PUBLIC HEALTH TEXT SERIES

The Guinea Worm Disease

Olajumoke A. Morenikeji

Textflow Limited
IBADAN • NIGERIA

First published November 2014

Details of cataloguing in Publication Data
are available with the National Library of Nigeria.

ISBN 978 2783 00 0

Published by
Textflow Limited
P.O. Box 7053, Secretariat 200004
6 Tunde Sowonola Close, Idi Ishin
Jericho Reservations
Ibadan, Nigeria.

Contents

Nigeria: Epidemiology and control of guinea worm disease

Guinea worm disease, dracunculiasis or dracontiasis, is a disabling, painful, debilitating, water-borne helminthic disease with multiple adverse consequences on health, agriculture, school attendance, and the overall quality of life of the affected communities, thus a matter of public health in endemic countries.

In 1986, World Health Organization (WHO) designated dracunculiasis as the next disease scheduled to be eradicated by 1995 after smallpox. This eradication deadline was not met and WHO then hoped to certify eradication by 2005. At the 2006 World Health Assembly (WHA) in Geneva, WHO discussed additional measures needed to stop transmission in all of the remaining endemic countries by the end of 2009.

There was need to study the disease and factors responsible for its persistence in different areas. This had implications for its control. These works were studies on control measures, treatment of pond water with Abate™, provision of boreholes, effectiveness of other intervention strategies, distribution and infective rates of cyclopoid copepod intermediate hosts of guinea worm in domestic water sources, the Knowledge, Attitude and Practice (KAP) of communities in the management of the disease. The KAP studies were so important because without the people's cooperation and understanding none of the control strategies or intervention efforts will have much chance to succeed.

There were several impediments to the eradication of the disease in Nigeria. These were highlighted in a series of studies conducted in several villages of south-western Nigeria; fifteen villages (1991-1998), six villages in 2004, eight villages in 2005 and ten villages in 2006.

It is noteworthy that Nigeria, which reported more cases than any other country (over 653,000 in 1988/89), reported zero case for an entire calendar year for the first time in 2009. The number of cases of dracunculiasis reported globally in 2009 (3,190) was the lowest annual number of cases reported since the eradication campaign began with approximately 3,500,000 cases. In all, eradication and control of guinea worm in Nigeria could be seen as a successful effort.

Olajumoke A. Morenikeji.

1

Origin and discovery

1.1 Clinical history

The parasite *Dracunculus medinensis* (guinea worm) causes the disease dracunculiasis or guinea worm disease. It is a disabling, painful, debilitating, water-borne helminthic disease with multiple adverse consequences on health, agriculture, school attendance, and the overall quality of life of the affected communities.

The disease was predominant among the rural communities of developing countries whose population depend for its domestic water supply on surface water, especially stagnant ponds and water holes, shallow unprotected wells, slow flowing brooks and streams (Kale, 1977).

The earliest evidence of dracunculiasis was found in ancient Egypt, where an account of how to properly extract the worm is included in the *Ebers Papyrus*, one of the oldest known collections of medical texts, dating from about 1550 BC: "The process of winding the worm around a stick – was appropriately described by the same verb used for drawing out thread during a similar type of spinning" (Hopkins and Hopkins, 1992). Some authors have even considered the guinea worm to be the "fiery serpent" that afflicted Israelites in the Sinai Desert (Numbers 21: 6) (Pilotto and Gorski, 1992).

Physical confirmation that dracunculiasis existed about 1000 BC in ancient Egypt came in the 1980s when a calcified guinea worm was found in the mummy of a thirteen-year old girl (Hopkins and Hopkins, 1992). Significantly, both lower legs had been amputated from this New Kingdom girl shortly before her death, perhaps in an unsuccessful attempt to combat a gangrenous complication of her guinea worm infection.

Guinea worm was mentioned in a myth about the Egyptian sun god, Ra, who was plagued by a worm that attacked his ankle (Hopkins and Hopkins, 1992). References to the disease were found in Greek, Roman and Arabic medical treatises throughout the centuries (Pilotto and Gorski, 1992) Galen (A.D. Ca. 130-2000) named it dracontiasis (Muller, 1971).

The medical Arab physician, Avicenna (980-1037) described the clinical syndrome in detail for the first time and called the parasite 'Medina Worm', because it was so common in Medina (Hopkins,1983). A European traveller to the coast of West Africa first called the disease "guinea worm" after the geographical region (Goonerates, 1969). The European from Basel, Switzerland, who first called the disease "guinea worm" in 1611, saw it among Africans along the West African Guinea Coast (Modern Nigeria) (Hopkins and Hopkins 1992).

The parasite's modern scientific name, *Dracunculus medinensis*, was bestowed by Linnaeus in his *System Naturae*, published in 1758. Ten years after Linnaeus first called the worm *D. medinensis*, the British naval surgeon, James Lind, after travelling in Africa, published his suspicions that the disease was transmitted by drinking water (Hopkins and Hopkins, 1992).

Scientific proof that the spectacular worm arose from drinking water contaminated by a type of water flea came only in 1871, when a young Russian scientist, Aleksey Fedchenko, published the results of his studies conducted two years earlier in Turkestan. He thus provided the first demonstration of an invertebrate host of a medically important disease of humans. His countrymen later used this knowledge to eliminate dracunculiasis from the southern reaches of the Soviet Union in the 1920s and 30s (Hopkins and Hopkins, 1992).

1.2 Characteristics and life cycle of the guinea worm

The female nematode attain enormous lengths (up to 50 to 100 cm), making it one of the largest nematodes. It has a thickness of about 1.0 mm so appear like a coarse thread forming loose coil in the subcutaneous tissue. The mature female nematode has a uterus filled with up to 3 million larvae (*Editorial*, 1983). The midget male (that is in comparison with the female has only rarely been seen (Ukoli, 1984). It was first described by Moorthy (1937) and measure 12-29 x 0.4 mm. It is characterized by four pairs of pre-anal and six pairs of post-anal papillae, a pair of sub-equal spicules and a gubernaculum.

After an incubation period which averages about 12 months (Muller, 1971), the adult female worm which lives in the connective tissue of the host migrates to a sub-cutaneous position, usually on the lower leg or foot where the head of the worm provokes the formation of a painful blister in the host (Figure 1). When the affected part of the body is bathed in water to release the burning pain or for any other reason, the blister ruptures and the worm extrudes part of the uterus releasing hundreds of thousands of sub-microscopic first stage larvae into the water (Hopkins, 1983; Pilotto and Gorski 1992). This process can be repeated a few times on subsequent re-immersion of the affected portion of the body. The adult female worm then emerges slowly through the wound created by the ruptured blister.

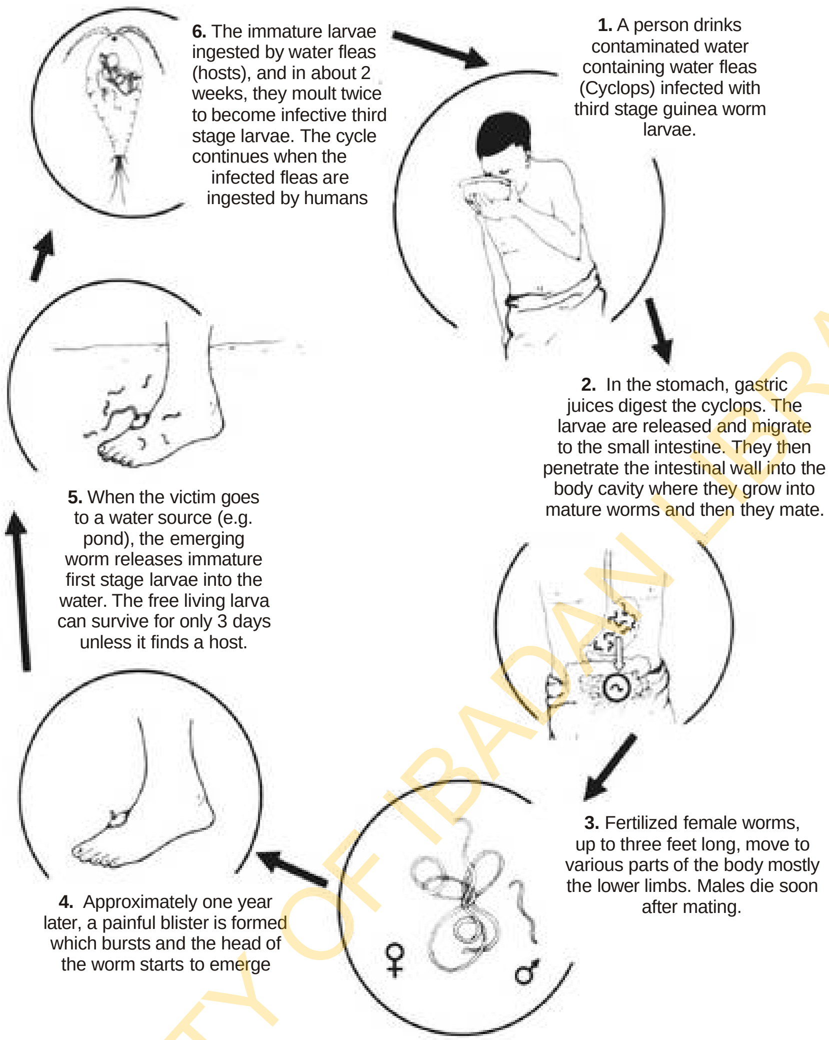

Figure 1.1: The life cycle of guinea worm.

Typically, the first stage larvae are expelled from the adult worm into ponds, step wells, stream or open water tanks which the patient has entered. These embryos have coiled bodies with rounded heads and long slender tapering tails measuring 650-750 m in length and 17-201 m in breadth (Olajide *et al* 1987).

In the water bodies, some of the larvae are ingested by Cyclops, or water fleas. The vectors are various species of Cyclops, but only those which are predatory (Muller, 1971) and live in stagnant water with a pH of 6.0 to 6.8 (Onabamiro, 1952a, 1952b) are likely to play a significant role in transmission.

Cyclops leuckarti commonly serves as the intermediate host transmission agent of *D. medinensis* in Africa and India (Ukoli, 1984). According to Onabamiro (1951, 1952 and 1954), the optimum Nigerian vector is *Thermocyclops nigerianus.* It was stated that of the thirty species and sub-species of Cyclops identified in Nigerian ponds and streams only *Thermocyclops nigerianus* was found to be naturally infected with *D. medinensis.* However, three other species, *Mesocyclops leuckarti, I. inopinus and Microcyclops varicans subaequalis,* could be successfully infected experimentally (Onabamiro, 1951a, 1952a, 1952b and 1952c).

All stages of a suitable predatory species of Cyclops can ingest guinea worm larvae, although by the time the larvae have developed to the third infective stage, the Cyclops are all likely to be adults, or at the least copepodid stage (Muller, 1985). Cyclops will only ingest active larvae, but dead larvae lying on the bottom of a container are ignored. The larvae are actively held by the mouth parts of the Cyclops and pushed into the mouth, the whole operation is less than a minute. Once in the stomach of a Cyclops the larvae undergo powerful bending movements and penetrate through the gut wall to the haemocoel in one to four hours, depending on the temperature. They go through head first, possibly aided by the action of the dorsal tooth. Larvae can retain their viability in water for about five days. Larvae moult twice in Cyclops, develop to third-stage in about twelve days but are not infective until the fourteenth day (Muller, 1985). Many larvae may be ingested by susceptible species of Cyclops but this results in a very high mortality following invasion through the stomach wall or after the first moult (Muller, 1985). Infected Cyclops were found to live for a maximum of 50 days (Muller, 1972) and it is unusual for any infected Cyclops in nature to have more than one third-stage larva (Muller, 1985).

Cyclops which contain third-stage larvae become sluggish and sink nearly to the bottom of a pond or step-well and this has been given as a partial explanation for the increase in transmission in the dry season in areas with abundant rainfall when water levels are low.

Infection is limited to tropical and sub-tropical regions because the larvae develop best between 25^0 and 30^0C and will not develop at all below 19^0C (Muller, 1979). Cyclops that contain third-stage larvae transmit the infection again to humans. Man becomes infected by drinking water containing Cyclops harboring third-stage infective larvae.

Suitable conditions for infection occur only where water for drinking is taken from stationary bodies of surface water such as ponds, but villages in endemic areas where draw-wells provide the only source of water, transmission of guinea worm is not commonly found because conditions are not suitable for the breeding of Cyclops (Muller, 1979). In a properly constructed well, there is a parapet which prevents larvae from reaching the water. Well-water which is also rich in lime salts kills embryos (larvae) (Fedchencko, 1971) and according to Ukoli (1984), people who obtain their drinking water from flowing rivers or streams are unlikely to be infected. Onabamiro (1952c) pointed out that *Dracunculus* larvae discharged from a human foot into moving water are doomed as the chances of their being swallowed by Cyclops before they are swept away by the river current are almost nil. He also pointed out that besides pond water, other sources like rain or sterile water appear to be lethal to these larvae.

Southwell and Krishner (1938) noted that "larvae (of *D. medinensis*) collected from the ulcer in sterile tap water died in about six hours". In order to keep them alive it was necessary to transfer them immediately to pond water.

Although the Cyclops are killed by the gastric juice (Hopkins, 1983), the larvae survive and are activated by the dilute hydrochloric acid in the stomach to escape from the dead Cyclops and eventually penetrate through the intestinal wall (Ukoli, 1984). The larvae then migrate into the abdominal or thoracic cavity (Hopkins, 1983). The male and female worms mate at about three months of age, after which the male dies (Hopkins, 1983). The female continues to grow, and by the time the adult migrates to the skin surface of a lower extremity, their bodies consist almost entirely of a distended uterus, packed with immature larvae.

Individual worms 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 (Muller, 1971).

There is little evidence of acquired immunity, because some individuals are being re-infected year after year (Johnson and Joshi, 1982). Since there is no carry-over of infection from one transmission season to the next, if transmission is interrupted for just one season the disease disappears from a community, unless it is re-introduced from another area (Lancet, 1983).

The interactions of humans, water and infected Cyclops perpetuate the cycle. Conversely, the exclusion of one of these will obstruct the transmission process, terminate the cycle and therefore eradicate guinea worm disease.

The most important feature of the life cycle is that dracunculiasis is transmitted only by ingesting contaminated water. It is the only water-borne infection which depends solely on this mode of transmission (Hopkins, 1983).

2

Distribution and clinical course

2.1 Global distribution

Stoll in 1947 estimated that there were 48.3 million cases of guinea worm infection annually throughout the world; 38 million in Asia, 15 million in Africa and 33 million in other parts. These according to Kale (1977), were probably overestimated, because he based his calculations on a 25 per cent attack rate in endemic areas and that true prevalence of the disease in 1977 is probably about the same as in 1947.

It was estimated in 1947 that in Africa around 3.32 million persons were infected and about 120 million were at risk of infection (Watts, 1987). In India and Pakistan, it was estimated that 28 million were at risk of infection (Hopkins, 1987). The area affected by dracunculiasis in Africa extended right across the northern part of the continent south of 10°N and in East Africa, extending almost to the equator. The infection was endemic in nineteen countries and was found in all ecological zones from the forest, through the savanna to the semi-desert Sahel to the desert (Watts, 1987). According to Watts (1987), transmission in desert area is not documented in Africa although it is known in Pakistan.

In West Africa, the most highly affected zones were found in parts of the Ivory Coast, Ghana, Togo, Benin, Upper Volta, Mali and Nigeria, so also, a small endemic focus in northern Cameroon was confirmed in 1975 by certain workers (Hopkins, 1983).

Dracunculiasis apparently no longer occur south of the equator in Africa (Hopkins, 1983). In East Africa, an insignificant but potentially dangerous focus of a few imported cases were reported in an area of northern Kenya near the Sudanese border and there were reliable unpublished reports of peak prevalence averaging 22 per cent in affected villages of Kitgum District in northern Uganda (Hopkins, 1983). Hopkins (1983) also pointed out that the largest remaining endemic area in East Africa is almost certainly the southern Sudan. There was a published report of cases in Ethiopia's Eritrea Province in 1969 resulting in 58 documented and 40 suspected human cases (Ten Eyck, 1971). Kenya shares international boundaries with Sudan, Uganda, and Ethiopia.

Iran, Saudi Arabia and Yemen in the Middle East, were the only countries from which dracunculiasis had been reported. 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 south-western Saudi Arabia (Hopkins, 1983). Studies on dracunculiasis in thirty-two villages in the southern part of Iran showed the presence of this disease in six of them and 145 cases were found in the six villages (Sahba *et al* 1973).

Attack rates in Indian villages were generally lower on average than in West Africa. Johnson and Joshi (1982) surveyed several villages in western Rajasthan, the most highly affected state of India, and reported incidence ranging from 0.2-25.7 per cent. Hopkins (1983) stated that in Pakistan, the only other state on the India sub-continent where dracunculiasis occured, the infection was largely limited to five districts in the north central and south-east regions of country. *Foci* of infection imported into the Americas during the slave trade died out. Niamey, Niger appeared to contain the significant urban focus of transmission (Hopkins, 1983).

Although receptive species of the Cyclops intermediate host were widely distributed, the distribution of dracunculiasis was surprisingly sporadic, with the intensity of infection, or even its presence, varying greatly among villages in the same area (Belcher *et al* 1975; Nwosu *et al* 1982) because of different patterns of drinking water usage. Reddy *et al* (1969) reported on a village in Indias' Andhra Pradesh State where 35 per cent of the population had infection as a result of using water from the town's only well; a step-well. Lower caste persons in the same village, who were forbidden to use the well, used water from the river, and remained uninfected.

Official reports were of little help since the disease was vastly under-reported (Hopkins, 1983). 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 (Hopkins, 1983). In another study in Rajasthan, India, in 1978-1979 (Johnson and Joshi, 1982), none of the 985 patients investigated had visited a health centre. Of 3,641 cases examined by Nwosu *et al* (1982) in Anambra State of Nigeria in 1978-1979, only 183 (5 per cent) had visited a hospital or health centre. Reporting is especially poor for dracunculiasis because the disease tends to occur in remote rural areas, it cripples its victims, and there was no rapid therapy to serve as an incentive for patients to visit health posts (Hopkins, 1983).

However figures provided to the World Health Organisation (WHO) by endemic countries which had conducted active case searches or national surveys gave a total of 614,497 cases in 1990 (Tayeh and Cairncross, 1993). Figures for the year 1991 indicated substantial reductions in the number of cases in several countries, such as Nigeria and Ghana, and increases in other countries as a result of national case searches, such as Ivory Coast, Mali, Niger and Uganda. Estimates for 1992 indicated a further fall in cases in Nigeria, Ghana, Cameroun, India and Pakistan (Tayeh and Cairncross, 1993).

Guinea worm disease was widespread in rural Nigeria. Hopkins (1988) reported that about 60 per cent of the estimated worldwide total (8 million) of active cases of the disease occured in Nigeria. Such alarming statistics led the Federal Government of Nigeria to embark on a systematic control campaign, especially in the most endemic states (Ofoezie and Adeniyi 1990). In a review of published reports from Nigeria by Kale (1977), a fairly high concentration of cases in the area of the old Western State which now comprises Ogun, Oyo, Osun, Ondo, Edo and Delta States, and part of Lagos State, with scattered *foci* in Kwara, Kogi and Imo States were indicated. Oyo State was not only one of the most endemic states in the country but, according to Kale (1977) and Edungbola (1984), it was also the most endemic of the four states (Oyo, Ogun, Ondo and Lagos) carved out of the former Western Region where the disease has long been known to be rampant (Onabamiro, 1951).

Few isolated cases from some of the northern states, which suffer some of the worst forms of draught in the Sahelian and sub-Sahelian zones of West Africa were also indicated.

2.2 Clinical course

The first signs of the disease appear when the gravid female is about to emerge from the subcutaneous tissues. In the geotropic course of the female worm towards water, the worm can be felt and seen as a tortuous varicose-like ridge on one of the extremities. Near the intended exit point the worm releases a substance in order to break through the skin. This substance causes intense burning and itching hence the 'fiery serpent' (Pilotto and Gorski, 1992). About one-third of patients have generalized allergic manifestation such as urticarial, fever, gastrointestinal disturbances and dyspnoea a few days before the appearance of a blister, lasting only a few days. On immersion of the affected part of the body in water to relieve the burning pain, the worm gradually emerges from the resultant ulcer.

Traditionally, humans have wrapped the exiting body of the worm around a small stick and extracted it slowly and painfully, centimeter by centimeter, over periods of days or weeks. If in the process, the worm breaks, a severe local reaction may ensue, with pain, swelling and inflammation of the affected extremity (Pilotto and Gorski, 1992). Sequelae that can lead to permanent disability including arthritis, synovitis and contractures of the Achilles tendons and hamstrings.

The process of releasing the larvae periodically till all the larvae are discharged and the entire worm expelled lasts from two to four weeks (Ukoli, 1984) and the ulcer heals rapidly after that. However, secondary bacterial infection arises in over half of all patients, with a spreading cellulitis along the tract of the worm. Not infrequently, worms burst in the tissues causing the formation of large abscesses.

The most serious secondary complication is tetanus, which yearly claims thousands of victims (Pilotto and Gorski, 1992). Worms that die in the body are absorbed or calcified and usually cause no symptoms (Hopkins, 1983). It is easy to diagnose dracontiasis in an endemic area especially just before the female worm begins to emerge. Then the patient experiences the intense itching and burning pain at the site of the blister as a characteristic of the disease. This is also accompanied by general urticaria. When the blister bursts, specific diagnosis can be made by finding actively moving larvae when a sample obtained from the milky fluid to which a drop of cold water has been added, is examined under the microscope (Ukoli, 1984).

Screening with X-rays has also been used. Injection of an opaque medium into the worm renders it opaque (Pilotto and Gorski, 1992). Effete and calcified worms are easily demonstrated by radiography

(Manson-Bahr, 1982). There is an intradermal test available; however, it is not very specific (Pilotto and Gorski, 1992).

2.2.1 Treatment

Pulling out guinea worm by winding them on a stick is a treatment used successfully since antiquity (Schmidt and Roberts, 1981). If cold water is applied to the worm, she will expel enough juveniles to allow about 5 cm of her body to be pulled out. The procedure is repeated once a day, complete removal requiring about three weeks (Schmidt and Roberts, 1981). This procedure is possible provided that bacterial infection or other complications have not occurred, combined with sterile dressing and acriflavin cream (Muller, 1975). Palpable pre-emergent worms can often be removed entirely by one or two small surgical incisions (Muller, 1975). However, if the worm is threaded through a tendon or deep fascia or is broken into several pieces, it may be impossible to remove completely (Schmidt and Roberts, 1981).

2.2.2 Traditional African methods

So far, no drug has been shown to have direct anthelminthic action on the worm although niridazole, thiabendazole and metronidazole have been known to reduce host tissue reaction and facilitate the removal of the female worms without pain (Oduntan, Lucas and Gilles, 1967, Lucas, Oduntan and Gilles, 1969; Padonu, 1973; and Kale, 1974, 1975). In any case, none of these drugs has a prophylactic effect, so, infection can always recur after successful treatment.

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 (Hopkins, 1983). In the absence of this, the hope for eradication then rests in prevention. Antibiotics can help prevent or resolve secondary infections and analgesics may be used to minimize pain associated with emergence of worm (Hopkins, 1983).

Where modern medicine seemed to have failed to produce a remedy, traditional African medicines have been widely acknowledged to provide varying degrees of cure (Muller, 1971). Most of the respondents with previous experience of guinea worm infection from a study by Falode (1992) in Akinyele Local Government Area of Oyo State, Nigeria, used palm oil and, or shea-butter for treatment. The use of local herbs was also practiced. Ekeh and Adeniyi (1986) also found that rubbing of palm oil on the leg ulcer resulting from guinea worm infection was widely practiced in Ibarapa District of Oyo State. Local herbs and oil mixtures offer some soothing effects to the villagers without the cost and inconveniences of clinic visits (Brieger *et al* 1981).

In Bahana District, Kwara State of Nigeria, there was no single commonly adopted method for the management of dracontiasis (Edungbola, 1983). In addition to the familiar method of extracting and winding the worm around a piece of stick or thread, the application of various substances-leaves, plant juices, palm oil and shea-butter to the lesions were observed.

The Director of the Ghana Centre for Scientific Research into Plant Medicine provided a well-documented report of how a decoction of the root of *Lenbretum mucronatum* (long prescribed by traditional herbalists) cured and brought about a complete extrusion of the worms and healing of the ulcers in two weeks following the application of sterile palm oil (Ampofo, 1977). Similarly, *Mitragyna stipulosa* also gave a 52.2 percent cure rate. Finally, he also proved that leaves of *Elaeophorbia drupifera* and *Hilleria latifolia* taken in palm soup are effective against guinea worm infection.

While technically it is true that the disease does not kill, death can result without proper treatment. In Idere, Oyo State, Nigeria, at least ten deaths due to tetanus infection of the guinea worm ulcers were reported in one of the guinea-worm infection seasons (Brieger *et al* 1982).

3

Impact of dracunculiasis on the affected population

3.1 Seasonality and water sources

In all areas, transmission of the disease was markedly seasonal and the maximum incidence coincided with the planting season, resulting in great economic hardship (Muller, 1975). The seasonality of the disease was studied by Onabamiro (1951, 1952b) and Lyons (1972). In semi-desert regions of Africa (northern Cameroun, Chad, Mauritania, Niger, northern Nigeria, Senegal, Sudan and Upper Volta) and in Pakistan, drinking water is obtained from ponds during the rainy season but from deep wells during the rest of the year when the ponds are dry. However, in the guinea savanna regions of West Africa, where rainfall exceeds 150 cm per year (Dahomey, Ghana, Guinea, Ivory Coast, southern Nigeria and Togo), there is no transmission during the rainy season when ponds turn into streams and Cyclops densities are low because of large volume and turbidity of the water. Similarly, infection is highest during the dry season in step wells in India (Muller, 1975).

In Sahel savanna region of northern West Africa, transmission occurs around the end of the rainy season when the pond water is available (Ukoli, 1984). In such cases, the incapacitating effect of the infection reaches its peak at the end of the following rainy season and this coincides with the harvesting season.

On the other hand, in the southern Guinea savannah areas, transmission is restricted to the tail end of the dry season when the ponds assumed their role as transmission sites (Ukoli, 1984). Thus, patent guinea worm disease reaches its peak in the following late dry season and early rainy season. This coincided with the main planting season (Muller, 1971 and 1979).

Apart from the stated annual seasonal patterns, another important effect of climate on dracunculiasis is the reported result of severe drought in endemic areas (Hopkins, 1983). If such droughts are sustained over one or two years, they can interrupt transmission of 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 re-infected. Hopkins (1983) stated that this beneficial effect of severe drought had been reported in the Sind desert area of Pakistan in the early 1930s and in the Nara Region of Mali in the 1970s.

3.2 Determinants of dracunculiasis

The majority of infected people (over 60 per cent in Nigeria) had only one worm emerging at any given time, the mean being 1.17 to 1.39 in Ghana (Lyons, 1972) and 1.5 worms per person in Nigeria (Kale, 1977). However, cases of multiple infections were found, up to fourteen worms having been obtained from a woman in Nigeria (Kale, 1977).

Onabamiro (1952c) showed that there were up to 152 Cyclops in five litres of water in a village pond of which about 5 per cent were infected with one or two guinea worm larvae. He estimated from this that a man in such a village would swallow between 75 and 200 infected Cyclops per year through drinking pond water. With such a high level of exposure to infection, it was surprising that the intensity of infection was not much higher with more worm per infected person than observed. Lyons (1972) found a much lower infective density of Cyclops per litre in Wa, Northern Ghana, a fact which may explain the lower incidence of the disease there.

In most endemic areas studied, dracunculiasis had been shown to be a disease of the working-age population. Kale (1977) reporting on a survey he conducted in Oyo State, Nigeria, from 1971 to 1975, documented higher age-specific incidence rates among 18-59 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 aged 40-49 years.

In Anambra State of Nigeria, Nwosu *et al* (1982) studied the infection in 1978 and 1979 and reported higher prevalence rates in persons between 15 and 40 years of age, the higher rates being in those aged 20-25 years. Belcher *et al* (1975) found higher attack rates among villagers aged 25-54 years in Ghana in 1973. In Iran however, Sahba *et al* (1973) found no significant differences in attack rates between adults and children and Scott (1960) reported higher attack rates in children than in adults in group he studied in Ghana.

The sex distribution of persons with infection also varied a great deal. Belcher *et al* (1975) reported strikingly higher attack rates in men than in women in their 1973 study in Ghana. Lyons (1972) on the other hand, reported higher attack rates in women than in men in a group he studied in Ghana. Sahba *et al* (1973) reported no significant difference in attack rates between male and female adults in Iran. Bhatt and Palan (1978) 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 (1982), also reported significant higher attack rates in male than in females in Rajasthan, as did Reddy *et al* (1969) in Andhra Pradesh. These variations are undoubtedly related to differing age-sex water consumption patterns.

Water consumption pattern was used by Belcher *et al* (1975) to explain differences in age and sex distribution in infection. They stated that the heavy physical exertion in the farms from February to April in preparation for the planting season and in June to September (weeding and harvesting) which are thirst-inducing activities may result in the consumption of large quantities of water from ponds near the farms. This may account for the greater exposure and hence prevalence of the disease among the older adults (18-14 years) who assist on the farms (Kale, 1977).

A remarkable feature of the disease is that in a community some individuals are susceptible to repeated infection year after year while others equally exposed; escape infection. Scott (1968) explained differences in susceptibility to be due to the level of acidity in the stomach, those with high gastric acidity being more protected because the larvae could not be killed in the stomach, while those with low gastric acidity being more prone to infection. However, Gilles and Ball, (1964) found no significant relationship between infection and gastric acid secretion and Muller (1971) found that the larvae were very sensitive to low concentration of acid, 68 per cent of emerged larvae kept in 0.05% HCl being dead within 1.5-3.5 hours.

Fedchenko (1971) pointed out that most often the parasite appear in the lower extremities and Kale (1977) in summarizing sites of lesions produced on the body by emerging guinea worm from his study and other studies found that of all lesions, 93.9% occurred on the lower limbs with over 58% appearing on the ankles and feet. This pronounced propensity for the most dependant parts of the body is believed to be a biological phenomenon that ensures the survival and perpetuation of *D. medinensis* as the predilection sites coincides with those most frequently in contact with water where the obligatory intermediate host, the Cyclops, is likely to be encountered by the embryos that are discharged on immersion in water (Kale, 1977).

The greater the number of worms emerging at any given time; the more likely is the victim to be incapacitated, but the factors that most closely correlated with the severest forms and largest number of those incapacitated was the location of the lesions (Kale, 1977). In his study, the proportion of persons with lesions on ankles and feet who were disabled was significantly higher than for other sites. The duration of physical disability associated with the emergence of the worms is considerable. Nwosu *et al* (1982) reported that victims were incapacitated for an average of three to four weeks. The period of disability (incapacitation or prostration) in untreated cases lasts five weeks (Belcher *et al* 1975) to ten weeks (Kale, 1977).

A report of fifty African immigrants treated for dracunculiasis in a Paris hospital during a three-year period showed that 32 were hospitalized between 15 and 60 days, and nine remained in the hospital for more than 60 days (Hopkins, 1983).

Edungbola (1983) noted that the excessive and prolonged guinea worm incapacitation were largely due to multiple infections, the anatomical location of the infection and secondary bacterial infection resulting from negligence, ignorance and the un-hygienic procedures of local management.

3.3 Social and economic effects of the disease

Temporary disability from dracunculiasis during the emergence of the worm (with the individual unable to leave his or her bed) has been assessed by Smith *et al* (1989) in a comprehensive longitudinal study in Nigeria, it was found that 58% of all cases resulted in severe disability lasting for approximately one month. This tend to occur during the peak of agricultural activities, usually during the planting season in the Indian studies (Muller, 1979) and during the peak of yam and rice harvest and the period of preparation for the planting season in Imo State, Nigeria (Smith *et al* 1989). This can have a very serious impact on agricultural production (Belcher *et al* 1975; Nwosu *et al* 1982; Brieger *et al* 1989; Brieger and Guyer, 1990) and on the availability of food in the household and therefore on the nutritional status of all the family members, especially the young children (Tayeh and Cairncross, 1993). A United Nations Children Fund (UNICEF) sponsored-study in a rice-growing region in south-eastern Nigeria estimated that guinea worm disease caused losses totaling about 20 million US dollars per year. Ondo State, which produces 60% of Nigeria's cocoa is one of the states' most heavily infected with guinea worm.

Sokoto State, that had over 600 infected villages, is the sorghum and millet cultivation belt. Sokoto, Katsina , Kano, Bauchi, all important cotton-growing states in Nigeria, had high rates of guinea worm disease as did Anambra State, a major rice-producer (Ransome-Kuti, 1991).

In areas where prevalence rates were high and in coincidence with farming activity, substitute agricultural labour were not available because of general labour demand during peak periods of planting or harvest seasons (Belcher *et al* 1975; Kale, 1977, Nwosu *et al* 1982). Quite apart from the direct loss, the prostrated farmers were incapable of increased agricultural production or of innovating improved farming technology (Belcher *et al* 1975).

Infection of the mother with dracunculiasis is expected to have an immediate impact on her children such as keeping them clean, reduction in actual time of feeding the child and preparation of meals (Watts *et al* 1989; Edungbola and Watts, 1990). Furthermore, the ability of infected mothers to provide care for their children, to fetch water, collect firewood, breast-feed and take children for immunization will be seriously affected and this will have serious implications on their health.

Guinea worm disease also interferes significantly with school attendance by children. In a study by Muller (1978) of a village in western Nigeria, 18 per cent of the children were absent from school for up to five weeks. In another study by Ekeh and Adeniyi, (1986) most of the absenteenism in many schools was ascribed to an outbreak of guinea worm infection. Nwosu *et al* (1982) 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 addition to the educational, agricultural, family health and economic impacts there were also personal health issues. Permanent disability occured in about 5,000 of Nigerian citizens with the disease each year. The disease was also associated with tetanus (Ransome Kuti, 1991). There was also a high rate of secondary infections associated with it which contributes to the severity, duration and cost of the disease. Infected persons could not participate in the village activities such as social gathering, dancing, funerals, hunting or going to the market (Tayeh and Cairncross, 1993).

3.4 Measures for prevention, control and eradication

Guinea worm disease was targeted for elimination in several countries, with the hope of global eradication before the end of the 1990s. It had been stated that among communi-cable diseases, dracunculiasis is the only one that is transmitted by drinking contaminated water. This means that it cannot be acquired by contact with an infected person (like measles), nor is it carried by a flying insect vector (like malaria), nor does it exist in the soil as other parasites. The solution then is simple: provide safe drinking water and the disease will disappear. It is a unique candidate for global eradication which, if achieved, would make it only the second disease – the other was smallpox (Bourne, 1982).

Global initiatives to eradicate dracunculiasis were given impetus by resolutions adopted in April 1981 and November 1987 by the Steering Committee of the International Drinking Water Supply and Sanitation Decade (1981-1990) (Nwobi, 1991). The committee called for the elimination of dracunculiasis in each country with endemic dracunculiasis. In 1986 the World Health Organization (WHO) designated dracunculiasis as the next disease scheduled to be eradicated (by 1995) after small pox (Hopkins and Ruiz-Tiben, 1995). The policy basis for this eradication effort was firmly established in resolutions by the African Regional Committee of the World Health Organization (WHO) in September 1988 (AFR/RC 38/R13), the executive board of the United Nations Children's Fund (UNICEF) in April 1989 and 42nd World Health Assembly in May 1989 (WHA 42.29) (Hopkins and Ruiz-Tiben, 1990).

The global eradication of dracunculiasis entered its final year (1995) with dramatic accomplishments. Globally, cases were reduced from an estimated 3.5 million in 1986 to less than 100,000 in 1994 (excluding Sudan), the number of endemic villages were reduced from over 23,000 at the beginning of 1993 to less than 10,000 at the beginning of 1995, and nearly all the remaining known endemic villages had one or more control measures in place (CDC, 1994).

Guinea worm eradication efforts in Nigeria began in 1985 with the hosting of the first national conference on dracunculiasis. In 1988, Minister of Health, Professor Olikoye Ransome-Kuti, on behalf of the Federal Government of Nigeria (FGN), signed the memorandum of understanding for the eradication of dracunculiasis in Nigeria with Jimmy Carter, Chairman, Global 2000 and The 39th President of United States of America. In May 1988, the Federal Republic of Nigeria, at its National Council on Health, made dracunculiasis a reportable disease, established the Nigerian Guinea Worm Eradication Programme (NIGEP) and mandated its National Task Force (NTF) to delineate the spread of the disease, describe the national endemicity pattern and formulate intervention strategies.

In pursuance of this mandate, NIGEP organized the first nationwide village-by-village active search for cases of dracunculiasis during the period of August 1988 through January 1989, in 1988, this search, the first of its kind in West Africa (Nwobi, 1991), found over 650,000 cases of guinea worm in about 6,000 villages of the twenty-one states and the FCT (Ransome-Kuti, 1991). Only Akwa Ibom State was guinea-worm-free. Thus, Nigeria with a population of more than 100 million (1991), had an estimated 800,000 cases of guinea worm annually and an estimated population at risk of more than several million people; making it the most highly endemic country in the world (Nwobi, 1991).

The objective of the Nigerian Guinea Worm Eradication Programme (NIGEP) was to eradicate guinea worm in Nigeria by the year 1995. To this end, two main strategies were in use: prevention of guinea worm transmission through health education plus safe water supply and case management of guinea worm disease with emphasis on the need for patient to avoid contact with sources of drinking contaminated water. The major intervention for carrying out these strategies were distribution and use of mono-filament nylon and cloth filters, protected sanitary wells and boreholes, and application of chemicals (Abate™) to destroy Cyclops in water sources. The interventions were supported with a strong component of health education.

In spite of the history of guinea worm disease in Nigeria, few control efforts had been made before NIGEP, except for some individual efforts mainly among university-based researchers (Ewoigbokhan and Rodman, 1993).

The eradication programme brought about substantial declines in the reported incidence of dracunculiasis in Nigeria. From the first case search in 1987-1988 to the Monthly Surveillance Case Reports of 1993, there was a reduction of 88% (Monthly Surveillance Report, January-December, 1993). With a reduction of about 200,000 cases between 1989 and 1990, Nigeria restored about 18.2 million person-days to agricultural production, and 6.2 million person-days to school attendance annually. It also doubled the *per capita* income of at least 66,000 women, and prevented the permanent crippling of over 1,000 persons each year (Hopkins, 1991).

This recorded substantial declines in the reported incidence of dracunoculiasis in Nigeria was possible due to the intensive and extensive efforts of NIGEP and all supporting agencies like United Nations Children's Fund (UNICEF), Japanese International Agency (JICA), United Nations Development Programme (UNDP)/World Bank, Canadian International Development Agency (CIDA), Canadian University Service Overseas (CUSO), World Health Organization (WHO), British High Commission, Royal Nertherlands Embassy, Global 2000, Leventis Foundation (Nigeria), the European Union, United States Embassy and other Non-Governmental Organization (NGO). Financial support for the searches' schemes came from cost-sharing by local governments, states and federal governments, Global 2000, UNICEF and other international or private donors. Substantial in-kind support was provided by villagers themselves, other levels of government and non-governmental organizations (Edungbola *et al* 1992).

At the second National Conference on Dracunculiasis, the Federal Government directed the Directorate of Foods, Roads and Rural Infrastructure (DFRII), which was responsible for providing water to rural communities, to use the presence of guinea worm disease as the primary criterion for targeting water supply such as hand-dug wells and boreholes. It also enjoined other agencies involved in water supply projects in Nigeria such as UNICEF, UNDP/World Bank to do the same, making Nigeria the first African nation to use presence of guinea worm as a criterion for prioritizing the supply of safe drinking water.

By 1991, DFRII had provided over 5,000 water points in 20 states in its Phase I Programme, UNDP had also provided water points in one Local Government Area in each of these states: 100 water points in Plateau State, 100 in Bauchi State, 100 in Benue State and 100 water points in Abuja FCT (estimated value was $60,000). Japanese International Agency (JICA) had provided 50 boreholes in Anambra State (estimated value was $3 million) and in Niger State (estimated value was $4 million). The Canadian Government also committed a large sum of money for the provision of water points in Ondo State.

UNICEF assisted Nigeria guinea worm disease control since the early 1980s through water and sanitation projects and through Primary Health Care (PHC) programmes. The programme provided improved water, sanitation, and health education facilities to 1.5 million people at a cost of 50 million US dollars out of which UNICEF contributed US $21.5 million (Laubjerg, 1991).

The 1991-1995 country programme of cooperation between UNICEF and the Government of Nigeria was directed at four main areas of cooperation: Primary Health Care, Water Sanitation, Education, and Household Food Security included U.S $7 million specifically for guinea worm eradication (FGN/UNICEF Master Plan of Operations 1991-1995).

A situation analysis report on Children and Women in Nigeria by UNICEF and the Federal Government of Nigeria in 1990 indicated an estimate of 58% and 21% for urban and rural areas respectively which had access to safe drinking water (Ewoigbokhan and Rodman, 1993). According to the General Household Survey (conducted between April 1991 to March 1992), pipe-borne water was available to 69% of urban household and only 11% of rural households, 40% of rural households fetched water from streams (Ewoigbokhan and Rosdman, 1993). Provision of safe drinking water was the most reliable and by far the most expensive (Hopkins, 1983). When piped-water was provided, guinea worm disease vanished, perhaps not quite overnight, but certainly in a couple of years (Muller, 1979). Muller (1971) cited an example of a Nigerian town of 30,000 population where construction of piped water supply in the 1960s reduced the incidence of guinea worm disesase from over 60 per cent to zero within two years. Bore-wells and tube-wells can also provide a constant supply of clean water, where their construction is technically feasible because the water is not liable to the contamination that can occur. Draw-wells when full, and if used with an elevated tank, can provide the convenience of water on tap.

Dracunculiasis had been eliminated in Igbon, Oyo State, Nigeria, through self-help activities including a series of cooperative efforts which culminated in the construction of wells which provided them, with a protected water supply (Edungbola and Watts, 1990). Hand-dug well could be provided on a family basis, it is cheaper and more reliable than other more expensive water intervention programs (Oteri, 1987).

The conversion of step-wells to draw-wells was sporadic in India, with some diminution in recent years, and this measure was primarily responsible for elimination of the disease in the Soviet Union (Uzbekistan) in 1932 (Editorial, 1983).

Studies have also shown that the provision of borehole reduced the prevalence of guinea worm in communities of Kwara State, Nigeria, from more than 50% to zero or near zero in three years (Edunghola *et al* 1986). This intervention strategy was relatively expensive and it was difficult for authorities to cover all areas which needed such sources. Nigeria's primary health care goal for potable water was that "40% of the population would live within 200 meters of a source of potable water" by the year 1992 (Ewoigbokhan and Rodman, 1993).

4

Control measures

4.1 Provision of potable water

The hand-pump-equipped boreholes have become a standard method of water supply in rural areas in much of the world. Boreholes can be drilled in most areas to give a yield of at least 750 litres per hour for a hand-pump (UNICEF Officer, 1995). The hand-pump previously used by UNICEF-Assisted Watsan Project in Nigeria was the India Mark-II piston pump which has a capacity of pumping up to 0.5 l/s., the maximum depth from which it can extract ground water is about 80 m. These two parameters combined to ground water quality dictate the feasibility of hand-pump water supply (Carel de Rooy, 1989). The India Mark III piston pump however replaced the India Mark II piston pump because of maintenance.

The high cost of sinking boreholes is reflected in all the resources needed for drilling and equipping boreholes. The human resources needed for drilling and equipping boreholes include at minimum, a hydro-geologist, drilling team(s), pump installation team, equipment repair team, community mobilization team(s) and necessary support personnel. Thirty to sixty people are usually involved in a project.

Equipment needed includes drilling rig, compressor(s), tools, transport and usually a workshop/garage. The initial drilling equipment as at 1995 was at least $400,000. There will be overhead costs, salaries and maintenance costs. In addition, each borehole will cost about $3,000 additional for fuel, casing, handpumps and other requirements (UNICEF Office, 1995).

Despite the phenomenal cost of sinking boreholes, there is sufficient reason to doubt their reliability and efficiency. For example, it was estimated that the number of boreholes which remained functional six months after sinking ranged from 20-96% (NIGEP, 1989). The reliability and efficiency of the boreholes are threatened by faulty pumps. Lack of a maintenance system with spare parts that the pumps (for boreholes used with power pumps), and the siting of the borehole in total disregard of geo-physical information and water levels.

However, studies in Nigeria showed that boreholes sited within a village and used exclusively for drinking water are most effective, while less accessible or malfunctioning boreholes have a less dramatic impact on guinea worm prevalence.

Providing a safe water supply alone is often not enough, measures must be taken to ensure that the new water sources are properly maintained. Without associated effort to motivate the population concerned, villagers may continue to use contaminated sources in preference to protected sources because water from contaminated site is perceived to be physically easier to collect, cheaper or tastier (Hopkins, 1983).

4.2 Filteration

Filtering drinking water to eliminate Cyclops which has been practiced for a very long time, is an effective prophylactic measure against dracunculiasis. Filtering of infected drinking water has been found to be relatively cheaper and more feasible on an individual basis than using chemicals to kill Cyclops in the water sources or providing alternative Cyclops-free sources of water such as protected well and pipe-borne treated water (Adeniyi *et al* 1991).

Although the cotton-cloth filters have been found very useful, particularly in rural communities where population may be too small and unstable to justify high investment in provision and maintenance of permanent safe water sources, these advantages of the filter are often negated by maintenance problems such as difficulty in keeping it clean and the short life-span of the cloth fabric. The pores are easily blocked with residual sediments in the raw water which makes filtration a very slow process and as a result of constant washing the filter gets torn very quickly, thus reducing its effectiveness in removing infected Cyclops (Adeniyi *et al* 1991).

In view of overcoming these problems, Steib, while working in Burkina Faso in 1982, designed, tested and showed the superiority of a monofilament polyester nylon gause (MPG) water filter over the popular cotton filter. This special material with a mesh size of 100 microns prevents the passage of even the smallest Cyclops larvae which carry the guinea worm, yet it does not clog up with silt and colloidal soil suspension from the local pond water, it is easily washed by rinsing. It is light but highly durable and preserves the "taste" of the water which the farmer likes (Duke, 1984). Guinea worm filtration of the drinking water is assured rapidly and effectively. This monofilament nylon gauze is an imported material and due to import restrictions, was not available in Nigeria.

In October 1990, DuPont and Precision Fabrics, an American company, announced the donation of monofilament nylon filter materials to Nigeria. Nwobi (1991) at the Fourth National Conference on Guinea Worm Disease in Nigeria stated that on 13 December, 1990, 49,000 square yards of monofilament nylon filter material donated by Dupont and Precision Fabrics arrived in the country. The shipment was valued at $250,000. Another shipment of 70,000 square yards was received in February 1991 and 130,000 metres he said, would arrive in May, 1991. In all, Nigeria's 1990 allocation of monofilament nylon filter material was 119,000 square yards, valued at over $700,000 but according to Nwobi 1991, over 626,000 square meters were needed to meet the estimated one-year filter requirement for all villages.

By 1994, filter coverage was found to be fairly good although distribution was often a one-time operation rather than a continuous exercise and many filters were found to be in need of replacement (Guineaworm Report, 1994).

4.3 Chemical treatment (Abate™)

An 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

Many insecticides have been found to be lethal to Cyclops indeed, 'were it not for its cumulative toxicity to other forms of life, Dichloro-Diphenyl-Trichloroethane (DDT) would probably be the most satisfactory compound on a cost/effectiveness basis' (Muller, 1970). Sahba *et al* (1973) attributed the great reduction in the prevalence of dracunculiasis in Iran to physical improvements and chlorination of the cistern as well as to incidental destruction of Cyclops by DDT which was sprayed on the inside roofs of the cistern for malaria control. Abate™ (Temephos), a chemical which was introduced in 1965 by the American Cyanamid Company, Atlanta, as a mosquito larvicide, was later found to be effective in the killing of Cyclops which are responsible for the transmission of guinea worm disease. It has also been used for the control of black flies (WHO, 1977).

Abate™ is an organo-phosphorus compound of minimal toxicity that has a molecular weight of 466.4 and an empirical formula of

$C_{16}H_2$-$O_6P_2S_3$ (Edungbola *et al* 1994). It is available in emulsifiable concentrate as brown viscous liquid or a white crystalline water dispersible powder, with granular formulation (ISG) (Sastry *et al* 1978). It has a specific gravity of 1.32, melts at 30.0 to 30.5^{0}C, is insoluble in water and stable indefinity at room temperature (Sastry *et al* 1978).

Abate™ inhibits the production of an enzyme called cholinesterase which is essential for synaptic transmission of nerve impulses in living organisms. The mode of action of Abate™ as a copepodcide is by selectively causing paralysis and ultimately death of cyclops through irreversible inhibition of cholinesterase enzymes by alkylphosphorylation (Edungbola *et al* 1994).

The organo-phosphorus compound, Abate™ (tempehos) have been shown both experimentally and in field trials to be effective and safe (Muller, 1970; Lyons, 1973) for controlling cyclops in the ponds. When properly applied at monthly intervals, Temephos is colourless, tasteless and odourless, with a wide margin of safety (Hopkins, 1983). The American Cyanamid Company announced the donation of Abate™ to endemic African countries in March 1990, the estimated value was $2.6 million of which Nigeria expected about $1.5 million over five years (Nwobi, 1991).

Sastry *et al* (1978) reported a 97 per cent reduction in number of cases (from 375 to 10) of dracunculiasis in a village of about 3,700 in Andra Pradesh State, India, within one year after they treated the villages' solitary source of drinking water, a step-well, with Temephos in 1975. The incidence of infection declined to only 11 per cent over the same period in a neighbouring control village.

4.4 Case containment

Studies carried out by Ward *et al* (1979) and Falode (1992) showed that respondents were unaware that adult males, particularly, farmers are at high risk. In the work of Belcher *et al* (1975) the highest attack rate was in adult male farmers with three out of four affected in some villages. Ward *et al* (1979) stated that it could be helpful to maintain a chronological ledger kept by the village with the assistance of the health worker, recording whether those who escaped the disease had been consistent in drinking only water which was known to be uncontaminated.

Educational efforts which draw the link between water use and susceptibility of adult males could be instrumental in dispelling notions of inherited susceptibility (Ward *et al* 1979).

Administration of anti-biotics has been shown to reduce the period of disability (Belcher *et al* 1975b) and this measure, combined with health education could serve to discourage practices such as incision (Ward *et al* 1979).

Four interrelated strategies (community organization, mass education, training of village health worker and advocacy) have been used to control guinea worm disease in Idere, Oyo State, Nigeria, where extensive studies on the disease have been carried out (Ewoigbokhan and Rodman, 1993).

The goal in 1994 had been to put case containment in place in every endemic village by the end of the year, being the way to ensure that no further transmission of the disease would occur in 1995.

Case containment means detecting every case within 24 hours of emergence of the worm, instituting all appropriate control measures within 24 hours of detecting the emerging worm, reporting each case promptly to the next level supervisor, and confirming each case by the next level supervisor within a week after it has been reported (CDC, 1994a). In Nigeria, by December 31 1994, approximately 71% of the remaining villages with known endemic disease had begun case containment measures designed to prevent further transmission (MMWR, 1995). Full containment of a case could be achieved if the worm was extracted prior to emergence; or if the worm was extracted or subjected to controlled immersion and, or occlusion of bandaging from the beginning of its emergence until it was completely expelled: or if all contaminated sources of drinking water were treated with appropriate amounts of Abate^{TM} within seven days of emergence of the worm (CDC, 1994b).

4.5 Village-based health workers

Community-Based Surveillance (CBS) has been defined as an ongoing process which involves community members in the collection and analysis of health data which leads to action on the health status of individuals and groups within the community (O'Neill, 1993). Its chief characteristics are that it is active, involving monthly visits to every household in the community; carried out by volunteer who is a member of the same community and therefore known to those visited. An essential component of the surveillance system is that the volunteers are supervised regularly. This is normally done once a month, usually by means of supervisory visits, but sometimes by attending meetings of several such volunteers organized by the supervisor (Cairncross, 1994). A number of CBS systems, some of them covering millions of people, have been operating in various countries. Most of these were established primarily for guinea worm eradication. The surveillance is often linked directly to specific interventions, such as nutrition education or guinea worm prevention. This kind of surveillance system was introduced by Nigerian Guinea-worm Eradication Programme (NIGEP). In every village that had guinea worm disease, a Village-Based Health Worker (VBHW) was trained to look for cases and report them every month. The surveillance system had improved through the training and retraining of these VBHWS (Ewoigbokhan and Rodman, 1993).

Once a village is free from the disease, it becomes vitally important to ensure that no outsider re-contaminates the drinking water source (Bourne, 1982). What seemed to be a straightforward solution was made more complex by human behaviour, population density, culture, human geography and technological capabilities (Brieger *et al* 1982). These could be illustrated in the town of Igbo Ora, Nigeria. After the introduction of piped-water some ten years ago, the incidence of guinea worm disease probably did drop nearly to zero, but today cases are still seen occasionally among residents (Brieger *et al* 1982).

The importance of health education cannot be under-stated because if it succeeds in its purported goal of changing people's behaviour, they will be less likely to revert to unhealthy habits when technology fails, such as the pump or when electricity power outage. The simple techniques of filtering or boiling the water and preventing the infected person from getting into the pond may be sufficient to contain the disease and prevent a major outbreak; this will happen only if the villagers believe in the transmission of the disease through contaminated water and not by evil spirit (Pilotto and Gorski, 1992).

4.6 The impact of health education

Besides the worm itself and the stagnant water with copepods combinations, the third element in the cycle of the disease is the human being. This is probably the key element, for without the peoples' cooperation and understanding none of the control measures will have much chance to succeed. Water is essential for life and even the most humble villager knows that, and the local village pond is almost sacred (Pilotto and Gorski, 1992).

Apart from the role of health education as an important adjunct to provision of safe water, health education can be an effective means of intervention in and of itself is relatively inexpensive (Hopkins, 1983). Health education is more than just the dissemination of information (in the media, or by means of posters or traditional channels, such as town criers). In order to better communicate, health education investigates factors antecedent to a given health problem, in relation to the desired outcomes in designing interventions. This ensures that interventions appropriately address the problem. Solutions can be presented to people in terms they understand and which make sense to them (Ewoigbokhan and Rodman, 1993). Antecedent factors include among others perceptions, beliefs and knowledge concerning the disease (causation, prevention and treatment) and use of water.

Urging patients with patent dracunculiasis infections not to go into 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. As of June 1993, NIGEP had supplied 89% of all endemic villages and 95% of the villages in most endemic local government areas with filters (Ewoigbokhan and Rodman, 1993).

Kale (1982) attributed a dramatic reduction in subsequent incidence of dracunculiasis to reduced contamination of the drinking water; the patients, whose wound were dressed as part of their treatment, wished to keep their bandages dry and therefore refrained from immersing them in the ponds (the treatment also shortened the duration of their infections). Over eight years, the annual incidence of infection fell from an average of about 28 per cent to zero in sixteen of seventeen villages studied.

5

Guinea worm eradication: Case studies in south-western Nigeria

5.1 Nigeria's experience

In 1986 the World Health Organisation (WHO) designated dracunculiasis as the next disease scheduled to be eradicated by 1995 after smallpox (Hopkins and Ruiz-Tiben, 1992). This eradication deadline was not met and WHO then hoped to certify eradication by 2005. Eradication was defined as the absence of guinea worm disease (and therefore the interruption of transmission) for three or more years (Hopkins *et al* 2007 and 2008). At the 2006 World Health Assembly (WHA) in Geneva, the World Health Organisation (WHO) convened an informal meeting on the eradication of dracunculiasis and additional measures needed to stop transmission of the disease in all of the remaining endemic countries by the end of 2009 (CDC, 2006).

However, at the end of 2009, WHO had certified a total of 187 countries and territories as free of dracunculiasis. Not yet certified by WHO were the four still-endemic countries (Ethiopia, Ghana, Mali, Sudan), seven recently-endemic countries that had interrupted transmission (Burkina Faso, Chad, Cote d'Ivoire, Kenya, Niger, Nigeria, Togo), and six never or not-recently-endemic countries (Angola, Democratic Republic of Congo, Eritrea, Somalia, South Africa) or territories (Greenland).

Despite the fact that the eradication goal was not achieved, the number of countries where dracunculiasis was reported were reduced to four, the number of cases reported were reduced to 3,190 against 3,500,000 cases reported when the eradication campaign began and the number of villages reporting indigenous cases of the disease were reduced to 645 (CDC, 2010).

Nigeria, which reported more cases than any other country (over 653,000 in 1988/89), reported zero cases for an entire calendar year for the first time in 2009.

Major support for the dracunculiasis eradication program is from The Carter Center who has received major grants from Bill and Melinda Gates Foundation, the United Kingdom, the Kingdom of Saudi Arabia, the Government of Oman, Vestergaard Frandsen, the John P. Hussman Foundation and the OPEC Fund for International Development.

Other major external support for the dracunculiasis eradication program was provided by the World Health Organization (WHO), the United Nations Children's Fund (UNICEF), Centers for Disease Control and Prevention (CDC), and many other donors. The Japan International Cooperation Agency (JICA) and UNICEF helped to support safe water sources in some dracunculiasis-endemic villages.

Only thiry-two cases were reported in 2007 as against the over 653,000 in 1988/89; just as Nigeria was preparing to cross the finish line to interrupting transmission nationwide, an unexpected outbreak of dracunculiasis was discovered. The 2007 outbreak was in two villages in Enugu State, twenty-eight active cases in Ezza Nkwubor Village and two cases in Ezza Ugwuomu Village (CDC, 2007). However, Nigeria reported no case for an entire calendar year for the first time in 2009.

There were several impediments which slowed down eradication of the disease in Nigeria. These are highlighted in series of studies conducted in several villages, fifteen villages (1991-1998) (Morenikeji, 1998; Morenikeji and Odaibo, 2007), six villages in 2004 (Morenikeji and Alade, 2007), eight villages in 2005 (Morenikeji and Adekolu, 2009) and ten villages in 2006 (Morenikeji and Alaka, 2010) in south-western Nigeria.

5.2 Treatment of guinea worm cases

One of the main control measures was simple treatment of cases by immersion of lesions in water to allow guinea worm come out and wind on a stick or thread before dressing and medication by health officials. Drugs given to infected villagers by these officials included panadol, aspirin, ampicillin, tetracycline, multivitamin tablets and ambilhar tablets.

The treatment kit box contained dettol, eusol lotion, savlon, bandage "Z", forceps, cotton-wool, pard parker handle (size 4), surgical scissors and vials of saclophen. Tetanus toxoid injections are sometimes given to the infected villagers.

Bandaging of the guinea worm ulcers helped in preventing the infected people from dipping affected parts in water source. As much as this would have gone a long way in preventing transmission, some infected villagers complained that the bandaging was not very effective because it did not allow the guinea worm 'breathe' and this they claimed make the worm retract and come out in another part of the body. Thus some villagers prefer leaving their ulcers open removing the bandage applied by health officials or protesting against its use on them. Villagers loved collecting tablets since it is free and they had no objection to the tetanus toxoid injections.

Plate 5.1: Treatment by immersion of lesion in water method.

Source:

5.2.1 Safe water supply

Safe water supply through boreholes was hampered by faulty hand-pumps, insufficiency of its water during dry season and in densely populated villages. More respondents 85/500 (17%) fetched from the ponds in the dry season when water level in wells was low especially in densely populated areas where they had to queue for borehole water and in areas with non-functioning boreholes than in the rainy season 21/500 (4.2%). Some villages were probably not included in the borehole programme because they were too small to justify the cost of drilling and maintenance of a hand-pump. It was impossible to put in place a hand-dug well in an endemic study-village due to the topography and geology of the area.

Attachment of villagers to ponds was a strong factor as most villages studied still used the pond for one thing or the other even when a safe source of water exists. Old villagers stated they preferred the "natural" taste of pond water. Villagers casually took water from ponds on their way to farm or while processing palm kennels or washing near ponds.

5.2.2 Filtering and boiling

Filtering and boiling are cumbersome, time-consuming and expensive procedures for villagers. It was observed that Global 2000 Field Officers did not visit some villages regularly and filters given to the villagers were used for sieving grounded maize rather than drinking water.

5.2.3 Abate™

It was observed that the volume of water in ponds in some of the villages was not accurately calculated before the application of Abate™. The Abate™ used on some occasions had expired.

The copepodcide, Abate™ (Temephos) was given to the Local Government Area in September 1984 by Nigeria Guineaworm Eradication Programme (NIGEP). The drug was donated to the Jimmy Carter Centre by the American Cyanamid Company, Atlanta. This was the first time Akinyele LGA was given Abate™. The LGA health officials were trained on how to apply the drug by NIGEP officials.

The procedures which was as follow:

1. The volume of water in a pond in an endemic village which must not exceed 500 cubic metres was calculated, for approximately circular ponds using the formula $\pi r^2 h$, for semi-circular ponds $\pi r^2 h$ and for rectangular or square ponds L x B x H (H = depth).

2. The depth was measured in four or more points and the average determined.
3. The volume was measured in cubic metres.
   1 cubic metre of water : 100 litres of water.
   0.1 ml of Abate™ will treat 50 litres of water.
   Therefore, 1 ml of Abate™ will treat 500 litres of water.

Before treatment of ponds in the study-area, the treatment team:

1. Obtained the consent of the community leaders.
2. Educated the villagers about the milky nature of Abate™ and its odour.
3. Informed the villagers that it was advisable to fetch water after six hours of treatment.
4. Educated villagers on the use, mode of action of the drug and reason for its application.

The ponds were treated between 6-7 pm. The treatment team consisted of a NIGEP official, local government guinea-worm eradication coordinator, other officials from the local government area health department and the team in this study. The village-based health workers and the villagers were also actively involved.

5.2.4 Village-based health workers

Effectiveness of Village-Based Health Workers (VBHWs) was hampered by lack of proper incentives and their commitment became hard to maintain as cases of guinea worm decreased and monthly visit to every household became unnecessary making case containment ineffective in study-villages.

Village health workers in infected villages in study-area were trained and given treatment kits to enable them carry out case containment in their villages.

For case containment, each case of guinea worm must be detected within 24 hours of first emergence and treated. The village health worker must bandage each lesion immediately, persuade patients and their families to ensure that no emerging worm is immersed in a source of drinking water, and report cases quickly to his or her supervisor in time for confirmation of the diagnosis and further containment measures (Cairncross *et al* 1995).

5.2.5 Beliefs

Beliefs like disease being caused by spiritual attack, inherited susceptibility, transference through sexual intercourse with an infected wife, infection from penetration of whitish worm in groundwater into body, bandaging wound will not allow the worm breathe and lack of understanding of the transmission and prevention of the disease have slowed down eradication.

Herbs were also used for treatment instead of antiseptic cleansing; applying antibiotic ointment and correct bandaging techniques. Imported cases were an issue in villages with immigrant farmers from endemic states.

These studies showed the need for continued health education, more provision, sustainance, maintainance of accessible safe water sources in all endemic and at-risk areas. Studies bring to fore the need for renewed commitment and energy input on the part of all agencies committed to the struggle to combat the last few cases of the guinea worm.

5.3 Case Studies

5.3.1 Odeda LGA of Ogun State, Nigeria

Of the 2,418 people in six villages (Abata, Apena Kemta, Bosero, Erinle, Ikaagbo, and Ikija) in Odeda LGA (the only infected LGA at the time of study), Ogun State, Nigeria, from January to December 2004, 24 (1.0%) were found to be infected with the guinea worm disease (Morenikeji and Alade, 2007).

Out of 250 Head of Households (HOHs) who were majorly farmers (87.2%), 80% had been infected before, 96% depended solely on pond water for drinking during the dry season (there were no boreholes in villages), 68% thought that guinea worm disease is caused by spiritual attack, only 4% associated the disease to bad drinking water. Further probing showed that 87.5% did not know it is transmitted through drinking water and 77.5% did not know if it is possible to prevent the guinea worm disease.

The plateau land formation of the Local Government Area (LGA) which results in relief rainfall is favourable for farming. It brought in migrant farmers from Benue State (most infected state in 2003) into this LGA, probably importing the disease. It was observed that the Global 2000 Field Officer in charge of eradication activities in this area did not visit villages regularly and filters given to the villagers were used for sieving grounded maize rather than drinking water. Beliefs like disease being caused by spiritual attack, inherited susceptibility and transference through sexual intercourse with an infected wife, infection from penetration of whitish worm from ground water into body and lack of understanding of the transmission and prevention of the disease slowed down eradication. There was also the unavailability of safe sources of water and it was observed that the volume of water in the ponds was not accurately calculated before the application of the Abate™ in one of the visits by health officials to treat the ponds.

Instances in which dracunculiasis can increase when it is not totally eradicated can be seen in the fact that only three cases were reported in Ogun State by December 2003 and 24 cases were recorded in this study in 2004.

5.3.2 Ibarapa North, Iseyin and Oorelope LGAs of Oyo State, Nigeria

Another study on the epidemiology of the guinea worm disease at the threshold of its eradication in Oyo State, south-west Nigeria, was carried out in three guinea-worm-infected LGAs, Ibarapa north, Iseyin and Ooreloppe (Morenikeji and Asiatu, 2010). Study-communities from these LGAs included Igitele, Isale-Oja, Isale Akao, Asunnara, Ayete, Oke Ola 1, Oniko, Ado Awaye, Adenko and Ologede.

A total of fifty-three (53) cases were reported in 2004 and 2005 in all the study-areas. However no cases were recorded from January to December 2006. Five hundred (500) HOHs were interviewed in the three LGAs. A total of 56% HOHs had been infected before.

Only 375/433 (89.4%) claimed to fetch drinking water from safe sources all the time out of those that had a safe source and more respondents 85/500 (17%) fetched from the ponds in the dry season when water level in wells was low especially where they had to queue for borehole water and in areas with non-functioning boreholes. It was observed that safe sources of water were inadequate or non-functional in densely populated villages of Ibarapa North and Oniko. In Oniko Village where the three boreholes were non-functional, the pond remained the only source of water. Some respondents out of those infected before 123/280 (43.9%) believed the disease was present in the blood, 280 (40.4%) believed it was from infected water and 104/280 (37.1%) used herbs for treatment. Filtering and boiling are cumbersome, time consuming and expensive procedures for villagers.

Imported cases might be an issue in Ologede where there were Igedes who were immigrant farmers from Benue State. These immigrant farmers might be the source of infection for Ologede and Adenko villages since the two villages share a pond. There was the need to sustain and maintain accessible safe water sources in all areas especially to guard against importation and spread of disease. Ignorance especially, belief in inherited susceptibility, lack of sustained safe water sources and influx of infected immigrant farmers were major variables responsible for the persistence of the disease in the study-communities.

WHO Collaborating Centre for Research, Training and Eradication of Drancunculiasis reported an imported case of dracunculiasis in a village of Bukkuyam Local Government Area of Zamfara State in north-east Nigeria in May 2006 (CDC, 2006). Transmission of drancunculiasis from the patient, a male farmer, was not contained. The village volunteer reported having seen a worm emerge, but his supervisor did not arrive in time (within seven days of worm emergence) to confirm the case and the containment process. Zamfara State had not reported indigenous cases of drancunculiasis since before January 2004. Preliminary investigations indicated that the patient visited several communities in Oyo State in 2005, but none of the villages he visited had documented indigenous cases of dracunculiasis since February 2004.The probable origin of this case of drancunculiasis remained to be ascertained.

Health education was very important for the success of every component of the intervention strategies and it was a continuous process. For instance, it was observed that in the villages under study, series of health education were carried out to enable the villagers accept the treatment of their pond with Abate™. Persistent and consistent health education messages were needed to change adverse behaviour patterns. More villagers used filters in this study and the need to use better source of water for drinking was awakened in the people. The combination of both education and implementation of measures to provide safe water had been shown to be effective in the reduction of the disease in previous studies (Nwobi *et al* 1996; Hopkins,1998). If health education succeeds in changing people's behaviour, they will be less likely to revert to unhealthy habits when technology fails, such as when the pump breaks down or when there was no other source of water except pond (Falode and Odaibo, 2002b).

A very promising strategy to control dracunculiasis was the application of Abate™ on suitable sources of water. Village Based

Health Workers (VBHWs) were trained in each village by Global 2000 field staff to treat their ponds. They did this on monthly basis. As noted by Morenikeji *et al* (2005), proper application of the potent Abate™ will go a long way in ensuring the eradication of guinea worm diseases. Amali (2000), reported a reduction of more than 90% in the number of cases in Ebonyi and Oziba in 1999/2000 due to intensified Abate™ treatment of the ponds in these areas.

5.3.3 Obafemi Owode LGAs of Ogun State, Nigeria

Another study on dracunculiasis at the threshold of its eradication was carried out in eight infected and at-risk non-infected villages in Obafemi Owode Local Government Area (LGA) of Ogun State between January and December 2005 (Morenikeji and Adekolu, 2009). One hundred and fifty-eight people were examined in three infected villages, cyclopoid copepods were recovered from all ponds in the study villages and 76 HOHs were interviewed. The overall prevalence of infection was 5.07%. There was no significant difference in the prevalence of infection between sexes, 5.04% and 4.76% in the females and males respectively (χ^2: 0.83, $p > 0.05$). The period of infection coincided with the dry season. Un-infected *Halicyclops* and *Thermocyclops* species of cyclops were recovered. Majority (74.1%) of HOHs in infected and 38.8% HOHs in at-risk villages had been infected before. Most (68.4%) HOHs believed the infection was from drinking water. Filtering of water was mostly practiced. Studies showed that with persistent eradication efforts the 2009 deadline for eradication of the guinea worm disease from the country would be met.

The location of guinea worm infection was in the lower limbs. The number of emerging worms was between one and three. Incapacitation lasted for a period of two weeks or a month after the blister burst. Of those infected, there were four farmers (50%), one trader (12.5%) and three students (37.5%). The cases were recorded in the months of March and April towards the end of dry season. A total of 42 and 13 cases were recorded in the three infected villages in 2003 and 2004 respectively. There were 83 and 15 cases in all in the LGA in 2003 and 2004 respectively, 81% and 98% reduction in the number of cases from 2003 to 2005 in the three infected villages and LGA respectively.

For treatment, apart from the winding of the worm around a stick, most from all villages (69.2%) claimed they used antibiotics while 12.8% used shear butter, another 12.8% used herbs and 5.2% used nothing.

Majority (65.8%) of the respondents got their drinking water from ponds, 15.8% from boreholes and a few (18.4%) from the well during the dry season. Some claimed to harvest rain water during the rains. The water treatment mostly practiced by the respondents that treat water: 67 (88.2%) was filtering (85.7%), a few boiled (7.1%) and a few (7.1%) add alum to their water.

The study showed that dracunculiasis was still present in the study-area and it also showed the efficacy of eradication measures in reducing the prevalence. This could be seen in the number of infections recorded in the previous years (2003 and 2004) as compared with the 2005. The decline in guinea worm cases was commendable. This reduction of cases could be attributed mainly to the provision of safe drinking water to these infected villages. Udonsi (1987) and Hopkins (1998) found similar effects in their studies. However efforts must be intensified to combat the last few cases considering the fact that a small *foci* of infection could bring about a resurgence of the disease.

The period when cases occured was towards the end of dry season (March and April). The dry season was often associated with the consumption of water from ponds or water holes formed (or dug) in the bed of seasonal rivers when flow had ceased. The study showed that in spite of alternative water sources in some villages such as boreholes and wells, it was almost impossible to stop villagers from going to the ponds. Most HOHs (81.5%) claimed villagers still drank the infested water without filtering especially on their way to or from the farm.

Some villages that had no other source of water other than the ponds like Oduro with only two ponds (Gamta and Morroco) relied solely on these ponds. Onibata and Jibowu villages relied on water that was supplied to them fortnightly by the government. Hence, the government's inability to regularly supply safe water also forced inhabitants to revert to these contaminated ponds at the period of high water needs. The only village that had one functional borehole constructed by the government was Agbedi, being the most infected village. Most of the respondents thought that the infection was caused by contaminated pond water because cases of infection were reduced when pond treatment and filtering of drinking water started.

The presence of the disease in the infected villages was probably as a result of the three infected communities sharing a common source of water (Semore Pond). These villagers, most especially the farmers' drank unfiltered pond water with their hands on their way to or from farm. Drinking of these unfiltered pond water could have been prevented if they made use of pipe filters given to them by Nigerian Guineaworm Eradication Programme (NIGEP) officials. The old villagers stated they preferred the "natural" taste of pond water. This had threatened the success of the complete eradication of the disease in the area.

The eradication measures in the villages included usage of cloth and pipe filters supplied monthly by Global 2000 staff, provision of boreholes and wells, application of Abate™ to ponds and most importantly health education, without which the other eradication measures would have failed.

Filters were seen hung outside each household after usage to dry after washing. Water filters though wildly acclaimed to be cheap and the most simple eradication measure had to be widely distributed to be highly effective. Village Based Health Workers

(VBHWs) were trained in each village to treat their ponds with Abate™. They did this on monthly basis as long as Abate™ was given to them by health officials. As noted by Amali (2000), the intensified Abate™ treatment in 1999/2000 in Ebonyi and Oziba might have caused more than 90% reduction in the number of cases.

Health education had played the most important role in reduction of the disease to a minimal level. It was very important for the success of every component of the intervention strategies and it was a continuous process. For instance, it was observed that in the endemic communities, series of health education were done to enable the villagers accept the treatment of their ponds with Abate™ which brought about a reduction in the density of cyclopoid copepod population. The combination of both persistent education to change behaviour patterns and implementation of measures to provide safe water had been shown to be effective in the reduction of the disease in previous studies (Nwobi and Ihe, 1996; Hopkins, 1998).

5.3.4 Final hurdle in guinea worm disease eradication

The guinea worm disease had been eradicated in many countries and the situtionis as shown in Table 5.1 and Figure 5.1. Importation was highlighted as the main reason for the continued presence in Nigeria in a report by Morenikeji, 2010, up till 2009 when the country reported zero case. However, there is the need for continuous monitoring to avoid unexpected resurgence due to importation.

Studies into its epidemiology in the last stages of its eradication continues to be relevant to check for factors responsible for the continued presence of the disease to serve as lessons for areas still battling with the disease and for helping in the control of other parasitic diseases.

There was need to let health education cover infected and at-risk non-infected communities and for continued unwavering energy input of all agencies committed to the eradication struggle.

Table 5.1: Cases of dracunculiasis reported in 2004-2009.

Country	Year					
	2004	2005	2006	2007	2008	2009
Sudan	7,266	5,569	20,582	5,815	3,618	2,733
Ghana	7,275	3,981	4,136	3,358	501	242
Nigeria	495	120	16	73	38	0
Mali	357	659	329	313	417	186
Togo	278	73	29	0	0	0
Nigeria	240	183	110	14	3	0
Burkina Faso	60	30	5	0	0	0
Cote d'Ivoire	21	10	5	0	0	0
Ethiopia	17	37	3	0	41	24
Benin	3	0	0	0	0	0
Mauritania	3	0	0	0	0	0
Cases exported from one country to another	11	12	2	12	1 5	
Total	16,026	10,674	25,217	9,585	4,619	3,190

Source: CDC (2010).

5.3.5 Chad Republic and South Sudan

Chad experienced an unexpected outbreak after reporting zero cases of guinea worm disease for more than ten years. In 2010, ten cases were reported, and two cases in the first six months of 2011. Many of the cases were detected along herdsmen's migratory paths, and none of the patients had a history of travelling outside Chad Republic. In March 2011, The Carter Center assigned a representative to the National Guinea Worm Eradication Program Secretariat in Chad Republic who immediately began working with

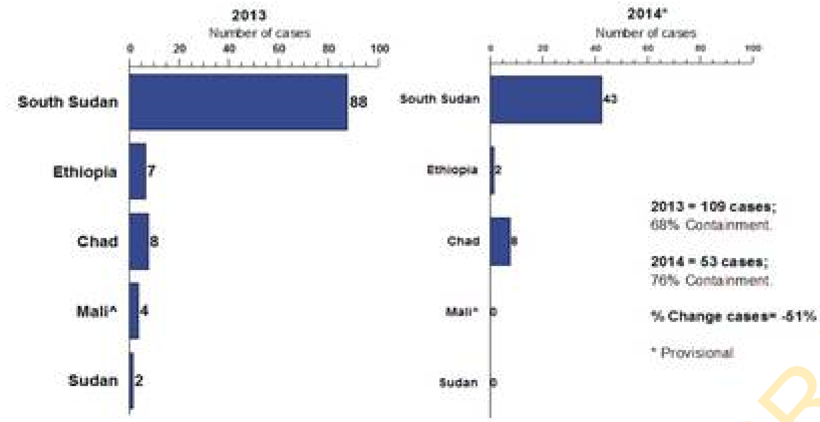

Figure 5.1: Reported cases of guinea worm by countries. January-July 2013 and 2014.

Source:

government staff to help revitalize the country's program. During 2010 and 2011, the staff trained health personnel to recognize the disease, implement surveillance, intensified advocacy and community mobilization in districts at highest risk.

Today, through the joint efforts of this initiative's many partners, the numbers of this disease had been reduced worldwide by 99 per cent: from an estimated 3.5 million cases in 1986 to fewer than 35,000 reported cases in 2003. Today, it is the last one per cent of the disease that is being fought (GHP, 2012).

One of the greater challenges in eradicating dracunculiasis had been and would continue to be in South Sudan (formerly southern Sudan), particularly, political uncertainty in the country with national elections in 2009 and the referendum on the status of southern Sudan in 2011 resulting in South Sudan's independence. Sporadic insecurity or widespread civil conflict could at any time ignite, thwarting eradication efforts (Hopkins *et al* 2008). To address some of the humanitarian needs in southern Sudan, in 1995, the longest ceasefire in the history of the war was achieved through negotiations by Jimmy Carter (Barry, 2007 and Carter, 2008). Commonly called the "Guinea worm cease-fire," both warring parties agreed to halt hostilities for nearly six months to allow public health officials to begin guinea worm eradication programming, among other interventions (Carter, 2008 and WHO, 2008).

The remaining endemic communities in South Sudan are remote, poor and devoid of infrastructure, presenting significant hurdles for effective delivery of interventions against disease. Moreover, residents in these communities are nomadic, moving seasonally with cattle in pursuit of water and pasture, making it very difficult to know where and when transmission occurred. The peak transmission season coincides with the rainy season, hampering travel by public health workers (WHO, 2008).

Public health officials cite the formal end of the war in 2005 as a turning point in guinea worm eradication because it allowed health-care workers greater access to southern Sudan's endemic areas (Hopkins *et al* 2005). One remaining area in West Africa besides Ghana, remains challenging to ending guinea worm is northern Mali, where Tuareg rebels made some affected areas unsafe for health workers.

References

Adeniyi, J. D., Brieger, W. R., Ramakrishna, J., Sridhar, M.K.C., Kale, O.O. and Ayeni, S. 1991. Acceptability and use of monofilament nylon filters in a guineaworm endemic area in Western Nigeria: An intervention study. UNDP/World Bank/WHO Special Programme of Research and Training in Tropical Diseases, Geneva, Social and Economic Research Project Report. No. 8.

Amali, O. 2000. Report of Global 2000 Guineaworm Consulting, Ebonyi Local Government Area of Ebonyi State, Nigeria. April 17-28, 2000.

Ampofo, O. 1977. Plants that heal: *World Health,* 7977, 26-30.

Anonymous. 1994a. Guineaworm wrap-up N46. Memorandum from WHO Collaborating Centre for Research Training and Eradication of Dracunculiasis. Department of Health and Human Services. December 30, 1994. 16pp.

Anonymous. 1994b. Guineaworm wrap-up N44. Memorandum from WHO Collaborating Centre for Research Training and Eradication of Dracunculiasis. Department of Health and Human Services. June 30, 1994. 16pp.

Belcher, D.W., Wurapa, F.K., Ward, W.B. and Lourie, I.M. 1975. Guineaworm in Southern Ghana: Its epidemiology and impacts on agricultural productivity. *Amer. J. Trop. Med. Hyg.,* 24(2) 243-249.

Belcher, D.W., Wurapa, F.K., Ward, W.B. 1975b. Failure of thiabendazole and metronidazole in treatment and suppression of guineaworm disease. *Amer. J. Trop. Med. Hyg.,* 24, 444-449.

Bhatt, A.N. and Palan, K.H. 1978. Guineaworm infection in Banaskantha District of Gujarat: Some important epidemiological aspects. *Ind. J. Med. Sci.,* 32:1-4.

Bourne, P.G. 1982. Global eradication of guineaworm. *J.R. Soc. Med., 75:*1-3.

Brieger, W.R. and Guyer, J. 1990. Farmers' losses due to guineaworm disease: A pilot study. *J. Trop. Med. Hyg. 93(2):* 106-111.

Brieger, W.R., Johnson, D.C., Adeniyi, J.D. and Akpovi, S.U. 1982. Complexity of Guineaworm Disease. *World Health Forum, 3,* 216.

Brieger, W.R., Luchock, E.E.K., Earp, J. and Lowenthall, N. 1989. Maternal Health and Guineaworm: The Idere experience. *Nig. J. Parasitol., 10 (suppl.1):* 65-66.

Brieger, W.R., Ramakrishna, J., Adeniyi, J.D., Sridhar, M.K.C. and Kale, O.O. 1991. Guineaworm control case study: Planning a multistrategy approach. *Soc. Sci. Med. 32 (12):* 1319-1326.

Cairncross, S. 1994. Community-based surveillance in support of EPI. Meeting of EPI Programme Managers, Yamoussoukro, 17-19.

Carel De Rooy. 1985. UNICEF-Assisted Water and Sanitation Project: A significant step towards improved health and living conditions in rural Nigeria. Proceedings of the First National conference on dracunculiasis in Nigeria. March 25-27, 1985:48-56

CDC. 2010. Guinea Worm Wrap-up #196. WHO Collaborating Center for Research, Training and Eradication of Dracunculiasis.

Centers for Disease Control. 2008. WHO Collaborating Center for Research, Training and Eradication of Dracunculiasis. Guinea Worm Wrap-up #182, July 7, 2008.

Centers for Disease Control and Prevention (CDC). 2007. Progress toward global eradication of dracunculiasis, January 2005-May. 2007. *MMWR Morb Mortal Wkly Rep 56:* 813-817.

Centers for Disease Control and Prevention (CDC). 2009. Progress toward global eradication of dracunculiasis, January 2008-June 2009 . *MMWR Morb Mortal Wkly Rep 58:* 1123-1125.

Donald, R. Hopkins, Ernesto Ruiz-Tiben, Philip Downs, P. Craig Withers, Jr. and Sharon Roy. 2008. *Am. J. Trop. Med. Hyg., 79(4)*, pp. 474-479.

Duke, B.O.L. 1984. Filtering out the guineaworm. *Wld. Hlth.* March 1984:29.

Editorial. 1983. After smallpox, guineaworm? *Lancet, 1:*161-2.

Edungbola, L.D. 1983. Babana parasitic disease project 11. Prevalence and impact of dracontiasis on Babana District, Kwara State, Nigeria. *Transactions of the Royal Society of Tropical Medicine and Hygiene 77:*310.

Edungbola, L.D. 1984. Dracunculiasis in Igbon, Oyo State, Nigeria. *J. Trop. Med. Hyg. 87:* 153.

Edungbola, L.D. and Watts, S J. 1984. An outbreak of dracunculiasis in a peri-urban community of Ilorin, Kwara State, Nigeria. *Acta Trop. 41*:155-163.

Edungbola, L.D., Watts, S.J., Kale, O.O, Smith, G.S. and Hopkins, D.R. 1986. A method of rapid assessment of the distribution and endemicity of dracunculiasis in Nigeria. *Soc. Sci. Med 23(6):* 550-8.

Edunbgola, L.D., Watts, S.J., Alabi, T.O. and Bello, A.B. 1988. The impact of a UNICEF-assisted rural water project on the prevalence of guineaworm disease in Asa, Kwara State, Nigeria. *Am. J. Trop. Med. Hyg. 39 (1):* 79-85.

Edungbola, L.D., Watts S. J. 1990. The elimination of dracunculiasis in Igbon, Oyo State, Nigeria. The success of self-help activities. *J. Trop. Med. Hyg. 93:* 1-6.

Edunbgola, L.D., Craig Withers, P., Braide, I.E., Kale, O.O., Sadiq, L.O., Nwobi, B.C., Alakija, T., McConnon and Hopkins, D.R. 1992. Mobilisation strategy for guineaworm eradication in Nigeria. *Am. J. Trop. Med. Hyg. 47 (5):* 529-538.

Edunbgola, L.D., Parakoyi, B.D., Kayode, O.O. and Bello, A.B. 1994. A study to ascertain the acceptance of abate (temephos) for the treatment of community drinking ponds in the Nigerian guineaworm eradication programme. *The Trop. J. Hlth. Sci. 1:* 49-54.

Ekeh, H.E. and Adeniyi, J.D. 1986. Treating school children for tropical diseases control: preliminary finding from a socio-behavioral research in Nigeria. *J. Trop. Med. Hyg. 89:* 1-6

Ewoigbokhan, S.E. and Rodman, A.E. 1993. Guineaworm Eradication. Astatus report. *Nig. Bull. Epidemiol. 3(2):*2-7.

Falode, O.A. 1992. Perception and management of guineaworm disease in Akinyele Local Government Area of Oyo State. A dissertation submitted in partial fulfillment of the degree of Master of Science in the Dept. of Zoology, University of Ibadan.

Falode, O.A. and Odaibo, A.B. 2002. Cyclopoid Copepods in domestic water sources in three villages in Akinyele Local Government Area of Oyo State, Nigeria. *The Zoologist (2):*16-22.

Falode, O.A. and Odaibo, A.B. 2002b. Management and Attitudes of villagers towards Boreholes in Guinea worm eradication in Akinyele Local Government Area of Oyo State, Nigeria. *The Zoologist: 1 (1):*61-68.

Fedchenko, A.P. 1971. Concerning the structure and reproduction of guineaworm. *Am. J. Trop. Med. Hyg. 20(4):* 511-523.

Federal Government of Nigeria (FGN) and United Nations Childrens Fund (UNICEF). 1991. Master Plan of Operations for the 1991-1995 Programme of Cooperation.

Gilles, H.M. and Ball, P.A.J. 1964. Guineaworm infection and gastric function. *Am. J. Trop. Med. Parasit 58:* 72-78.

Global. 2000. The Carter Center. Case Report Data. Global 2000 The Carter Center South-West Zonal Office, Nigeria. 2004.

Gooneratne, B.W.M. 1969. An additional historical note on the transmission of Dracunculus medinensis. *Trans. R. Soc. Trop. Med. Hyg. 63*:546.

Guineaworm Report. 1994. Vol.7 No. 1. November, 1994.

Hopkins, D. R. 1983. Dracunculiasis: An eradicable scourge. *Epidemiol.Rev. 5:* 208-219.

Hopkins, D.R. 1987. Dracunculiasis Eradication: A mid-decade status report. *Am. J. Trop. Med. Hyg. 37 (1):* 115-118.

Hopkins, D.R. 1988. Forgotten scourge. Decade Watch. 7:4pp.

Hopkins, D.R. 1991. Surveillance approaches and intervention efforts with guineaworm disease. *Nig. J. Parasit. Suppl No. 2:* 63-64.

Hopkins, D.R.1998. The guineaworm eradication effort: Lessons for the future. *Emerg. Infect. Dis. 4(3):* 414-415.

Hopkins, D.R. and Hopkins, E.M. 1992. Guineaworm: The end in sight. In Medical and Health Annual: Chicago: *Encyclopaedia Britannica.* pp. 10-27.

Hopkins, D. R. and Ruiz-Tiben, E. 1990. Dracunculiasis Eradication: Target 1995. *Am. J. Trop. Med. Hyg. 43 (3):* 296-300.

Hopkins, D. R. and Ruiz-Tiben, E. 1992. Surveillance for dracunculiasis, 1981-1991 *MMWR-CDC-Surv-Summ. 41 (1):* 1-13.

Johnson, S. and Joshi, V. 1982. Dracontiasis in Western Rajasthan, India. *Trans. R. Soc. Trop. Med. Hyg. 76*: 36-40.

Kale, O. O. 1974. A controlled field trial of the treatment of dracontiasis with metronidazole and niridazole. *Ann. Trop. Med. Parasit. 68:* 91-95.

Kale, O.O. 1975. Mebendazole in the treatment of dracontiasis. *Amer. J. Trop. Med. Hyg. 24:* 600-605.

Kale, O.O. 1977. Clinico-epidemiological profile of guineaworm in the Ibadan district of Nigeria. *Amer. J. Trop. Med. Hyg. 26:* 208-214.

Kale, O.O. 1982. Fall in incidence of guineaworm infection in western Nigeria after periodic treatment on infected persons. *Bull. WHO. 60:* 951-7.

Lancet. 1983. After smallpox, guineaworm? *Lancet 321(8317):* 161-162.

Laubjerg, K. 1991. Dracunculiasis and its effect on the welfare of children and the family. *Nig. J. Parasit. Suppl. No. 2:* 3-4.

Lucas, A.O., Oduntan, S.O. and Gilles, H.M. 1969. Niridazole in guineaworm infection. *Ann. N. Y. Acad. Sci. 160:* 729-739.

Lyons, G.R.L. 1972. Guineaworm infection in the Wa District of north-western Ghana. *Bull. WHO. 47(5):* 601-610.

Lyons, G.R.L. 1973. The control of guineaworm with Abate: A trial in a village of north-west Ghana. *Bull. WHO. 49:* 215-16.

Manson-Bahr, P.E.C., Apted, F.I.C. 1982. *Manson's Tropical Diseases. 18th Edition,* London: Bariliere Tindall, 231-733.

MMWR. 1995. C.D.C. Update, Dracunculiasis eradication: Ghana and Nigeria, 1994. 44(10): 189-191.

Monthly Surveillance Report. January-December. 1993. Nigeria Guinea Worm Eradication Programme.

Moorthy, R. 1937. A redescription of *Dracunculus medinensis. J. Parasit. 23:* 220-224.

Morenikeji, O.A. and Alade, A.O. 2007. Guinea worm disease and its persistence in some rural communities of Nigeria. *Nigerian Journal of Parasitology; 28(1):* 15-19.

Morenikeji, Olajumoke and Adekolu, Abimbola. 2009. Progress in Dracunculiasis Eradication: Ogun State, south-west Nigeria as case study. *Journal of American Science; 5(4):*189-193.

Morenikeji, O.A., Ajulo, B.O., Odaibo, A.B. 2005. Evaluation of the effectiveness of Abate™ used for the treatment of community drinking ponds in the Nigeria Guineaworm Eradication Programme. *Int. J. Nat. App. Sci.; 1(1):* 55-58.

Morenikeji, O. and Asiatu, A. 2010. Progress in dracunculiasis eradication in Oyo State, south-west Nigeria: A case study. *African Health Sciences, 10:* 298-302.

Muller, R. 1970. Laboratory experiments on the control of cyclops transmitting guineaworm. *Bull. Wld. Hlth. Org. 42:* 563-567.

Muller, R. 1971. Dracunculus and dracunculiasis. *Adv. Parasit. 64:* 107-116.

Muller, R. 1972. Maintenance of *Dracunculus medinensis* (L) in the laboratory and observations on experimental infactions. *Parasit. 64:* 107-116.

Muller, R. 1975. Worms and diseases. *A manual of medical helminthology,* William Heinman Medical Books Ltd., London, 161pp.

Muller, R. 1978. Dracontiasis in deseases of children in the sub-tropic and tropics. (eds. D.B. Jelliffe and J.P. Stanfield).

Muller, R. 1979. Guinea worm disease: Epidemiology, control and treatment. *Bull. Wld. Hlth. Org. 57:* (5)683-689.

Muller, R. 1985. Life cycle of *Dracunculus medinensis.* In: Workshop on opportunities for control of Dracunculiasis: contributed papers. Washington D.C. 16-19 June, 1982. Nat. Acad. Press. Washington D.C.

NIGEP. 1989. Nigerian Guineaworm Eradication Programme: National plan of action: Document presented at the 2nd National Conferences on Dracunculiasis: March 1989, by Federal Ministry of Health, Lagos, 27pp.

Nwobi, B.C. 1991. The status of guineaworm disease in Nigeria: An update. *Nig. J. Parasitol. Suppl. 2:* 31-62.

Nwobi, B.C. and Ibe, K.K. 1996. Dracunculiasis transmission and variations in geological formation. A preliminary study in Afiko-Amasiri-Okposi region of south-eastern Nigeria. *The Nigerian Journal of Parasitology: 17:* 75-81.

Nwobi, B.C., Ibe, A.U.C., Oguoma, C.E.O and Sallau, A.K. 1996a. Field experiences on the use of ''ABATE'' in the north-east zone of Nigeria. *The Nig. J. Parasit. Vol.17,* 63-68.

Nwobi, A.B.C., Ifezulike, E.O. and Anya, A.O. 1982. Endemic dracontiasis in Anambra State of Nigeria: Geographical distribution, clinical features, epidemiology and socio-economic impact of the disease. *Ann. Trop. Med. Parasit. 76:* 187-200.

Nwosu, A.B.C., Ifezulike, E.O. and Anya, A.O. 1982. Endemic dracontiasis in Anambra State of Nigeria: Geographical distribution, clinical features, epidemiology and socio-economic impact of the disease. *Ann. Trop. Med. Parasit. 76*: 187-200.

Oduntan, S.O., Lucas, A.O. and Gilles, H.M. 1967. Treatment of dracontiasis with niridazole. *Lancet, 2 (7506):* 73-75.

Ofoezie, I.E. and Adeniyi, I.F. 1990. The prevalence and endemicity of dracunculiasis in Akowide Village, Oyo State, Nigeria. *Ann. Trop. Med. Parasit. 84 (2):* 163-169.

Olajide, I., Sridhar, M.K.C. and Kale, O.O. 1987. Guineaworm control in an endemic area in Western Nigeria. *Aqua. No 6*: 333-339.

Onabamiro, S.D. 1951. The transmission of *Dracunculusmedinensis* by *Thermocyclops nigerianus* as observed in a village in south-west Nigeria. *UIAnn. Trop. Med. Parasit. 45:* 1-10.

Onabamiro, S.D. 1952a. Four new species of Cyclops *sensu lat.* (Crustacea: copepod) from Nigeria. *Proc. Zool. Soc. Lond. 122 (1):* 253-266.

Onabamiro, S.D. 1952b. On the diurnal migration and seasonal fluctuation in the numbers of *Thermocyclops nigerianus,* Keifer in a Nigerian pond. *Ann. Trop. Med. Parasit. 46 (1):* 38-47.

Onabamiro, S.D. 1952c. The geographical distribution and clinical features of Dracunculus medinensis in south-west Nigeria. *W. Afr. Med, J. 1 (4):* 159-165.

Onabamiro, S.D. 1954. The diurnal migration of cyclops infected with larvae of *Dracunculus medinensis,* with some observations of the larval worms. *W. Afr. Med. J. 3*: 189-194.

O'Neill, K. 1993. Community-based surveillance: A critical examination of nine case studies. M.Sc. dissertation, London School of Hygiene and Tropical Medicine.

Oteri, A.U. 1987. The role of shallow wells in the provision of safe water supply. In: Dracunculiasis in Nigeria: Conference proceedings. First National Conference on Dracunculiasis in Nigeria, Ilorin 1985. (eds L.D. Edungbola, O.O. Kale and S.J. Watts). Emene, Nigeria, pp. 123-131.

Padonu, K.O. 1973. A controlled trial with metronidazole in the treatment of dracontiasis in Nigeria. *Trop. Geogr. Med. 25 (3):* 238-241.

Pilotto, L. and Gorski, J. 1992. Progress towards the eradication of Dracunculiasis. *J. Roy. Soc. Hlth. April 1992,* 78-83.

Ransome-Kuti, O. 1991. Eradication of guineaworm by 1995: An integral approach. *Nig. J. Parasit. Suppl. No. 2*: 17-26.

Reddy, C.R.R.M., Narasaiah, I.L. and Parvath, G. 1969. Epidemiological studies on guineaworm infection. *Bull. Wld. Hlth. Org. 40*: 521-529.

Sahba, G.H., Afraa, F., Fardin, A. and Ardalan, A. 1973. Studies on dracontiasis in Iran. *Am. J. Trop. Med. Hyg. 22 (3):* 343-7.

Sastry, S.C., Jakumar, K., Lakshaminarayana, V. and Seethapathi Rao, V.N. 1978. Abate: Its value as a cyclopscide. *J. Trop. Med. Hyg. 81*: 156-158.

Schmidt, G.D. and Roberts, L.S. 1981. *Foundations of Parasitology* 2nd Ed. The C.V. Mosby Company, Saint Louis, 795pp.

Scott, D. 1960. An epidemiological note on guineaworm infection in north-west Ashanti, Ghana. *Ann. Trop. Med. Parasit. 54*: 32-43.

Smith, G.S., Blum, D., Huttly, S.R.A., Okeke, N., Kirkwood, B.R. and Feachem, R.G. 1989. Disability from dracunculiasis: effect on mobility. *Ann. Trop. Med. Parasit. 83 (2):* 151-8.

Southwell, T. and Krishner, A. 1938. Some observations on guineaworm larvae. *Ann. Trop. Med. Parasit. 32:* 193-6.

Stoll, N. 1947. This wormy world. *J. Parasit. 33:* 193-6.

Tayeh, A. and Cairncross, S. 1993. Dracunculiasis eradication by 1995. Will endemic countries meet the targets? *Health Policy and Planning. 8(3):* 191-207.

Ten Eyck, D.R. 1971. Report on an outbreak of dracunculiasis in Ethiopia. *Ethiop. Med. J. 9:*149-152.

Udonsi, J. K. 1987. Control of endemic dracontiasis by provision of water supply in rural communities of Imo State, Nigeria. *Pub. Hlth. 100 :* 63-70.

Ukoli F. M.A. 1984. *Introduction to parasitology in Tropical Africal.* John Willey and sons. 464pp.

UNICEF, South-West Zonal Office, Nigeria. 1995. Personal Communication.

Ward, W.B., Belcher, D.W., Wurapa, F.K., and Pappoe, M.E. 1979. Perception and management of guineaworm disease among Ghanaian villagers. A framework for differential health education planning. *Trop. Georgr. Med. 31 (1):* 155-164.

Watts, S.J. 1987. Dracunculiasis in Africa in 1986: Its geographical extent, incidence and at risk population. *Am. J. Trop. Med. Hyg. 37:* 119-125.

Watts, S. J., Brieger, W. R. and Yacoob, M. 1989. Guineaworm: An in-depth study of what happens to mothers, families and communities. *Soc. Sci. Med. 29(9):* 1043-9.

WHO 1977. Notes and News: Onchocerciasis Programmes in the Volta River Basin Area: (OCP) makes encouraging progress. *WHO Chronicles 31:* 113-114.