



LIBRARY, INTERNATIONAL REFERENCE  
CENTRE FOR COMMUNITY WATER SUPPLY  
AND SANITATION (IRC)

P.O. Box 9300, 2309 AD The Hague

Tel. (070) 814911 ext. 141/142

RN: ish 1593

LO: 245.3 83 DR

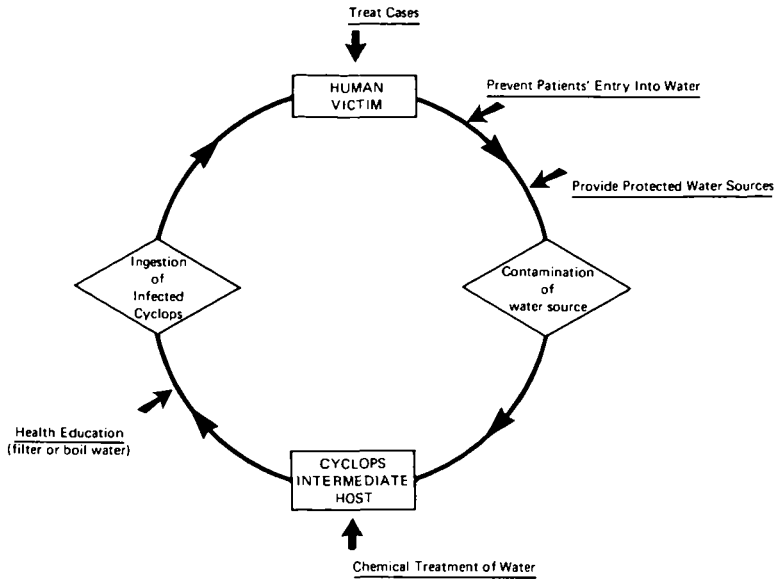


FIGURE 1. Life cycle of *Dracunculus medinensis* and potential control measures.



FIGURE 2. Adult female guinea worm emerging from a patient's thigh.

stage larvae which transmit the infection again to humans. The moving *Cyclops* are barely visible to the naked eye if water containing them is held up to a light.

When humans drink water containing *Cyclops* that include the infective third-stage larvae of the parasite, gastric juices kill the *Cyclops* and liberate the larvae. The larvae penetrate the wall of the digestive tract and migrate into the abdominal or thoracic cavity. Male and female worms mate at about three months of age, after which the males die. The females continue to grow, and by the time the adults migrate to the skin surface of a lower extremity, their body consists almost entirely of a distended uterus, packed with immature larvae.

Individual worms apparently do not survive in humans much more than one year. Thus, larvae that are ingested in any given transmission season either emerge, or die before reaching maturity, during the ensuing year (1).

Probably the most important feature of the life cycle is that dracunculiasis is transmitted *only* by ingesting contaminated water, unlike cholera, for example, which can also be transmitted via direct fecal-oral contact or contaminated food. Moreover, it is the only waterborne infection which depends solely on this mode of transmission for its perpetuation (4).

#### *The disease*

Following the incubation period of 10–14 months, the mature female worm may present clinically as a serpiginous form just beneath the skin, a painful bleb or blister, a white cord emerging from a superficial ulcer, or at the center of a painful abscess. Worms that die in the body are absorbed or calcify, and usually cause no symptoms. Victims generally experience no signs or symptoms of the infection during the long incubation period. When the adult worm does emerge through the skin, usually several weeks must pass before it is expelled completely, because it is so long and emerges so slowly.

During the patent stage, patients are usually disabled, because emergence of the worm is often accompanied by secondary infection of the wound from which the worm leaves the body, an abscess, or arthritis, all of which are painful. These complications result in prolonged incapacitation and give dracunculiasis most of its social and economic significance (see below).

One lethal complication which is not rare in endemic areas is tetanus, which results from secondary infection of the ulcer caused by the worm. Pirame and Becquet (5) studied 211 cases of tetanus seen at Ouagadougou Hospital in Upper Volta over a two-year period, and reported that 15 (7.1 per cent) had contracted the tetanus infection in a wound caused by an emerging guinea worm(s). In a similar study at University College Hospital in Ibadan, Nigeria, guinea worm ulcers were found to be the third commonest portal of entry for tetanus (6). This complication of dracunculiasis by secondary tetanus has also been reported from the Republic of Benin (7, 8).

As many as half of all victims may show evidence of secondary infection of one type or another (1, 9). Some 2.4 per cent suffered tendon contractures in one series (10), while about 0.5 per cent of dracunculiasis victims suffer permanently frozen joints. Rarely, the adult worms may cause serious neurologic complications by migrating into or near the spinal cord (8, 11).

No matter how often individuals are infected—and many victims in endemic areas are infected year after year (12)—or how many worms may emerge at the same time (as many as 40 have been reported (1)), patients develop no effective immunity to the parasite.

## EPIDEMIOLOGY

### *Geographic distribution*

This parasitic infection occurs only in the Old World, from India to West Africa (figure 3). Muller summarized the litera-

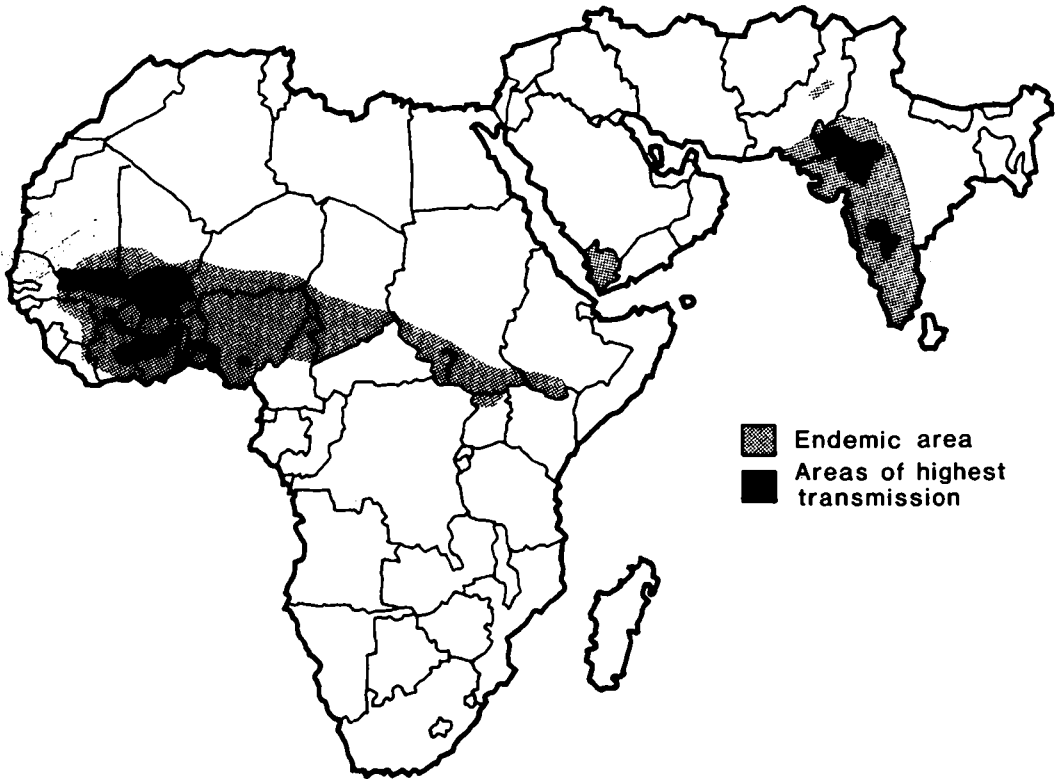


FIGURE 3. Areas where dracunculiasis still occurs. Most heavily affected regions are colored black (18, 24, 38). Question mark indicates largest endemic area for which there is no information.

ture to 1971 concerning the parasite's geographic distribution (1). Foci of infection imported into the Americas during the slave trade died out. In endemic areas, dracunculiasis is almost exclusively confined to rural villages. Niamey, Niger, appears to contain the only significant urban focus of transmission (13). Although receptive species of the *Cyclops* intermediate host are widely distributed, the distribution of dracunculiasis is surprisingly sporadic, with the intensity of infection, or even its presence, varying greatly among villages in the same area (9, 12) because of different patterns of drinking water usage.

It is not known even approximately how many persons suffer from dracunculiasis. In 1947, Stoll (14) estimated that some 48 million cases occurred annually worldwide. More recently, Duke, a parasitolo-

gist with the World Health Organization, estimated that about 10 million persons are infected each year (15). Official reports are of little help since the disease is vastly underreported. A study conducted in Togo, West Africa, in 1977 revealed that fewer than 4 per cent of the cases of dracunculiasis seen had been officially reported (16). In another study in Rajasthan, India, in 1978–1979 (17), none of the 985 patients investigated had visited a health center. Of 3641 cases examined by Nwosu and his colleagues (12) in Anambra State of Nigeria in 1978–1979, only 183 (5 per cent) had visited a hospital or health center. Reporting is especially poor for dracunculiasis because the disease tends to occur in remote rural areas, it cripples its victims, and there is no rapid therapy to serve as an incentive for patients to visit health posts. Furthermore,

in many endemic countries, dracunculiasis is not one of the officially designated "reportable diseases."

Numbers of reported cases are summarized in table 1. In West Africa, the most severely affected region, at least 50 million persons are potentially *at risk* of the infection (live in the endemic zone). Over the past decade, annual reported incidence rates of infection in individual villages in the region ranged from 2.2–58.5 per cent in Ibadan District of Oyo State in Nigeria (10); from 3–28 per cent in the Danfa area of southern Ghana (9); and from 14.1–69.1 per cent in eastern Anambra State of Nigeria (12). Assuming an average annual attack rate of 3–5 per cent, Kale in 1977 (10) conservatively estimated an annual incidence of between 150,000 and 250,000 cases in the region of Nigeria southwest of the Niger River alone (among about 5 million people at risk). Yet, less than 16,000 cases were reported from all of West Africa that year. If we assume that the 15,662 cases offi-

cially reported to West African health authorities in 1977 represent between 1 and 5 per cent of the actual number of cases which occurred, an estimated annual incidence for that region of between 1.5 and 7.5 million cases is derived. In West Africa, the most highly affected zones are found in parts of the Ivory Coast, Ghana, Togo, Benin, Nigeria, Upper Volta, Mali, and Niger (18). Issoufa et al. (20) confirmed the presence of a small endemic focus in northern Cameroon in 1975.

Dracunculiasis apparently no longer occurs south of the equator in Africa. In East Africa, an insignificant, but potentially dangerous, focus of a few imported cases was recently reported in an area of northern Kenya near the Sudanese border (21), and there are reliable unpublished reports of peak prevalences averaging 22 per cent in affected villages of Kitgum District in northern Uganda (I. Rizzo, personal communication, 1983). The largest remaining endemic area in East Africa is almost certainly the southern Sudan (22),

TABLE 1  
*Reported cases of dracunculiasis by year, 1973–1982 (18, 26)\**

	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982
Africa										
Benin		820								
Cameroon			251							
Chad						172				
Ghana	1606	1226	4052	1421	1617	1676	2489	2703	803	1382
Ivory Coast	4891	4654	6283	4971	4656	5207	6993	6712	7978	
Mali	668	786	737	542	760	1084				
Mauritania					127					
Niger				2600	3000	5560	5305			
Nigeria			1007						1693	
Senegal	334	208	65	137						
Togo		3261	1648		2617	2673	981	1749		
Upper Volta	4008	6277	1557		2885	2694	2565			
Asia										
India					6533	7615	4186	2729	5406	29906
Pakistan						250		14155 <sup>†</sup>		
Saudi Arabia	4									
Yemen	25									
Total	11536	17232	15600	9671	22195	26931	22519	28048	15880	31288

\* No data are available for blank spaces.

† Reported from Punjab Province.

but no recent statistical data on the extent of the disease in that region are available. There was a published report of cases in Ethiopia's Eritrea Province in 1969 (23). The disease probably occurs in the southwestern lowlands of Ethiopia near the border with Sudan, but there are no published reports to confirm this. The threat of large-scale movements of infected residents from endemic areas to previously nonendemic areas because of economic pressures or political upheavals, which allow the disease to become (re)established by imported cases, may become an increasing problem if the disease is not eradicated.

Iran, Saudi Arabia, and Yemen in the Middle East, are the only countries from which dracunculiasis has been reported in the last 15 years. Twenty-five cases of the disease were seen among Yemeni migrants in Saudi Arabia in 1973, when four indigenous cases were also recorded from Bisha in southwestern Saudi Arabia (2). Sabha et al. (24) examined a small persistent focus of transmission in Fars Province of southwestern Iran in 1971, where they found 145 cases in six of the 32 villages studied. They attributed the great reduction in the prevalence of dracunculiasis in Iran in recent years to physical improvements and chlorination of the cisterns, as well as to incidental destruction of *Cyclops* by DDT which was sprayed on the inside roofs of cisterns for malaria control. In 1983, the government of Iran informed the World Health Organization that despite active surveillance for cases of dracunculiasis in the residual formerly endemic zone, no cases of the disease had been found or reported in Iran in the past five years.

Since 1981, the status of the disease in India is better known than anywhere else because Indian health workers began conducting semiannual active searches to detect foci of dracunculiasis. In 1979, when less than 3000 cases were officially re-

ported in India, a questionnaire survey of Indian health officials yielded reports of an estimated 1.8 million persons at risk. When suspect areas were systematically visited in 1981, some 5.9 million Indians were found to be at risk of infection (defined as living in villages where at least one active case was found) (25, 26). An attempted complete enumeration of all cases in India in the spring of 1982 yielded a total of over 29,000 cases in India (19).

Attack rates in Indian villages are generally lower, on average, than in West Africa. In 1978-1979, Johnson and Joshi (17) surveyed several villages in western Rajasthan, the most highly affected state of India, and reported incidence rates ranging from 0.2-25.7 per cent. In a survey of 16 villages in Gujarat, Bhatt and Palan (27) attributed the relatively low attack rates to the local cultural habit of filtering water through a cloth.

Ansari and Chaudhury (28) reported in 1969 that in Pakistan, the only other state on the Indian subcontinent where dracunculiasis occurs, the infection was largely limited to five districts in the north central and southeast regions of the country.

Because transmission of the infection requires that people drink water containing *Cyclops* that are infected with *D. medinensis* larvae, dracunculiasis usually does not occur in urban areas, which tend to have piped and/or treated water, except in individuals who were infected elsewhere. It is therefore mainly a rural disease. Moreover, the *Cyclops* intermediate host flourishes only in standing, stagnant surface water, such as that found in step wells, ponds, or open water tanks. Dracunculiasis is uncommon in villages that take their drinking water from flowing streams or rivers. Reddy et al. (29) reported on a village in India's Andhra Pradesh State where 35 per cent of the population was infected as a result of using water from the town's only well, a step well. Lower caste persons in the same vil-

lage, who were forbidden to use the well, used water from the river, and remained uninfected.

#### *Seasonal occurrence*

Broadly speaking, dracunculiasis occurs in one of at least three seasonal patterns, depending on the climate (1). In semiarid areas, such as the Sahel in West Africa, where contaminative surface sources of drinking water are present only during the brief rainy season, the infections become patent, and transmission occurs, in the rainy season. Since the incubation period averages about 12 months, the transmission season remains perfectly synchronized with the annual period when the local environment is most receptive to the parasite. In wetter endemic areas where there are surface sources of water year-round, but where a distinct rainy season and dry season are still found, transmission usually occurs during the dry season, when the surface sources are scanty and most polluted. In some other areas, transmission may occur year-round with little seasonal variation (1).

Apart from the above-mentioned annual seasonal pattern, another important effect of climate on dracunculiasis is the reported result of severe droughts in endemic areas. If such droughts are sustained over one or two years, they can interrupt transmission of the parasite, since affected populations are forced to use drinking water from other than surface sources, and the infection cannot persist in affected persons more than one or two years if the individual has not been reinfected. This beneficial effect of severe drought has been reported in the Sind desert area of Pakistan in the early 1930s (30), and in the Nara region of Mali in the 1970s (31).

#### *Personal characteristics of cases*

In most endemic areas studied, dracunculiasis has been shown to be a disease of the working age population. Kale (10), re-

porting on a survey he conducted in Oyo State of Nigeria from 1971 to 1975, documented higher age-specific incidence rates among 10- to 59-year-old persons (mean of 16.6 per cent) than in older and younger age groups (mean of 8.9 per cent), with peak attack rates (30.3 per cent) in those 40-49 years of age. In Anambra State of Nigeria, Nwosu et al. (12) studied the infection in 1978 and 1979 and reported higher prevalence rates in persons between 15 and 40 years of age, the highest rates being in those aged 20-25 years. Belcher et al. (9) found highest attack rates among villagers aged 25-54 years in Ghana in 1973. In Iran, however, Sabha et al. (24) found no significant differences in attack rates between adults and children, and Scott (32) reported finding higher attack rates in children than in adults in a group he studied in Ghana in 1960.

Belcher et al. (9) also reported strikingly higher attack rates in men than in women in their 1973 study in Ghana. Lyons (33), on the other hand, reported higher attack rates in women than in men in a group he studied in Ghana in 1972. Sabha et al. (24) reported no significant difference in attack rates between male and female adults in Iran in 1973. Bhatt and Palan (27) found higher attack rates in males (27.5 per cent) than in females (18.6 per cent) in their study of 268 affected families in Gujarat State, India. Johnson and Joshi (17, 34) also reported significantly higher attack rates in males than in females in Rajasthan, as did Reddy and his colleagues (29) in Andhra Pradesh. These variations are undoubtedly related to differing age-sex water consumption patterns.

Farmers in endemic areas are particularly prone to contract the infection, apparently because they tend to satisfy the thirst brought on by their arduous physical labor by drinking comparatively large volumes of water from nearby ponds or other contaminated sources (9, 10, 12).



To my knowledge, no studies to identify the chief *contaminators* of implicated water sources, similar to research on water contact patterns in schistosomiasis for example (35, 36), have been conducted. In African or the other societies where young girls and women are responsible for collecting water for household use, they may be the group most responsible for contaminating the communal water source even where adult male farmers, as a group, are the major victims. This subject deserves careful investigation to help target appropriate educational efforts. It is conceivable, for example, that adult male farmers are the chief sources of contamination of the village's drinking water supply, because of farming, bathing, religious, or recreational use of water.

#### *Impact on affected populations*

The primary signs, symptoms, and adverse clinical effects of dracunculiasis are well known. The duration of physical disability associated with the emergence of the worm is considerable. Nwosu et al. (12) reported that victims were incapacitated for an average of three to four weeks. In Belcher et al.'s study (9), the average period of work loss in untreated adults was over five weeks, and Kale (10) reported an average duration of 100 days of incapacity from effective work. Reddy et al. (29) found that nearly three fourths of the Indian victims they studied had suffered for a month or more. According to one report of 50 African immigrants treated for dracunculiasis in a Paris hospital during a recent three-year period, 32 were hospitalized between 15 and 60 days, and nine remained in the hospital for more than 60 days (37).

Nwosu et al. (12) also reported a three-fold rise in absenteeism rates among school children during the "guinea worm season," presumably related to their own disability or because of the need to substitute for an incapacitated parent on the family farm. In areas where prevalence

rates are high, in coincidence with farming activity, substitute agricultural labor cannot be obtained because of general labor demand during peak periods of planting or harvest seasons (9, 10, 12). Although the adverse secondary effect of the disease on agricultural productivity appears to be substantial, and is one of the most important features of dracunculiasis, it has never been properly measured. Potential tertiary adverse effects on the nutrition of young children, especially in marginal subsistence economies, also need to be documented.

#### CONTROL MEASURES

##### *Provision of safe drinking water*

This mode of intervention is the most reliable and by far the most expensive. Where populations affected by dracunculiasis are provided access to safe drinking water and use it, the disease has been eliminated within one or two years. The most often cited example is that of a Nigerian town of 30,000 persons where construction of a piped water supply in the 1960s reduced the incidence of guinea worm disease from over 60 per cent to zero within two years (1). More recently, Prod'hon and Desfontaine (38) reported that a well-drilling program in the rural health sector of Dimbokro area of the Ivory Coast in the 1970s reduced the prevalence of dracunculiasis from 30 per cent to about 1 per cent. A broad strategy based mainly on providing a safe water supply and filling in of open step wells successfully eliminated dracunculiasis from the last remaining foci in the Soviet Union (Bukhara and surrounding areas) in the early 1930s (39).

Providing a safe water supply alone is often not enough. Measures must be taken to ensure that the new water sources are properly maintained (40). Without associated efforts to motivate the populations concerned, villagers may continue to use contaminated sources in preference to

protected sources because water from the contaminated site is perceived to be physically easier to collect, cheaper, or tastier (27, 41, 42).

### *Health education*

Apart from its role as an important adjunct to provision of safe water, health education can be an effective means of intervention in and of itself, and is relatively inexpensive. Urging patients with patent dracunculiasis infections not to enter drinking water sources, and persuading potential victims in endemic areas to filter or boil their drinking water, are two actions villagers themselves could take to interrupt transmission of the disease (figure 1). Akpovi et al. (43) in Nigeria recently showed health education measures to be practical. They documented changes in the health behavior of the target population and a significant reduction in dracunculiasis incidence after two years.

In a recent series of drug trials in other Nigerian communities, Kale (44) attributed a dramatic reduction in subsequent incidence of dracunculiasis to reduced contamination of the drinking water: the patients, whose wounds were dressed as part of their treatment, wished to keep their bandages dry and therefore refrained from immersing them (the treatment also shortened the duration of their infections). Over eight years, the annual incidence of infection fell from an average of about 20 per cent to zero in 16 of 17 villages studied.

### *Chemical treatment of water*

Another intervention well suited to some circumstances, for example as a temporary measure until more permanent steps can be taken, is chemical treatment of drinking water to kill *Cyclops*. Temephos (Abate), applied to open water sources at monthly intervals during the transmission season, can prevent transmission (30, 45). Sastry et al. (46) re-

ported a 97 per cent reduction in numbers of cases (from 375 to 10) of dracunculiasis in a village of about 3700 persons in Andhra Pradesh State of India within one year after they treated the village's solitary source of drinking water, a step well, with temephos in 1975. The incidence of infection declined only 11 per cent over the same period in a neighboring control village. Properly applied, temephos is colorless, tasteless, and odorless, with a wide margin of safety (47). It has been used extensively in the regional onchocerciasis control program in West Africa since 1975 (48).

### *Treatment of patients*

No anthelmintic, including mebendazole, niridazole, thiabendazole, and metronidazole, has proven effective against dracunculiasis, although some have anti-inflammatory effects. Thus, no available drug is suitable for effective mass treatment (30). Development of a prophylactic drug which would prevent ingested larvae or developing worms from maturing in infected humans would permit an additional point of intervention (figure 1). Antibiotics can help prevent or resolve secondary infections. Analgesics may be used to minimize pain associated with emergence of the worm(s).

India began a Guinea Worm Eradication Programme in 1980, aimed at eliminating dracunculiasis from India within five years (26). The strategy for the Indian program makes maximum use of the country's plans to provide safe drinking water to rural populations during the Water and Sanitation Decade. It also includes health education, active surveillance for cases, and temporary chemical treatment of contaminated water sources (24, 25, 49). Of the seven affected states in India, Tamil Nadu has apparently interrupted transmission, having undertaken concerted antidracunculiasis activities for several years before the recent national program began (19).

CHARACTERISTICS WHICH  
PERMIT ERADICATION

Table 2 compares some key characteristics of smallpox, which is the first disease of humans to be eradicated, and dracunculiasis, which should be considered a prime candidate to follow smallpox (50, 51). Both diseases are easily diagnosed. Indeed, no other infection or condition is likely to be mistaken for an emerging guinea worm. Both diseases often exhibit(ed) predictable seasonal declines in prevalence, a characteristic that was exploited to great advantage in the successful Smallpox Eradication Program (52). Although zoonotic species of *Dracunculus* occur naturally in wild vertebrates all over the world, species of the parasite other than *D. medinensis* appear to pose no threat to humans. *D. medinensis* apparently does not have a natural reservoir of infection besides the *Cyclops*-human-*Cyclops* cycle (30). Further assurance on this point is provided by the failure of the disease to reappear after it was eliminated from the Soviet Union.

Dracunculiasis is less vulnerable to eradication than smallpox because infection with the guinea worm induces no immunity, and there is no effective treat-

ment, prophylactic drug, or vaccine. Providing safe drinking water is effective, but it is expensive, takes time, and requires associated changes in human behavior. Moreover, unlike smallpox, dracunculiasis poses no epidemic threat outside of its endemic regions in the tropics, so the industrialized countries of Europe, North America, and Asia have scant national motivation, except humanitarianism, to support a global effort to eradicate dracunculiasis.

On the other hand, dracunculiasis offers the potential advantage of a naturally limited geographic range. Furthermore, dracunculiasis does not spread as readily as smallpox. The year-long incubation period of dracunculiasis is a disadvantage because infected persons cannot be identified until long after the transmission occurs, but the same characteristic provides an advantage in providing ample time to prevent continued transmission to a second or third generation of victims. Unlike smallpox, dracunculiasis is dependent on an intermediate insect vector host, which provides other possibilities for interrupting transmission.

Because providing safe drinking water, which is the most reliable way to interrupt transmission of dracunculiasis, is so

TABLE 2  
*Comparison of smallpox and dracunculiasis*

Characteristic	Dracunculiasis	Smallpox
Agent		
Low infectivity	Yes	No
Humans as sole reservoir	Yes	Yes
Dependent on intermediate vector	Yes	No
Host		
Victims easily recognized	Yes	Yes
Disease produces immunity	No	Yes
Distribution		
Limited geographic occurrence	Yes	No
Seasonal decline in prevalence	Yes	Yes
Intervention		
Stable, cheap, effective vaccine or treatment	No	Yes
Modification of human behavior important	Yes	No

difficult and expensive, the International Drinking Water Supply and Sanitation Decade represents the best opportunity to eradicate dracunculiasis. Although some additional dracunculiasis-specific measures are needed to ensure eradication of the disease, the Decade is already committed to doing the hard part, namely, providing safe drinking water. The benefit which would accrue from eliminating dracunculiasis adds considerably to the health benefits of providing safe drinking water to areas without dracunculiasis (53). Moreover, having such a visible health benefit directly related to development would be a boon to backers of the Decade—a boon which no other water-associated disease could provide. Also, since endemic areas comprise only a small fraction (less than 10 per cent) of the total unserved rural population targeted for provision of safe drinking water during the Decade (54), the dramatic subgoal of eradicating dracunculiasis is still feasible even if the overall goals of the Decade are not met.

#### CONCLUSIONS AND SUMMARY

As an incapacitating disease which has a direct negative effect on the self-sufficiency of rural populations in parts of Asia and Africa, dracunculiasis is a serious, but neglected, hindrance to economic development. It is the only communicable disease that is transmitted solely by drinking contaminated water. Several intervention measures have been shown to be effective in reducing or interrupting transmission, the most effective of which is provision of safe drinking water. Its vulnerability to well planned control measures has been demonstrated in India, the Ivory Coast, Nigeria, the Soviet Union, and elsewhere. The International Drinking Water Supply and Sanitation Decade (1981–1990) presents an unparalleled opportunity to eradicate dracunculiasis, and linking the Decade and an effort to eradicate dracunculiasis would be mutually

beneficial. Additional epidemiologic studies to document further the economic impact of the disease on affected populations, the changes in incidence which result when effective interventions are made, and the role of subgroups in affected villages as transmitters of the infection, would be very useful in the struggle to eradicate another scourge of mankind.

#### REFERENCES

1. Muller R. *Dracunculus* and dracunculiasis. In: Dawes B, ed. *Advances in parasitology*, Vol 9. New York: Academic Press, 1971:73-151.
2. Morsy TA, Sebai ZA. Dracunculiasis in Saudi Arabia. *J Egypt Soc Parasitol* 1975;5:103-8.
3. Gooneratne BWM. An additional historical note on the transmission of *Dracunculus medinensis*. *Trans R Soc Trop Med Hyg* 1969;63:546.
4. White GF, Bradley DJ, White AU. *Drawers of water: domestic water use in East Africa*. Chicago: University of Chicago Press, 1972.
5. Pirame Y, Becquet R. Dracunculose et tetanos: a propos de 15 observations. *Bull Soc Pathol Exot* 1969;56:469-74.
6. Lauckner TR, Rankin AM, Adi FC. Analysis of medical admissions to University College Hospital, Ibadan. *West Afr Med J* 1961;10:3.
7. Labegorre J, Pelloux H, Piacentini M, et al. Le tetanos de l'adulte a Cotonou: a propos de 353 cas. *Med Trop (Mars)* 1969;29:702-10.
8. Monteiro B, Hountondji A. Les complications graves de la dracunculose a propos d'un cas de paraplegie et d'un cas de tetanos. *Med Afr Noire* 1982;29:43-6.
9. Belcher DW, Wurapa FK, Ward WB, et al. Guinea worm in southern Ghana: its epidemiology and impact on agricultural productivity. *Am J Trop Med Hyg* 1975;24:243-9.
10. Kale OO. The clinico-epidemiological profile of guinea worm in the Ibadan district of Nigeria. *Am J Trop Med Hyg* 1977;26:208-14.
11. Mathur PPS, Dharker SR, Hiran S, et al. Lumbar extradural compression by guinea worm infestation. *Surg Neurol* 1982;17:127-9.
12. Nwosu ABC, Ifezulike EO, Anya AO. Endemic dracontiasis in Anambra State of Nigeria: geographical distribution, clinical features, epidemiology and socioeconomic impact of the disease. *Ann Trop Med Parasitol* 1982;76:187-200.
13. Organisation de Coordination et de Cooperation pour la Lutte Contre les Grande Endemies. Special: Dracunculose. *Bull OCCGE Info* 1980; No. 69 (Sep-Oct).
14. Stoll NR. This wormy world. *J Parasitol* 1947;32:1-18.
15. Duke BOL. Filariasis. World Health Organization TDR/WP/76.10. 1976:3.
16. Setodji A. La dracunculose en milieu rural Togolais: situation dans la circonscription du Haho.

- Memoire presented to Ecole des Assistants Medicaux, University du Benin, 1979.
17. Johnson S, Joshi V. Dracontiasis in western Rajasthan, India. *Trans R Soc Trop Med Hyg* 1982;76:36-40.
  18. World Health Organization. Dracunculiasis surveillance. *Wkly Epidemiol Rec* 1982;57:65-7.
  19. World Health Organization. Dracunculiasis surveillance—India. *Wkly Epidemiol Rec* 1983;58:21-3.
  20. Issoufa H, Monekosso G, Ripert C. Etude epidemiologique de la dracunculose chez les Podokwas des Monts du Mandara (Nord Cameroun). *Bull Soc Pathol Exot* 1979;72:135-44.
  21. Macpherson CNL. The existence of *Dracunculus medinensis* (Linnaeus, 1758) in Turkana, Kenya. *Trans R Soc Trop Med Hyg* 1981;75:680-1.
  22. Simmons JS, Whayne TF, Anderson GW, et al. *Global epidemiology*, Vol 2. Philadelphia: Lippincott, 1951.
  23. Ten Eyck DR. Report on an outbreak of dracunculiasis in Ethiopia. *Ehiop Med J* 1971;9:149-52.
  24. Sabha GH, Afraa F, Fardin A, et al. Studies on dracontiasis in Iran. *Am J Trop Med Hyg* 1973;22:343-7.
  25. Division of Helminthology. Operational Manual: guinea worm eradication programme in India. Delhi: National Institute of Communicable Diseases, 1982.
  26. Rao CK, Paul RC, Sharma MID, et al. Guinea worm disease in India: current status and strategy of its eradication. *J Commun Dis* 1981;13:1-7.
  27. Bhatt AN, Palan KH. Guinea-worm infection in Banaskantha District of Gujarat—some important epidemiological aspects. *Ind J Med Sci* 1978;32:1-4.
  28. Ansari MAR, Chaudhury MA. Dracontiasis: a problem of West Pakistan. *Pak J Health* 1969;19:1-16.
  29. Reddy CRRM, Narasaiah IL, Parvath G. Epidemiological studies on guinea-worm infection. *Bull WHO* 1969;40:521-9.
  30. Muller R. Guinea worm disease: epidemiology, control, and treatment. *Bull WHO* 1979;57:683-9.
  31. Prod'hon J, Ovazza L, Sellin B. Enquete sur le dracunculose dans les regions de Yelimane, Nioro du Sahel et Nara (Republique du Mali), November 1977. *Doc Techn OCCGE* 1978; No. 06.675.
  32. Scott D. An epidemiological note on guinea-worm infection in north-west Ashanti, Ghana. *Ann Trop Med Parasitol* 1960;54:32-43.
  33. Lyons GRL. Guinea worm infection in the Wa district of northwestern Ghana. *Bull WHO* 1972;47:601-10.
  34. Johnson S, Joshi V. Dracontiasis in Rajasthan. VI. Epidemiology of dracontiasis in Barmer District. *Int J Epidemiol* 1982;11:26-30.
  35. Dalton PR. A socioecological approach to the control of *Schistosoma mansoni* in Saint Lucia. *Bull WHO* 1976;54:587-95.
  36. Farooq M, Mallah MB. The behavioral pattern of social and religious water-contact activities in the Egypt-49 bilharziasis project area. *Bull WHO* 1966;35:377-87.
  37. Carme B, Duda M, Detry A, et al. La filariose de Medine (dracunculose) au decours de vacances en Afrique de l'Oueste. *Nouv Presse Med* 1981;10:2711-13.
  38. Prod'hon J, Desfontaine M. Epidemio-geographic data on the dracunculosis in French-speaking West Africa. Paper presented at Workshop on Opportunities for Control of Dracunculiasis, Washington, DC, June 16-19, 1982.
  39. Litvinov SK, Lysenko AYA. Dracunculiasis. I. History of the discovery of the intermediate host and the eradication of foci of invasion in USSR. Paper written for Workshop on Opportunities for Control of Dracunculiasis, Washington, DC, June 16-19, 1982.
  40. Brieger WR, Johnson DC, Adeniyi JD, et al. Complexities of guinea worm disease. *World Health Forum* 1982;3:216-17.
  41. Paul RC, Mathur ABL, Pal SP, et al. Guinea worm disease in selected rural areas of Durgapur District, Rajasthan. *J Commun Dis* 1981;13:207-10.
  42. Abolarin MO. Guinea worm infection in a Nigerian village. *Trop Geogr Med* 1981;33:83-8.
  43. Akpovi SU, Johnson DC, Brieger WR. Guinea worm control: testing the efficacy of health education in primary care. *Int J Health Educ* 1981;24:229-37.
  44. Kale OO. Fall in incidence of guinea worm infection in western Nigeria after periodic treatment of infected persons. *Bull WHO* 1982;60:951-7.
  45. Lyons GRL. The control of guinea worm with Abate: a trial in a village of northwest Ghana. *Bull WHO* 1973;49:215-16.
  46. Sastry SC, Jayakumar K, Lakshminarayana V, et al. Abate—its value as a cyclospide. *J Trop Med Hyg* 1978;81:156-8.
  47. Laws ER Jr, Sedlak VA, Miles JW, et al. Field study of the safety of Abate for treating potable water and observations on the effectiveness of a control programme involving both Abate and malathion. *Bull WHO* 1968;38:439-45.
  48. Walsh JF, Davies JB, LeBere R. Entomological aspects of the first five years of the Onchocerciasis Control Programme in the Volta River Basin. *Tropenmed Parasitol* 1979;30:328-44.
  49. Sharma MID. Lessons learnt from the intensified campaign against smallpox in India and their possible applicability to other health programmes with particular reference to eradication of dracunculiasis. *J Commun Dis* 1980; 12:59-64.
  50. Bourne PG. Global eradication of guinea worm. *J R Soc Med* 1982;75:1-3.
  51. Editorial. After smallpox, guineaworm? *Lancet* 1983;1:161-2.
  52. Foege WH, Millar JD, Henderson DA. Smallpox eradication in West and Central Africa. *Bull WHO* 1975;52:209-22.
  53. Biswas AK. Water for the Third World. *Foreign Affairs* 1981;60:148-66.
  54. Hopkins DR, Foege WH. Guinea worm disease. *Science* 1981;212:495.