaneous lesions (including the clinically distinct mucocutaneous leishmaniasis) and those causing a systemic infection with predominantly visceral lesions. In the Old World, *Leishmania donovani* and *Leishmania infantum* are the causative agents of visceral leishmaniasis, or kala-azar, whereas *Leishmania aethiopica*, *Leishmania major*, and *Leishmania tropica* cause cutaneous leishmaniasis [165].

Leishmaniasis was the vector-borne parasitic disease that had the greatest impact on U.S. troops involved in ODSS; to date, however, only 28 of the more than 500,000 participants in ODSS have been diagnosed as having leishmaniasis: 17 developed cutaneous leishmaniasis, and 11 experienced a

syndrome that has been termed viscerotropic leishmaniasis and is caused by *L. tropica* [166, 167]. Because the organisms were isolated from bone marrow and the extent of the problem was unknown, all participants in ODSS were prohibited from donating blood in the fall of 1991. Since no additional cases of leishmaniasis have been diagnosed since May 1992 despite active case-finding efforts by both the Army and the Navy, the ban on blood donation was lifted in January 1993.

Whereas in Saudi Arabia cutaneous leishmaniasis is well described, the vector is seasonally abundant, and more than 16,000 cases have been reported annually, in Somalia the disease is considered rare. In Cahill's extensive review of his work in Somalia in 1971, he noted that there had been no reports of cutaneous leishmaniasis. In addition, in a leishmanin skin-test survey of more than 700 patients in an area of Somalia endemic for visceral leishmaniasis (see below), Cahill found no typical cutaneous scars [2]. The most commonly implicated vector in the Middle East, *Phlebotomus papatasi*, has not been reliably described in Somalia [165], although other vector species have been noted.

Visceral leishmaniasis does appear to have areas of focal endemicity in Somalia. Baruffa described the first endemic focus in southern Somalia in 1965, reporting 12 cases documented by splenic aspiration in nomads from an area about 50 miles north of Mogadishu, around the city of Giohar [2]. The British had noted a focus in northern Somalia, with a few cases diagnosed annually, during the 1950s. In an attempt to better delineate the prevalence of visceral leishmaniasis, Cahill administered the leishmanin skin test (*L. donovani*, Kenya strain) to more than 700 healthy subjects, as mentioned above. He found that skin tests were positive for 13%–22% of those tested—a figure in the range of rates reported from other areas of Africa known to be hyperendemic for kala-azar [2].

The incubation period of leishmaniasis ranges from at least a week to many months—or even up to 2 years in visceral disease. Kala-azar is a chronic disease characterized by fever, hepatosplenomegaly, lymphadenopathy, anemia, leukopenia, and progressive emaciation and weakness. Untreated, it is usually fatal. The diagnosis of kala-azar is made preferably by culture of the organism from biopsy or aspirated material or by demonstration of intracellular amastigotes (Leishman-Donovan bodies) in stained smears from bone marrow, spleen, liver, lymph node, or blood. In cutaneous leishmaniasis, the disease starts with a papule that enlarges and becomes an indolent ulcer. Lesions may be single or multiple. They may heal spontaneously within weeks or last for a year or more. Cutaneous leishmaniasis is diagnosed by microscopic identification of intracellular amastigotes in stained smears of material from the edges of the lesions or by culture on suitable medium (i.e., Novy, MacNeal, Nicolle [NNN] medium).

The prevention of leishmaniasis relies primarily on personal protective measures to reduce exposure to sandflies.

Long protective clothing and insect repellent (applied to uncovered skin) are useful, especially during bivouacking in the field. Bed nets impregnated with a synthetic pyrethroid (e.g., permethrin) enhance protection against sandflies, which can easily penetrate untreated mesh. Other control measures vary from area to area, depending on the habits of the mammalian hosts and the vector phlebotomines. These measures include case detection and treatment, periodic application of residual insecticides, elimination of potential breeding places (e.g., rubbish heaps, termite hills), destruction of known animal reservoirs, and appropriate environmental management [150]. Sandflies have so far shown little resistance to insecticides [168].

### Intestinal Parasitic Diseases

As would be expected in a rural, predominantly nomadic or seminomadic population, the prevalence of intestinal parasites is high in Somalia. In a survey in the southern riverine areas of the country, Cahill found that 75% of 740 men surveyed had one or more intestinal parasites [2]. *Ascaris* eggs were found in 44%, while *Enterobius* and *Ancylostoma* were found in 39% and 32% of stools, respectively [2]. Peltola et al. found intestinal parasitism rates of 48%–88% among rural Somali children and their mothers [169]; in this study *Trichuris* had the highest prevalence (79%), *Ascaris* the next highest (30%), and *Ancylostoma* the third highest (26%). Other studies have found similar rates [170]. Rates of *Ancylostoma* in the moist banana-growing areas along the Jubba River have been reported to be almost 100% [2]. Among protozoa, *Giardia* has been most prevalent (15%–17%), with much lower rates of *Entamoeba histolytica* (1.1%–2.5%) and *Cryptosporidium* (0.4%–1.3%) [2, 169].

A consistent finding has been higher rates of intestinal parasitism among the settled farm communities in the south than among the nomadic tribes in the drier north. For example, in the Peltola study [169], low rates of parasitism were reported from Burao in the north, with only 3.4%, 0.4%, and 0.9% of stools positive for *Trichuris*, *Ascaris*, and *Ancylostoma*, respectively.

Even when living in the south, nomads have had low rates of intestinal parasitism. Particularly low among nomads have been the rates of infection with intestinal protozoa. Ilardi et al. [171] found *Giardia* in only 1.6% of Somali nomads and detected essentially no *Entamoeba*.

It has been hypothesized that milk protects against intestinal protozoal infections. For example, *E. histolytica* needs high levels of iron for maximal growth; 44  $\mu\text{mol}$  of iron/L is estimated to be optimal and 12  $\mu\text{mol}$ /L to be minimal [172]. In a study among the Masai from the eastern tier of the Rift Valley in Kenya, Murray et al. [173] found that milk from Zebu cattle contained only 1.5  $\mu\text{mol}$  of non-enzyme-associated iron/L; in addition, the milk contained unsaturated transferrin and lactoferrin, which could actively compete for

colonic iron. These investigators found less serological evidence of *E. histolytica* infection among members of the Turkana tribe of northwestern Kenya who consumed milk only than among other members of the same tribe who consumed a mixed diet of milk and fish [174]. This concept of host defense due to the withholding of nutrients from the pathogen has been termed "nutritional immunity" [175]. Conversely, the requirement of *E. histolytica* for iron may explain the high prevalence of invasive amebiasis among the Zulu of South Africa, whose traditional diet and home-brewed beer are both high in iron [175]. The iron-deficiency anemia that is so common among Somali nomads may also be protective against other systemic diseases. Murray et al. found that iron replacement in Somali nomads led to a significant reactivation of preexisting diseases such as malaria, tuberculosis, and brucellosis [176]. They hypothesized that iron deficiency may be an "ecological compromise" allowing improved host defense against certain pathogens. Thus re-feeding and iron supplementation may have mixed consequences in displaced populations.

The risk of acquisition of intestinal parasites will probably be moderate among civilian relief workers in Somalia. If American troops adhere to sound preventive measures, their risk should also be low. In a survey of 422 U.S. marines who had served as frontline troops in ODSS, living under arduous conditions for 5 months in Saudi Arabia and Kuwait but adhering to excellent preventive-medicine discipline and having limited contact with the local population, all were found to be negative for intestinal helminths. Only nine (2%) of these marines were excreting *Giardia lamblia* cysts, and all nine were asymptomatic [177].

### Leptospirosis

Relatively old, unvalidated seroepidemiological data suggest that leptospirosis may currently be a problem in Somalia. In a serological survey conducted in 1975-1976 among 372 Somalis, the microscopic agglutination test was performed and 16 serovars of *Leptospira interrogans* were screened for; 50.5% of sera tested were positive [178]. The prevalence ranged from 63.5% in the rural areas near the rivers to 37% in urban Mogadishu. Almost half of the sera reacted to multiple serovars. The most common serovar was bratislava (serogroup Australis); reactivity to this serovar was found in 45.6%-76.9% of sera and had not been previously recognized in Africa. In a serosurvey of domestic and wild animals, Moch et al. found leptospirosis prevalence rates of >50% among domestic animals in many regions of nearby Ethiopia [179]. The combination of these seroepidemiological data, the close association of nomads with their domestic animals, and the use of the same small depressions and muddy pools as a source of water for both makes it reasonable to suspect that leptospirosis will be a problem for displaced Somali populations and possibly for relief workers as well.

Doxycycline (200 mg administered once weekly) was shown to be 95% efficacious in the prevention of leptospirosis during jungle training of U.S. troops in Panama [180]. This result suggests that the prevention of leptospirosis may be an added benefit of doxycycline prophylaxis for malaria. A 7-day course of intravenous penicillin or doxycycline constitutes effective treatment for leptospirosis [181, 182].

### Rickettsial Diseases

Rickettsial disease is a potential problem for visitors to rural Africa. Recently, 23% of 169 U.S. soldiers deployed to Botswana for 2 weeks developed clinical or serological evidence of rickettsial infection (B. Smoak, personal communication). Although few data are available on rickettsial infections in Somalia, disease due to these organisms is quite common in neighboring countries. Both Kenya and Ethiopia are known to have *Rickettsia prowazekii*, *Rickettsia conorii*, and *Rickettsia typhi* [183-185]. A high prevalence of antibodies to *R. conorii* (40%) and *C. burnetii* (40%) was noted in an ELISA survey in Berbera in 1989 [42]. A 1990 serological study of 95 Somalis living in a Djibouti refugee camp revealed high titers of antibodies to a number of rickettsial organisms. The refugees (mean age, 27 years; range, 1-80 years) had antibodies to *R. conorii* (29%), *R. typhi* (13%), and *C. burnetii* (7%); these antibodies indicated prior exposure to these agents (G. R. Rodier, unpublished data). These two studies suggest the possibility of a high prevalence of rickettsial disease in Somalia.

*R. conorii* is the most common cause of rickettsial disease in U.S. travelers [186]. The rickettsia is transmitted by ticks and causes tick typhus, which is also known as boutonneuse fever, Kenya tick typhus, and African tick-bite fever. The classic clinical picture includes fever, headache, an eschar at the site of the tick bite, and a maculopapular rash. Normally, the disease is self-limiting.

*R. typhi* causes murine typhus, a febrile illness associated with a maculopapular rash. Fleas transmit the rickettsia to humans, while rodents act as a reservoir for the fleas. The disease is usually self-limiting, may go unnoticed, and may be confused with measles or rubella. A single case of murine typhus had been diagnosed serologically in a French nurse as of mid-January 1993.

*C. burnetii* is transmitted by inhalation and causes the atypical pneumonia of Q fever. Mammals—most commonly cattle, sheep, and goats—are the animal reservoir. The distribution of *C. burnetii* is not well understood in Africa. A number of epidemics of Q fever occurred in military personnel during World War II when troops stirred up contaminated dust while setting up sleeping quarters in the rural barns of Italy [187]. Attack rates may be high in nonimmune persons, but the disease is seldom fatal.

Fortunately, *R. prowazekii*, the agent causing the very serious disease known as epidemic typhus, is not thought to be endemic to Somalia [188]. However, sporadic cases have oc-

curred in refugees from Ethiopia, who apparently brought the vector (the body louse) and *R. prowazekii* with them from the highlands of Ethiopia [189]. *R. prowazekii* is also reported to be endemic in Kenya [188]. Epidemic typhus is manifested by symptoms of fever, headache, hypotension, cough, maculopapular or petechial rash, and prostration, with an estimated mortality of 20% in untreated cases [188].

A diagnosis of rickettsial disease is confirmed by isolation of the organism or by positive serological tests. Treatment with tetracycline, doxycycline, or chloramphenicol is highly effective and normally results in defervescence in 2–3 days [186]. Treatment should continue until the patient has been afebrile for 48 hours. A single dose of doxycycline (100–200 mg for adults) has been effective against louse-borne typhus, but its success against other rickettsial diseases is unknown [188, 190].

Good personal hygiene, insect repellents, protective clothing, berthing away from animal housing, and rodent control are the best preventive measures against rickettsial agents. Oral doxycycline (200 mg each week) has been effective in preventing clinical disease due to a similar pathogen, *Rickettsia tsutsugamushi*, which is endemic to rural Asia [191]; this regimen may also be effective in preventing symptoms in infections with rickettsiae endemic to the Horn of Africa [188].

### Brucellosis

Brucellosis is a common infectious disease in East Africa [185, 192] and is likely to be a significant problem among the Somali people, although epidemiological data on its prevalence are lacking. The culture of the nomadic Somali people includes a close association with domestic animals and the consumption of their raw milk, which, as has already been mentioned, may make up >90% of the men's diet. (In fact, it has been said that the name Somali comes from the words *so*, meaning "go", and *mal*, meaning "milk" [193].) Since consumption of infected raw milk is an effective means of transmission of brucellosis, the disease is likely to be endemic.

Brucellosis is also transmitted through close contact with animals infected with the causative organisms, chiefly *Brucella melitensis* and *Brucella abortus* [194, 195]. The reservoirs of disease in Africa are goats, sheep, and cattle and their blood, tissue, aborted fetuses, and placentas, which can be especially infectious. Airborne transmission can take place during the slaughter of animals and during their handling in pens or corrals.

After an incubation period of 2–3 weeks, the acute clinical manifestations of human brucellosis include fever, chills, arthralgia, myalgia, hepatosplenomegaly, and general malaise [196]. Complications of the acute infection may lead to arthritis, spondylitis, osteomyelitis, orchitis, hepatitis, and endocarditis. The diagnosis is made by isolation of the organism from the blood (15%–40% of cases) or by documentation

of serological evidence of infection [197]. With prior use of antibiotics or subacute infection, bone marrow cultures are more sensitive than blood cultures.

Many therapeutic regimens have been used, but effective treatment is characterized by the use of multiple drugs and a prolonged course. The recommended regimens include tetracycline (500 mg four times daily) or doxycycline (100 mg twice daily) for 4–6 weeks. Streptomycin (1 g im daily for 2 weeks) should be given in combination with tetracycline or doxycycline [198]. Another regimen with strong proponents is the combination of doxycycline and rifampin (900 mg daily) for 30 days; for children <8 years old, doxycycline is replaced with TMP-SMZ [199]. Others investigators have recommended gentamicin in combination with TMP-SMZ for children [200].

Good personal hygiene during the handling of animals and the boiling of their milk will prevent brucellosis. In addition, a number of attenuated vaccines can prevent infection in domestic animals, including the Rev-1 strain of *B. melitensis* and strain 19 of *B. abortus*.

### Relapsing Fever

Relapsing fever is an acute febrile illness caused by a variety of *Borrelia* species. In the Horn of Africa, two major forms have been reported. Tick-borne relapsing fever is caused by *Borrelia* species for which rodents and other mammals are generally the reservoirs and ticks of the genus *Ornithodoros* are the vectors. In East Africa, the common vector of *Borrelia duttonii* is *Ornithodoros moubata*, but humans (rather than other mammals) serve as the reservoir. The spirochete is present in both saliva and feces of ticks and is transmitted transovarially from generation to generation. These ticks feed primarily at night via a relatively painless bite lasting for 5–20 minutes and often not noticed by the victim [201]. Louse-borne relapsing fever is caused by *Borrelia recurrentis*, for which humans serve as the reservoir and the human body louse as the vector. The organism is not transmitted via louse saliva or excrement but rather when the infected individual crushes a louse over an excoriated area.

Tick-borne relapsing fever is endemic in Somalia [202–204]. In 1935, an epidemic of 76 cases occurred among Italian soldiers in what is now southern Somalia. The soldiers were living in mud-floored huts infested with ticks [202]. Louse-borne relapsing fever has not been reported among Somalis. The disease is endemic to the highland plains of Ethiopia, and cases have recently been reported among Ethiopian refugees living in northern Somalia [189]. In a 4-month period of 1991, a regional referral hospital in northern Ethiopia was the site of an epidemic of 104 cases of louse-borne relapsing fever among 370 admissions. The fatality rate for hospitalized cases was 3.8% [205].

The clinical symptoms of the two relapsing fevers are similar except that louse-borne disease is often more severe and, if untreated, is fatal in up to 40% of cases in epidemic situa-

tions. The symptoms of louse-borne infection include fever, headache, fatigue, hepatic tenderness, generalized myalgias, and sometimes a truncal petechial rash. The patient undergoes repeated cycles in which febrile episodes alternate with afebrile periods when the spirochetes are absent from the peripheral blood. The relapses are related to antigenic variation of the spirochete that results in a return of symptoms followed by a new specific antibody response and clinical recovery. In louse-borne relapsing fever, the initial febrile episode of ~6 days is followed by an intervening afebrile period of ~9 days; then there is a single relapse. For tick-borne disease, the initial febrile episode is usually only 3 days in duration and the average number of relapses is 3, however, up to 10 relapses have been reported [201]. The diagnosis is made by identification of the spirochete in a thin blood smear. Borreliæ are large (5–40 µm in length) and are easily identified with Wright's or Giemsa stain during a paroxysm, when there may be  $10^6$ – $10^8$  spirochetes/mL of blood (several organisms per high-power field). Treatment with a single 500-mg oral dose of tetracycline or erythromycin has been shown to be effective [206].

A Jarisch-Herxheimer reaction, with rigors, myalgia, severe headache, hypotension, and decreased platelet and leukocyte counts, may occur with therapy. The reaction is more common in louse-borne disease but does develop occasionally in tick-borne disease. The best approach is to be aware of the possibility of a reaction and of the need for supportive care should it occur [207]. Pretreatment with acetaminophen and hydrocortisone does not prevent rigors, has only a modest impact on other parameters, and is not routinely used [208].

Relapsing fever is prevented by eradication of the tick and louse vectors. Although a 1956 publication claimed that tick-borne relapsing fever had been eradicated from northern Somalia by the spraying of dwelling places with insecticide [204], it is unlikely that these efforts have continued to the present. The efficacy of human delousing programs may be hampered by crowding and poor hygiene, especially among displaced populations [205].

### Filariasis

Although there are few current published data on the prevalence of bancroftian filariasis in Somalia, the disease is endemic [192]. The prevalence of infection may be similar to that in neighboring Kenya, where pockets of the disease have been documented [209]. A 1977 report described a study of two villages in the Coast Province of Kenya; microfilariae were detected in the peripheral blood of 22% of inhabitants, and rates of male hydrocele were >15% [210]. Lower socioeconomic housing was associated with a higher prevalence.

In Somalia, filariasis is caused by the nematode *Wuchereria bancrofti* and is transmitted by mosquitoes. The incubation period between infection and clinical disease is 3.5–12

months. The first clinical manifestation of disease is most often an acute inflammatory lesion of the lymphatics, with erythema, edema, and pain. This presentation was almost universal in U.S. military personnel infected during World War II; the initial lesions were most commonly on the genitalia (55.5%), arms (38.3%), or legs (3.6%) [211]. Localized lymphadenitis and lymphangitis accompanied by fever, malaise, sweats, headache, myalgia, and arthralgia are typical, as are filarial orchitis and epididymitis. Lymphatic inflammation increases over the first 24 hours and may lead to peripheral edema. Most distinctive is the occurrence of lymphangitis in a retrograde fashion—unlike the ascending lymphangitis of bacterial infections [212]. Bacteria have not been implicated in these episodes of acute lymphangitis, nor have antibiotics proven efficacious in their treatment [213].

Recurrences are quite variable. Some patients have one or a few episodes of lymphangitis in their lifetime, while others have several attacks each month. The late chronic obstructive changes follow repeated attacks of acute inflammation and rarely develop until at least 10 years after the first exposure. The most common late manifestations are hydrocele, lymphedema, and elephantiasis—most commonly of the legs, scrotum, and arms and less commonly involving the breasts, penis, and labia.

Filariasis is best diagnosed by the identification of microfilariae in the peripheral blood. Because of the nocturnal periodicity of the microfilariae, blood samples are best obtained at night [214]. If Giemsa- or Field's-stained films of blood are negative, diagnostic sensitivity can be enhanced by concentration techniques such as the use of a Nucleopore membrane filter with a pore diameter of 3–5 µm (Nucleopore Corp., Pleasanton, CA). Lymph node biopsy should be avoided because the adult microfilariae live in the afferent lymphatic vessels, not in the lymph nodes. Biopsy may only compromise lymphatic drainage further. Although the available serological tests lack specificity, they are highly sensitive [214] and are especially valuable in cases with the low microfilarial burdens that would be expected among recently infected relief workers. Treatment generally consists of a single oral dose of ivermectin (100 µg/kg of body weight) [215–217]. The best preventive measures are the screening of sleeping quarters and the use of insect repellent.

### Echinococcosis (Hydatid Disease)

Echinococcosis is a zoonosis caused by ingestion of the eggs of *Echinococcus granulosus* and the subsequent development of the larvae into unilocular hydatid cysts. The presence of echinococcal infection in Somalia was documented by a small-scale serological investigation in the country's southern region; 67% of cattle, 50% of goats, 31% of camels, and 4.2% of humans had evidence of prior infection [2]. It has been estimated that 1.8% of nomadic pastoralists

in northern Kenya have ultrasonographic evidence of hydatid disease [218].

The adult cestode resides in the intestine of the definitive host: dogs and other canines. The eggs are eliminated in the feces and infect herbivores and humans when ingested from the contaminated environment. The oncospheres released from the eggs penetrate the bloodstream and lodge in the liver, lungs, and other viscera, where they grow at an annual rate of 1–5 cm. Most infected humans are asymptomatic. However, as the cysts enlarge, they may produce a variety of symptoms depending on their location, size, and number. Clinical manifestations include hepatomegaly, obstructive jaundice, abdominal pain, cough, and hemoptysis.

Persons at greatest risk of infection are shepherds in close contact with their work dogs. Hydatid disease is especially common among the Turkana tribe of northwestern Kenya, who place a very high value on their dogs and maintain intimate contact with them. All slaughtering of animals is done at home, and any hydatid cysts that are found are fed to the dogs [219]. The women have especially close contact with the dogs; in this desert region where water is scarce, these animals are used to clean the feces from the perianal region of children with diarrhea [220]. Rates of echinococcosis among the Turkana are the highest in the world, reaching 6%–9% [221]. The relatively low prevalence of hydatid disease among nomadic Somali herdsmen is most likely related to the Islamic view that dogs are unclean animals and the resulting lack of a close association with canines.

The diagnosis of echinococcosis is suggested by cystic lesions (noted on sonography or radiography) in a patient with a history of residence or travel in an endemic area [222]. This diagnosis is supported by serological evidence of infection [223]. Infection in humans has been treated with albendazole, but the effectiveness of this drug is controversial [224]. Human disease is best prevented by control of stray canine populations and use of praziquantel to eliminate the adult worms from their canine hosts.

### Histoplasmosis

Histoplasmosis is a fungal disease with a cosmopolitan distribution. Two distinct clinical forms are endemic in Africa: the classic form due to *Histoplasma capsulatum* var *capsulatum* and the African form due to *H. capsulatum* var *duboisii* [225]. The classic form has been well described and is familiar to Western physicians [226, 227]. In culture, the isolate from the African form is identical to that from the classic form; in tissue, the yeast is much larger in the African form (diameter, 8–15  $\mu$ m vs. 2–4  $\mu$ m). The most common clinical manifestations of African histoplasmosis are cutaneous, with papules that may progress to nodules, abscesses, and ulcers. Bone lesions, including circumscribed lytic lesions of the long bones, ribs, and skull, are also common [228].

A skin-test survey using histoplasmin was conducted

among 405 Somalis hospitalized in Mogadishu; 25.7% of these patients had positive skin-test results. Among those tested, 19.6% of female patients and 29.4% of male patients were reactive, with a significantly higher rate of positivity in the moister, rivered southern area (39.5%) than in the more arid northern area (12%) [229]. Unfortunately, the skin test does not differentiate between the two forms of histoplasmosis. Moreover, since clinical data are not available to validate the survey, the results—and thus the potential impact of histoplasmosis—remain subject to speculation.

### Laboratory Facilities in Somalia

Even before the current period of social upheaval, diagnostic laboratory facilities in Somalia were minimal. Almost all epidemiological studies were conducted with imported reagents and equipment. The broad spectrum of infectious agents endemic to Somalia that can be treated or prevented if appropriate diagnostic and surveillance capabilities are available makes laboratory support a critical component of the safe completion of ORH.

Fortunately, at the beginning of ODSS, the U.S. Navy developed the Navy Forward Laboratory as a theater-wide reference center for the diagnosis of endemic disease. This laboratory was composed of physicians and scientists from the Naval Medical Research Institute in Bethesda, Maryland, and Navy Medical Research Unit #3 in Cairo, Egypt, who had field experience in the Middle East and Africa, including extensive experience in transporting, constructing, and successfully operating laboratories under primitive conditions. Additional support was provided by the U.S. Army Medical Research Institute of Infectious Disease at Fort Detrick, Maryland, in the form of procedures and reagents for serological tests. This reference laboratory made it possible to determine the cause of the diarrheal epidemic early in ODSS, to identify the first operational outbreak of Norwalk virus infection, and to report on the impact of parasitic and arboviral diseases [18, 156, 177].

For ORH, this approach to the provision of sophisticated laboratory capabilities where none exist has been expanded and refined. A Joint Forward Laboratory was developed and is operating in Somalia to provide rapid diagnostic capabilities for infectious diseases in the field in conjunction with disease surveillance. This combined Army-Navy approach should optimize the identification, control, and prevention of the endemic infectious diseases of Somalia.

This new use of the clinical and diagnostic resources of the U.S. Army's and Navy's overseas laboratories seems a natural accompaniment to the developing role of the U.S. military in humanitarian relief operations. Such research facilities currently exist in Indonesia, Thailand, Peru, Kenya, Egypt, and Brazil.

## Conclusions

The endemic infectious diseases of Somalia have already had—and will continue to have—a profound impact on the local population. Diarrhea, measles, and malaria will continue to be major causes of mortality in displaced populations. The impact of these diseases on military and civilian humanitarians is more unpredictable and will depend largely on the degree of adherence to preventive measures and on the local prevalence of vectors and pathogens.

When the drought has ended and order has been restored to Somalia, the problems will not be over; the loss of livestock by the nomads will be extensive, and the available alternatives in this harsh land are minimal. A previous drought in 1974 forced 250,000 nomadic and seminomadic persons into emergency relief camps and compelled another 750,000 to seek relief outside the camps [3]. The widespread loss of animals (more than 5 million sheep and goats and 500,000 camels) prevented the resumption of a nomadic existence for many. The government attempted to relocate almost half of the displaced persons as farmers in settlements along the rivers in the south or as fishermen along the coast. However, the traditional nomadic contempt for farming and especially for fishing complicated resettlement, and many of those involved eventually returned to a marginal nomadic existence.

In short, the combination of ancient clan rivalries, an absence of natural resources, a lack of both industrial and medical infrastructure, and the recurrence of droughts in this semi-arid land make it likely that the current relief efforts of ORH are just one step in resolving the difficulties of Somalia.

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13. ABSTRACT (Maximum 200 words)  The endemic infectious diseases of Somalia have already had - and will continue to have - a profound impact on the local population. Diarrhea, measles, and malaria will continue to be major causes of mortality in displaced populations. The impact of these diseases on military; and civilian humanitarians is more unpredictable and will depend largely on the degree of adherence to preventive measures and on the local prevalence of vectors and pathogens. When the drought has ended and order has been restored to Somalia, the problems will not be over; the loss of live-stock by the nomads will be extensive, and the available alternatives in this harsh land are minimal. A previous drought in 1974 forced 250,000 nomadic and seminomadic persons into emergency relief camps and compelled another 750,000 to seek relief outside the camps. The widespread loss of animals (more than 5 million sheep and goats and 500,000 camels) prevented the resumption of a nomadic existence for many. The government attempted to relocate almost half of the displaced persons as farmers in settlement along the rivers in the south or as fishermen along the coast. However, the traditional nomadic contempt for farming and especially for fishing complicated resettlement, and many of those involved eventually returned to a marginal nomadic existence. In short, the combination of ancient clan rivalries, and absence of natural resources, a lack of both industrial and medical infrastructure, and the recurrence of droughts in this semiarid land make it likely that the current relief efforts of ORH are just one step in resolving the difficulties of Somalia.				
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