Dracunculiasis in the Caribbean and South America: A Contribution to the History of Dracunculiasis Eradication

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Introduction

Dracunculiasis, Guinea worm disease, is a debilitating parasitic infection of humans most commonly associated with sub-Saharan Africa and India. However, well into the nineteenth century it was intermittently linked with the unregulated importation of humans, the trade in slaves, from areas of West Africa where it was endemic to plantations in the Caribbean islands and South America. In a few of these places local transmission was established for short periods of time at drinking water sources. But, so far as can be determined, no local transmission was recorded after the end of the slave trade, which in the Spanish world occurred in the 1860s. Thereafter, occasional cases were noted among immigrants and travellers, but these did not establish a chain of local transmission.

It is the aim of this paper to trace the latest records of the presence, and short-lived local transmission, of Guinea worm in the Caribbean and South America. Most of these accounts were generated by medical practitioners, though a few were the work of lay people with an eye for disease curiosities; those written before 1870 lack our modern understanding of the epidemiology of dracunculiasis. Their validity, therefore, needs to be assessed in the light of current epidemiological knowledge of the disease—the environmental setting and human behaviour necessary for its transmission from year to year in a particular place. Many of these records also point to an African origin for dracunculiasis, as they contain information about the empirical knowledge of local non-literate peoples, slaves and free, which may have derived from their earlier residence in a Guinea worm endemic area in Africa.

The subject is of interest because of the programme for the global eradication of dracunculiasis, which has been underway for the past fifteen years. To date, this has resulted in a worldwide decline in the number of dracunculiasis cases from about

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The author is grateful for a Wellcome Research Travel Grant in the History of Medicine, which enabled her to look at sources in London during July and August 1996. Thanks are also extended to Donald Hopkins of Global 2000, Philippe Ranque of WHO, Trenton Ruebush of CDC, Pedro Pruna at the Cuba Society for the History of Science, Julian Pearse at San Francisco State University, and to Sheldon Watts in Cairo, for comments and encouragement during the writing and revision of the paper.
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3.5 million in 1986 to around 150,000 in 1996 and just under 78,000 in 1997. In association with the eradication programme, as part of the process whereby individual countries are certified as being free of the disease, the World Health Organization (WHO) is co-ordinating the documentation of the end of the infection in areas where it was previously recorded, including the Americas.

Local and Long Distance Transmission of Dracunculiasis

The Guinea worm, Dracunculus medinensis, evolved a life-pattern and reproductive cycle particularly well suited to tropical areas with pronounced wet and dry seasons. The worm has a twelve-month incubation period, the time between the human activity of drinking water containing Guinea worm larvae and the appearance of the female worm on the surface of the sufferer’s skin. During these twelve months, the unknowing carrier might travel long distances. In the seventeenth and eighteenth centuries, the easternmost portion of the dracunculiasis-prone area of West Africa was known as the Guinea Coast, hence the name Guinea worm, which was used by William Dampier as early as the 1680s. In the era of sailing ships, sailors, travellers or slaves leaving the West African coast may have suffered from emerging worms on reaching other tropical areas, or north-west Europe, far from the original site of infection.

For the Guinea worm to be transmitted from one human being to another in a new region requires the near duplication of the earlier environmental setting in space and in time, and the appropriate human behaviour. These requirements are so specific that (once they became known) it was not difficult to prevent the spread of the disease. Yet even without this understanding of the worm’s life cycle, the specific environmental conditions and human behaviour needed for further transmission from one human to another via the intermediate copepod host in an entirely new setting seem to have coincided in only a few places in tropical America, and for limited periods of time.

The first requirement for transmission is that an infected human immerses the part of her or his body from which the worm is emerging into water containing copepods, minute water crustaceans, the intermediate hosts necessary for the completion of the reproductive cycle of the Guinea worm. On contact with the water, the emerging female worm releases thousands of first-stage larvae which, within a

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1 World Health Organization, 'Dracunculiasis: global surveillance summary, 1997', Wkly Epidemi. Rec., 1998, 73 (18): 129–35; WHO, 'Dracunculiasis: global surveillance summary, 1996', Wkly Epidemi. Rec., 1997, 72 (19): 133–9; Donald R Hopkins, Ernesto Ruiz-Tiben and Trenton K Ruebush, 'Dracunculiasis eradication: almost a reality', Am. J. trop. Med. Hyg., 1997, 57: 252–9; S Watts, 'Dracunculiasis in Africa in 1986: its geographic extent, incidence, and at-risk population', Am. J. trop. Med. Hyg., 1987, 37: 119–25.

2 WHO, 'Dracunculiasis: certification of eradication', Wkly Epidem. Rec. 1997, 72 (6): 33–5; WHO, Criteria for the certification of dracunculiasis eradication, rev. version, 1996, WHO, Division of Control of Tropical Diseases, WHO/FIL/96.187, p. 8. In epidemiological parlance, the term “eradication” refers to complete, global eradication, while the term “elimination” is used for individual countries.

3 Susan J Watts, 'Population mobility and disease transmission: the example of Guinea worm', Soc. Sci. Med., 1987, 25:1073–81.

4 William Dampier, A new voyage round the world, 1st ed. 1693, London, James Knapton, 1703–5, vol. 2, pt 2, p. 89.
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Figure 1: The dracunculiasis transmission cycle.

Source: Centers for Disease Control.

few days, must be ingested by the copepod hosts. Approximately two weeks later, a person has to drink the water containing the copepods encasing the now infective larvae. In the victim’s gut the copepods dissolve and the male and female larvae develop and mate. Subsequently, the male worm is absorbed into the human tissues. The female emerges, about a year after the ingestion of the larvae into the human body, to continue the cycle of infection, as shown in Figure 1.5

Copepod species are widely distributed in Africa, the Americas, and in Europe, but only a few of them are able to act as intermediate hosts of the Dracunculus. For example, in Cuba, where local transmission existed in the 1860s, 27 species of copepods have been found, but Macrocylops leuckarti, identified as an intermediate host in Africa, was not among the most common.6 As the optimum water temperature

5 For a brief history of dracunculiasis and disease transmission, see Donald R Hopkins, ‘Dracunculiasis’, in Kenneth F Kiple (ed.), The Cambridge world history of human disease, Cambridge University Press, 1993, pp. 687–9; Ahmed Tayeh, ‘Dracunculiasis’, in F E G Cox (ed.), The Wellcome Trust illustrated history of tropical diseases, London, Wellcome Trust, 1996, pp. 287–93. On the epidemiology of dracunculiasis, see Donald R Hopkins

6 Julio Burbano Diago, Jorge Delgado Bustillo and José A Cepero Martín, Certification of eradication of dracunculiasis: evaluation of the risk of reintroduction of the disease in Cuba, Havana, Cuba, November 1996, pp. 5–6.
for the development of the Guinea worm larvae within the copepods is 25°–30° C, people can be infected only in the tropics or sub-tropics.\(^7\)

Considerations of time, as well as place are relevant in considering the spread of the disease. The twelve-month cycle ensures that the period of maximum patency, when the worms emerge, coincides with water conditions and human water-use behaviour most suitable for transmission. In the coastal and Guinea savannah areas of West Africa where the wet season lasts for six months or more, the disease is usually contracted in the dry season, when many people use small, stagnant ponds. In northern interior areas of West Africa, in the Sahel, on the other hand, transmission occurs principally in the short wet season, in the summer, when people use temporary ponds as sources of drinking water.\(^8\) Thus, in considering the possibility of local transmission in the various habitats on the other side of the Atlantic, in the Americas, there must be a symmetry between seasonal conditions in the new location and the old.

For the disease to continue in the new area, an affected person has to arrive at a water source at the time of year when it provides local people with their drinking water. The most likely sites are shallow ponds or wells which enable an individual to enter and immerse an affected limb, usually a leg, in the water. After collection, transmission can be interrupted if the water is filtered through a fine cloth to prevent the passage of the cyclops into the storage jar, and from thence into a human gut.

**Records of Dracunculiasis in the Americas**

This paper focuses on reports of dracunculiasis in the Americas prior to about 1900. This cut-off date marks the beginning of widespread understanding among medical practitioners of the transmission cycle of dracunculiasis. Before then, there were many different hypotheses about how people acquired the disease. Some Western medically-trained observers asserted that drinking water played a role, and that water from suspect sources should be boiled or filtered. Other commentators held that the larvae penetrated the human skin; others that the disease was contagious, transmitted directly from person to person.

In 1870, the Russian scientist Aleksej Fedchenko (1844–1873) published an article identifying the copepod as the intermediate host. The study was based on his own microscopic investigations of drinking water from a cistern used by Guinea worm sufferers. Fedchenko’s great breakthrough was actually to observe the larvae inside the copepod. This experiment was repeated by Sir Patrick Manson in 1895, using copepods from a pond in a London park.\(^9\) These findings provided proven methods for breaking the disease transmission cycle, by either

\(^7\) Hopkins, ‘Eradication’, op. cit., note 5 above, p. 95.

\(^8\) Susan Watts, ‘Seasonality and dracunculiasis transmission: the relevance for global eradication’, *Health Policy and Planning*, 1994, 9: 279–87.

\(^9\) A P Fedchenko, ‘Concerning the structure and reproduction of the guinea worm (*Filaria medinensis* L.)’, *Am. J. trop. Med. Hyg.*, 1971, 20: 511–23 (trans. by Emily Naust); Patrick Manson, ‘On the Guinea-worm’, *Br. med. J.*, 1895, ii: 1350–1.
filtering or boiling the drinking water or preventing Guinea worm infected people from entering a drinking water source. Such preventive strategies, together with the fact that there are no known non-human disease reservoirs, made a global eradication programme feasible.

The search for records of Guinea worm in the Americas has been encouraged by the World Health Organization. As part of the global eradication programme, the WHO has co-ordinated efforts to document the disappearance of the disease on a country by country basis. This effort has uncovered very few documentary sources not listed in recent bibliographies. In the Americas, WHO classified some countries as belonging to group C, “countries and territories with a possible history of endemic dracunculiasis”; these included Brazil, Colombia, Cuba, the Dominican Republic, French Guiana, Grenada, Guyana, Haiti, Mexico and Surinam. In December 1996, Cuba was certified free of the disease after a consultant’s visit and a thorough review of historical documents, current health provisions and surveillance activities. In 1997 and 1998 further countries in the Americas were similarly certified, including Barbados, Brazil, Colombia, the Dominican Republic, Trinidad and Tobago, Grenada, Jamaica and Mexico.

An assessment of written evidence for dracunculiasis is facilitated by the fact that the pathology is distinctive, with the swelling and watery bleb followed by the emergence of the long, white, threadlike worm. Thus, reports of the phenomenon by Western-trained doctors or observant lay people in printed or manuscript sources can be accepted with a high degree of confidence in the diagnosis. It is, however, important to determine the provenance of such accounts by referring whenever possible to the original rather than relying on later compilations. Until the late nineteenth century, medical authors writing the history of disease often uncritically, or inaccurately, repeated the observations of earlier authorities, and tended to give them as much credence as more recent ones.

In endemic areas, dracunculiasis most commonly affected poor people, who did not have access to protected water sources. From the vantage point of European medical knowledge in the eighteenth and nineteenth centuries, Guinea worm was a

10 Ralph Muller, ‘Bibliography’, in Workshop on opportunities for control of dracunculiasis; contributed papers, Washington, D.C., June 16–19 1982, Washington, DC, National Academy Press, 1985, pp. 1–176, and V A Inglis and R T Leiper, ‘Bibliography of dracunculiasis’, J. Lond. Sch. trop. Med., 1912, 2: Suppl. 1, 1–24.

11 WHO, Criteria, op. cit., note 2 above, p. 14.

12 WHO, ‘Dracunculiasis’, op. cit., note 2 above, pp. 33–4; WHO, ‘Dracunculiasis: certification of transmission-free status’, Wkly Epidem. Rec., 1998, 73 (10): 68–9.

13 Compare, J G Bremser, Traité zoologique et physiologique sur les vers intestinaux de l’homme, trans. from the German by Grundler, Paris, C L F Panckouke, 1824, pp. 214–48, with the later compilations by Hirsch and Cobbold which incorporated Fedchenko’s findings; August Hirsch, Handbook of geographical and historical pathology, trans. of 1881 rev. ed. by Charles Creighton, London, New Sydenham Society, 1885, vol. 2, pp. 337–59; T Spencer Cobbold, Parasites: a treatise on the entozoa of man and animals, London, J & A Churchill, 1879, pp. 216–27. The bibliographies in Hirsch and Cobbold appear to be the basis for most of the sources in the later bibliographies of Muller, and Inglis and Leiper, op. cit., note 10 above.
"tropical" disease, found in areas politically and socially peripheral to the metropolitan centres. Until the beginning of the global eradication programme, official records vastly underestimated the number of people infected.¹⁴

There are key questions which need to be asked of reports suggestive of the presence of dracunculiasis infection, and of possible local transmission at a particular place and time: (1) what link did the parts of the Americas and Caribbean reporting dracunculiasis have with the trade in slaves from Africa? (2) what were the African source areas for people with the infection? and (3) what local conditions in the Americas favoured transmission? Figure 2 and Table 1 provide information on the locations where dracunculiasis was noted and transmitted, together with the dates and sources of the observations.

**The Guinea Worm crosses the Atlantic**

Accounts of dracunculiasis generally agree that the worm was brought to the Americas primarily by slaves, although a few white crew members may also have

¹⁴ Watts, op. cit., note 1 above; Susan Watts, ‘Perceptions and priorities in disease eradication: dracunculiasis eradication in Africa,’ *Soc. Sci. Med.*, 1998, 46: 799–810.
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**Table 1**
Records of dracunculiasis in South and Central America before 1900

| Transmission recorded | Place, if known | Sources and dates of recorded transmission |
|-----------------------|-----------------|------------------------------------------|
| Brazil                | Inland from Bahia (San Salvador) town of Ferra de Santa Anna & parish of San José | Pereira\(^1\) and Da Silva Lima\(^2\) (1849–1865) |
| Cuba                  | sugar plantations town of Samaa | Dumont (1876)\(^3\) Oxamendi (1864)\(^4\) |
| Grenada               | St George’s Parish Port Saline | Chisholm (1787–1794)\(^5\) |
| Netherlands Antilles  | Curaçao          | Bremser (c. 1820)\(^6\) Dampier (early 1680s)\(^7\) |
| St Vincent            | plantation       | Chisholm (1793)\(^8\) |

**Possible transmission**

| Barbados              | coffee plantation | Hillary (1750s)\(^9\) |
|-----------------------|-------------------|-----------------------|
| Surinam               |                   | Ferg (1801)\(^10\) |

**Records of dracunculiasis**

| Argentina             | Rio de la Plata (Buenos Aires) | Hoeppli (1594)\(^11\) |
|-----------------------|---------------------------------|-----------------------|
| Brazil                | Rio de Janeiro                  | Sigaud (1817, 1819, 1831)\(^12\) |
| Colombia              | Cartagena                       | Gumilla (1740/1)\(^13\) |
| Dominican Republic    |                                  |                        |
| Saint Domingue (Haiti)|                                  |                       |
| French Guiana         |                                  |                        |
| Jamaica               | Spanish Town                    |                        |
| Mexico                |                                  |                        |
| Surinam (Dutch Guyana)|                                  |                        |

Except for the islands of Jamaica and St Vincent, all the above countries are WHO group C.

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1. Pereira, op. cit., note 51 of main text.
2. Da Silva Lima, op. cit., note 37 of main text.
3. Dumont, op. cit., note 38 of main text, pp. 12–15. map p. 8.
4. Oxamendi, op. cit., note 50 of main text.
5. Chisholm, 'On the malis dracunculus', op. cit., note 58 of main text.
6. Bremser, op. cit., note 13 of main text, pp. 214, 217.
7. Dampier, op. cit., note 4 of main text, vol. 2, pt 2, pp. 89–90.
8. Chisholm, 'On the malis dracunculus', op. cit., note 58 of main text.
9. Hillary, op. cit., note 71 of main text, pp. 229–30.
10. Ferg, 'Remarques sur les insectes de Surinam dont la piqûre est nuisible', Bibliothèque Médicale, 1814, 43: 100, as reported by Hirsch, op. cit., note 13 of main text, vol. 2, p. 354.
11. Hoeppli, op. cit., note 15 of main text, p. 131.
12. J F X Sigaud, Du climat et des maladies du Brésil, Paris, Fortin Masson, 1844, p. 133.
13. Gumilla, op. cit., note 33 of main text.
14. Père, op. cit., note 17 of main text.
15. Mongin, op. cit., note 34 of main text.
16. Poupé Desportes, op. cit., note 27 of main text.
17. Bajon, op. cit., note 28 of main text, pp. 321–29.
18. Williamson, op. cit., note 39 of main text, p. 408.
19. Van Leent, 'Contributions à la géographie médicale: les possessions Néerlandaises aux Indes Occidentales: La Guyane Néerlandaises', Arch. Méd. Nav., 1881, 25: 81–102, p. 83. Source cannot be verified as being a personal observation or that of contemporary informants.
20. Fermin, op. cit., note 27 of main text, pp. 333–4.
21. Bancroft, op. cit., note 27 of main text, pp. 388–9.
Figure 3: Areas of West Africa in which dracunculiasis was reported or known to exist, 1985.

Source: Watts, op. cit., note 1, p. 120.

been infected.\textsuperscript{15} Reported cases reflect the destination areas of slaves through time, and the source areas along the African coast and inland where the disease was found. Coastal districts of West Africa from where slaves affected by dracunculiasis are reported to have originated stretched from the Ivory Coast in the west to Nigeria in the east.\textsuperscript{16} Areas in which the disease was present or known to exist in 1985, in the early stages of the global eradication programme, are shown in Figure 3.

Dracunculiasis sufferers from the Guinea coast are mentioned in several records.\textsuperscript{17} In 1744, two doctors in Bermuda reported the case of a boy “lately brought from Guinea” whom they treated for dracunculiasis; they wound out many worms, and kept him on a strict diet, “by which Management he became a strong jolly young Lad”.\textsuperscript{18} In 1707, Sir Hans Sloane, writing of the British West Indian islands, commented: “The Blacks which come from Angola and Gambia are not troubled by them, but those from the Gold Coast [modern Ghana] very much.”\textsuperscript{19} The mouth of the Gambia is one of the northernmost points along the coast from which there are some early, scattered references to Guinea worm.\textsuperscript{20} South of the endemic area,
Angola is not on the WHO “provisional list of 70 countries and territories with a history of dracunculiasis in humans”.21

In the days of sail, a triangular trade along the routes of prevailing winds and currents brought slaves to the Caribbean, Brazil and North America, then took silver and gold, sugar, rum and molasses and other tropical produce along the coast of North America and across the Atlantic to the metropolitan centres in England, Holland, Spain, Portugal and France. Ships linking Portugal and Brazil engaged primarily in a two-way trade. Manufactured goods, chiefly firearms and cotton cloth, were transported along the African coast as far south as Angola to exchange for slaves.

The shortest journey made by slave ships from West Africa to Brazil took about six weeks, but the journey to Cuba, to Colombia and the isthmus of Panama might take twice as long. Such were the complexities of the trade, with ships sailing up and down the coast for weeks and even months, awaiting a full cargo of slaves, that it is possible that some slaves taken on board in a Guinea worm endemic area of West Africa were actually shipped to Brazil from Angola. Shipment from West Africa to the Caribbean might take two or three months. Thus, even the longest journeys allowed sufficient time for a worm to emerge from the leg of an infected slave long after he or she had arrived at an American destination.

The European sailors manning the slave ships are generally reported to have spent as short a period as possible ashore on the African coast, long known as the “White Man’s Grave”. Transmission of the worm from drinking water stored on board ship was possible, but unlikely, as infective cyclops in water collected from land would die within several days of being brought on board. Such was the image of Blacks in the eyes of Whites in the days of Black slavery that Whites were tempted to emphasize the extent to which dracunculiasis was a disease of Black people.22 However, in 1881, after an exhaustive review of existing documentation, August Hirsch (1817–1894), professor of medicine at the University of Berlin, stated emphatically that dracunculiasis “has been found among all races and nationalities, in all classes of society, at all periods of life, and in both sexes”, and that the sole reason for infection was drinking contaminated water.23

Written long before Hirsch’s book, some records did note cases among seamen landing in the Americas. One example was the crew of a Dutch ship arriving at Buenos Aires in 1599 from Amsterdam via the Guinea coast.24 Another was a report of an outbreak of dracunculiasis among sailors from Flushing, in the Netherlands, sailing via the Guinea Coast to the Netherlands Antilles.25 Louis Rouppe, in 1764,

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21 WHO, Criteria, op. cit., note 2 above, p. 14; the vast collection of eighteenth-century archives dealing with Angola may yet yield some information about this disease: David Birmingham, ‘Review article: Joseph Miller’s Way of death’, Past and Present, 1991, 131: 204–16, p. 209.

22 Kenneth F Kiple, The Caribbean slave: a biological history, Cambridge University Press, 1984, esp. ch. 10.

23 Hirsch, op. cit., note 13 above, vol. 2, p. 348; italics as in original.

24 Hoepli, op. cit., note 15 above, p. 131.

25 D H Gallandat, ‘Sur le dragonneau, ou veine de Médine’, J. Méd. Chir. Pharm. Paris, 1760, 12: 24–8.
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affirmed that Guinea worm infection could be present on ships, for example among crew members going from Amsterdam to the island of Curacao. However, reports from the French colony of Saint Domingue (later Haiti) in 1730, and from Surinam in 1769, explicitly stated that Blacks were affected by the Guinea worm, but not Whites. Similarly, the infection was noted among newly arrived African slaves in French Guiana in 1777.

When did the Worm arrive in the Americas?

The earliest known record of dracunculiasis in the Americas dates from 1599, among the Dutch sailors in Buenos Aires who had come from the Guinea Coast. The sixteenth century saw the beginning of the trade in African slaves; records suggest that 370,000 were brought across the Atlantic in that century and 1,870,000 in the next. However, the height of the slave trade occurred in the eighteenth and early nineteenth centuries; between 1701 and 1800 an estimated 6,130,000 slaves were taken from Africa. Thus, it is perhaps not surprising that records of dracunculiasis are more common during this period.

Several of the major points of entry for slaves, where they were fattened up after the horrors of the Middle Passage and then sold and re-exported, were recorded as having dracunculiasis cases. Writing of the island of Curacao, which contained the best natural harbour in the Caribbean, the British seaman, William Dampier reported that in the early 1680s “Guinea worms” were common among both Whites and “negroes” because the “island was formerly a Magazin of Negroes, while the Dutch drove that Trade with the Spaniards”. Another major port of disembarkation was Cartagena, on the Caribbean coast of Nueva Granada (Colombia); between 1595 and 1640, approximately half of the 268,000 slaves imported into Spanish America arrived there. In 1740/1, the Jesuit priest José Gumilla described dracunculiasis in Cartagena as “a source of suffering, though not frequently”.

26 Louis Roupppe, De morbis navigantium, Leyden, T Haak, 1764, pp. 282, 285.
27 [J-B-R] Pouppe Desportes, Histoire des maladies de S. Domingue, 3 vols, Paris, Lejay, 1770, vol. 2, pp. 270–1; Philippe Fermin, Description générale, historique, géographique et physique de la colonie de Surinam, Amsterdam, van Harrevelt, 1769, vol. 2, p. 333; Edward Bancroft, An essay on the natural history of Guiana in South America, London, T Becket and P A de Hondt, 1769, p. 388.
28 Bajon, Mémoires pour servir à l’histoire de Cayenne, et de la Guiane française, Paris, 1777, vol. 1, pp. 325–39.
29 Hoeppli, op. cit., note 15 above, p. 131.
30 Robin Blackburn, The making of New World slavery: from the baroque to the modern, 1492–1800, London, Verso, 1997, p. 377.
31 Dampier, op. cit., note 4 above, vol. 2, pt 2, p. 90.
32 Blackburn, op. cit., note 30 above, p. 143.
33 José Gumilla, Historia natural, civil y geográfica de las naciones del Orinoco, Colombia, 1740/41, quoted in Fernando A Beltrán-Hernández, ‘Absence of dracunculiasis transmission in the Americas’; a preliminary report submitted to the International Commission, Dracunculiasis Eradication Certification Division of Control of Tropical Diseases, WHO, by Communicable Disease Program (HCP/HCT PAHO/WHO. February 26, 1996), pp. 8–9.
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In 1730, 1770 and 1771 French doctors in Saint Domingue reported cases among recently arrived Africans. By 1791, there were on the island approximately 40,000 Whites, 28,000 mulattos (of mixed blood) and free Blacks, and 452,000 slaves, many only recently arrived from Africa. That year, however, saw the first successful Black freedmen rebellion in the Americas and with it the end of the institution of Black slavery on the island. With the sudden cessation of the importation of slaves from Guinea worm endemic areas in Africa, there were no more records of the disease.

Opportunities for the transmission of Guinea worm to the Americas further decreased with the British abolition of the slave trade in 1807. However, among Spanish, Portuguese and other privateers (such as those from Bristol, Rhode Island) who remained outside the range of British influence, the trade continued and, with it, the importation of Guinea worm. As in the past, a crucial variable was the place of origin and point of embarkation of the slaves. After 1807, in Brazil, slaves were more likely to be imported from the non-endemic areas of Angola and the Congo, than from the West African coast, as previously. Moreover, from mid-century, the official Brazilian policy of “whitening” its local population encouraged the emigration of free Whites from Europe. This policy was facilitated by the ending of slavery in Brazil in 1888. Writing in the 1870s, Dr José Francisco da Silva Lima contended that, despite the clandestine trade in slaves, the Guinea worm was by then rarely encountered.

Knowledge of Symptoms and Treatment in the Americas

The knowledge of Africans in the Americas about the symptoms and treatment of dracunculiasis reflected the experience they and their forebears had acquired during the time they lived in endemic areas of West Africa. Writing of Cuba in 1876, when slaves were still being smuggled in, Dr Henri Dumont noted that: “Blacks are skilled at identifying the disease which they can recognize with astonishing certainty. To many of their race who appear to be suffering from other infections that we [physicians] called recurring or chronic, they would say: ‘tu tienes sovia: tu es una filaria’.” Sovia is almost identical to the Yoruba term sobia, found today in Guinea worm endemic areas of western Nigeria.

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34 Pouppé Desportes, op. cit., note 27 above; Mongin, ‘Sur un ver trouvé sous la conjunctive à Maribarou, Isle Saint-Domingue’, J. Méd. Chir. Pharm. Paris, 1770, 32: 338–9; Péré, op. cit., note 17 above.
35 Paul Farmer, AIDS and accusation: Haiti and the geography of blame, Berkeley, University of California Press, 1992, p. 159.
36 Sheldon Watts, Epidemics and empire: disease, power and imperialism, London and New Haven, Yale University Press, 1997, pp. 249–50.
37 J F da Silva Lima, ‘De la filaire de Médine ou ver de Guinée rencontrée à l’état endémique dans la province de Bahia et de son introduction dans le corps humain par l’eau en boisson’, Arch. Méd. Nav., 1881, 35: 395–406, p. 396. The author’s name is incorrectly given at the head of the article as “Silva Lina”.
38 Henri Dumont, Comparative anthropology and pathology of black slaves, Colección Cubana de Libros y Documentos, inéditos o raros, vol. 2, Havana, 1922 (first published in 1876), section 33 transcribed in Certification of dracunculiasis eradication: country report, Cuba, prepared by Cuban Ministry of Public Health for WHO, October 1996, p. 14.
Several White observers recorded that Africans wound out the Guinea worm around a stick, taking care not to break it. John Williamson, noting cases in Spanish Town, Jamaica, in 1817, wrote that this procedure was “intimately known, and . . . well performed by the negro doctor in charge of the sick”.39 Other references to Africans winding out the worm were made by Pouppé Desportes in Saint Domingue in 1770, by Péré in 1774, and by Bajon in French Guiana in 1777.40 Dampier wrote that he had “known some that have been scarified and cut strangely, to take out the worm”.41 This is the only record of the use of surgical procedures in connection with this disease in the Americas.

Dampier also mentioned the use of a poultice made from the roasted root of white lilies, which he claimed was ineffective.42 Edward Bancroft (1744–1821), in 1763, reported the local use of a “cataplasm” or poultice, of “onions and bread, boiled with milk” applied to the swelling, and the recommendation that a mixture of powdered black pepper, bruised garlic and flour of sulphur “infused in a quart of rum, of which half a gill is to be drunk morning and evening”.43 The use of rum, made from West Indian grown sugar cane, sounds very like a local remedy, and, like the poultices, seems quite different from remedies used elsewhere. In the Americas, Africans would have had to undertake a new search for local plants which could be used for dressings, but they could still continue to use the method of extracting the worm, by winding it around a stick, which they had practised in Africa.

Transmission in the Americas
Evidence for local Transmission

By “local transmission” is meant the completion of the full cycle of the development of the Guinea worm in humans and in the body of host cyclops found in local ponds. There were commentators in the Americas who recognized that the occurrence of the disease in people who had not recently crossed the Atlantic might indicate local transmission. Some writers thought that the disease might be contagious, but a greater number suggested that certain drinking water sources could be implicated. Looking back at earlier records, it can be seen that those writers who mention the seasonality of the infection are clearly referring to cases of local transmission. In 1881, Hirsch, writing of the Americas, noted that Fedchenko had demonstrated the “transmissibility of dracontiasis or of the parasite which underlies it”, but that the conditions of such transmissibility could not yet be fully understood.44 It was not until the early twentieth century that the twelve-month disease cycle was definitively...
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established, and the relationship of this periodicity to the annual patterns of the disease, water supply and water use could be appreciated.\textsuperscript{45}

Cuba

Two independent records dating from the 1860s strongly suggest the transmission of dracunculiasis in Cuba. Slaves, who could act as reservoirs of infection, continued to be imported illegally into Cuba long after Spain signed the British sponsored agreement to abolish the slave trade in 1817. Though honoured more in theory than in practice, the agreement allowed for British slave inspectors to be stationed in Havana. Not until 1845 did Spain enact legislation to abolish the slave trade with Africa. After abolition the price of slaves shot up, making the trade more profitable, although risky. The now illicit importation of slaves into Cuba continued, reaching a peak in the late 1850s.

In 1866, Dumont reported that in the sugar plantations of the interior many cases occurred in summer and autumn “as if this were an epidemic disease”. He gave as an example the 13 sufferers recorded in the infirmary of the España sugar plantation on 1 September 1866, in the central area of Matanzas province, east of Havana; all had been admitted during July and August. Of the five women and eight men listed, four were of unidentified origin, eight had been born in Africa, and one was a Creole, a locally born slave of African descent.\textsuperscript{46}

The date of the Guinea worm cases at the España plantation is of particular interest. This plantation was opened up in the early 1860s by one of the wealthiest men in Cuba, the Spanish-born Julián de Zulueta, elevated to the rank of marquis in 1879. He was reported as importing over 1,800 slaves into Matanzas province in 1858, and more in 1864 and in 1865. On none of these occasions was the influential Zulueta prosecuted.\textsuperscript{47} The dracunculiasis cases at the España plantation were reported in 1866, the year after the first harvest, which would have been preceded by several years of work clearing the land, planting the sugar and installing a sugar mill. It was clearly in the economic interest of plantation owners to provide care for such cases; Laird Bergad suggests that after three years labour a slave had paid for himself or herself.\textsuperscript{48} The fact that the patients were of prime working age, between 17 and 27, suggests a motive for their admission to the plantation infirmary. On such a large plantation, with over 2,000 slaves, there may well have been other unrecorded cases among older slaves whose labour was less valuable or who were less seriously incapacitated.

Dumont considered the infection was transmitted “probably through contagion”, from person to person, and that the parasite resulted from “spontaneous

\textsuperscript{45} Tayeh, op. cit., note 5 above; Watts, op. cit., note 8 above; Sir Patrick Manson, ‘The life-span of Filaria medinensis’, Br. med. J., 1903, ii: 10.
\textsuperscript{46} Dumont, op. cit., note 38 above, pp. 8–9, 12–15.
\textsuperscript{47} Laird W Bergad, Cuban rural society in the nineteenth century: the social and economic history of a monoculture in Matanzas, Princeton University Press, 1990, pp. 127–9, 156, 226.
\textsuperscript{48} Ibid., p. 226.
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generation". Though he was wrong on both of these counts, for the purpose of this paper his report is important for its clues to local transmission. He wrote of the seasonality of occurrence, and of cases among Creoles as well as Africans, on sugar plantations inland.

Also in Cuba, in 1864, Dr Juan Oxamendi reported two European sufferers from Guinea worm—neighbours in the town of Sama, on the eastern end of the island. The first, a mason from Asturias (Spain), “who was working in a street where he would come into contact with emancipated blacks”, sought treatment from an African healer. The second worked in the Sama dry dock. Oxamendi made a special note of these two cases because of what he considered to be the extreme rarity of the disease among Europeans. That they were neighbours led him to conclude that the infection was contagious, the result of human contact. With hindsight, this observation is important as evidence for probable local transmission.

Curaçao

The island of Curaçao, a Dutch colony off the coast of Venezuela and a station for the trans-shipment of slaves, was often mentioned as a place where the disease was endemic. The evidence, though comparatively sparse, seems to support this claim. The most frequently quoted sources were the reports of the British seaman William Dampier (1652–1715), referring to the situation in the early 1680s, and of the German physician J G Bremser (1767–1827), writing in 1824. As noted above, Dampier reported that dracunculiasis was not confined to Blacks; for this reason many people thought that the Europeans caught the infection from Blacks. But he disagreed:

I rather judge that [the worms] are generated by drinking bad Water; and 'tis as likely that the Water of the other Island of Aruba and Bonariry may produce the same Effects; for many of those that went with me from thence to Virginia . . . were troubled with them after our

49 Dumont, op. cit., note 38 above, p. 15.
50 Juan C Oxamendi, ‘Guinea-worm in the white race’, Ann. R. Acad. med., Physical, Nat. Sci. Havana, 1864, I, transcribed in Certification, op. cit., note 38 above, pp. 10–11. Oxamendi’s work was mentioned in the bibliographies of Inglis and Leiper in 1912, and of Muller in 1985 (see note 10 above). However, this source and that of Dumont, remained inaccessible and untranslated for an audience beyond Cuba until the Cuba country report for certification of eradication was prepared in October 1996. Dr Pedro Pruna drew my attention to a recollection by the president of the Academy of Sciences of Havana, Nicolás José Gutierrez, in 1884, that he had observed a “Guinea dragon” in a woman’s eye in 1842; Ann. R. Acad. med., Physical Nat. Sci. Havana, 1884, 21: 85–7.
51 Küsenmuller, 1796, was quoted by Cobbold, op. cit., note 13 above, p. 218, and also by Manoel Victorino Pereira, ‘Transport de la filaire de Médine en Amérique par les nègres d’Afrique: preuves de son endémicité dans la province de Bahia, et de son introduction dans le corps humain par l’estomac’, Arch. Méd. Nav., 1877, 28: 295–302, p. 298. See also F Küchenmeister, On animal and vegetable parasites of the human body: a manual of their natural history, diagnosis, and treatment, trans. of 2nd German ed. by Edwin Lankester, London, Sydenham Society, 1857, vol. 1, p. 400.
52 Bremser, op. cit., note 13 above, pp. 214, 217; Dampier, op. cit., note 4 above, vol. 2, pt 2, pp. 89–90.
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arrival there: particularly I . . . had one broke out in my Ancle, after I had been there five or six Months.53

Dampier’s conclusion that he got his Guinea worm in Curaçao is persuasive, as he arrived in Virginia in July 1682, after being in the West Indies for over a year. Dampier, a “buccaneer, pirate, circumnavigator, captain in the navy, and hydrographer” is a more reliable observer than his career might suggest. He has been described as possessing “an almost unique talent for observing and recording natural phenomena. . . . and his treatment of the many other subjects which fell within his experience is perhaps equally good.”54 In 1764, Rouppe briefly mentioned the frequency of cases in Curaçao in his De morbis navigantium (The diseases of sailors).55

In 1824, Bremer wrote that a quarter of the population of Curaçao, both Blacks and Whites, suffered from Guinea worm.56 He obtained this information directly from Baron de Jaquin, who told him that two of his European companions on a voyage to Curaçao, who had never been to Asia or Africa, had become infected on the island. Jaquin was told by local people that drinking water could transmit the worm. As he was not accustomed to drinking alcohol, he admitted that he had no choice but to drink the local water. Yet he remained uninfected, while one of his companions, said to have been determined to drink only alcohol, was infected. On the basis of the Baron’s report, Bremer dismissed the local version of the origin of the worm.57 However, this account supports the thesis of local transmission of the disease.

Grenada and St Vincent

In the 1780s and 1790s, a remarkably thorough investigation by the physician Colin Chisholm (1755–1825) showed that transmission was taking place in the islands of Grenada and St Vincent. Chisholm’s report, ‘On the malis Dracunculus, or Guinea-Worm’, was published in 1815 in the Edinburgh Medical and Surgical Journal. It was a response to letters about a long footnote on Guinea worm which he had included in his book on yellow fever, first published in 1794.58 His evidence was based on the cases he had treated and information from people on affected plantations. On the basis of this empirical study, he came to the conclusion that certain sources of drinking water were the origin of the disease.

In Grenada, where he was practising, Chisholm noted that dracunculiasis was endemic on Mr Thornton’s plantation, in St George’s parish, Port Saline, from

53 Dampier, op. cit., note 4 above, vol. 2, pt 2, p. 90.
54 Dictionary of national biography, ed. Leslie Stephen and Sidney Lee, 21 vols, London, Smith, Elder, 1908–9 (hereafter DNB), vol. 5, pp. 455, 452.
55 Rouppe, op. cit., note 26 above, pp. 282, 285.
56 Bremer, op. cit., note 13 above, p. 214.
57 Ibid., p. 217.
58 Colin Chisholm, ‘On the malis dracunculus, or Guinea-worm’, Edin. med. surg. J., 1815, 11: 145–64, p. 145. See also his entry in DNB, vol. 4, p. 261; Colin Chisholm, An essay on the malignant pestilential fever introduced into the West Indian islands from Boullam, on the coast of Guinea, as it appeared in 1793, 1794, 1795 and 1796, 2nd ed., London, for J Mawman, 1801, vol. 1, pp. 56, 57n.
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1787 to 1794. Of the 300 sufferers, some had up to ten worms, and 50 were too debilitated to work. The infection occurred among “field negroes” (i.e. slaves) who had drunk well water; no cases occurred among Whites and “domestic negroes” who always drank rainwater stored in cisterns. As the parish had no springs or flowing streams, all domestic supplies came from wells or cisterns storing rainwater.

At a plantation in Grande Ance, Mr Scott also suspected that Guinea worm affected field slaves because they drank well water. He had cisterns built for rainwater, with the result that Guinea worm ceased to occur on the plantation. Following this example, Mr Thornton had cisterns built on his plantation as well, and it was reported that the disease died out thereafter.

In the island of St Vincent, not far from Grenada, Chisholm reported that a plantation proprietor noticed the disease for the first time in 1793. Sufferers were field hands who drank well water. Once again, house slaves who drank from rainwater cisterns did not become infected; the only European to be affected confessed to having drunk well water once or twice. Three hundred of the 500 labourers were incapacitated, for periods ranging from six to eight weeks.

In his analysis of cases observed in Grenada and St Vincent, Chisholm drew on medical literature on the transmission of the Guinea worm in India. He dismissed the view that the worm, in some form or another, penetrated the human skin, on the grounds that the islanders did not bathe in the suspect wells. Rather, people who drank water at particular sources, at shallow and brackish wells, became infected with the worm, but the disease no longer occurred when these drinking water sources were replaced by cisterns.

Chisholm’s interest in water as the source of transmission led him to examine it with a microscope, whereupon he discovered: “extremely minute and agile animalcules ... and of innumerable white granulated substances, little more than perceptible, even with the magnifier I used, which I concluded ... to be, the former the embryos, the latter the ova, of the dracunculi”.59 This appears to be the first attempt to use a microscope to identify Guinea worms at any stage of their development.

Chisholm believed that the Guinea worm had been recently established in both islands, and that the slaves brought it with them from Africa; the fact that locally-born Creoles were also subject to the infection proved to him that it was contracted on the islands. He noted the seasonal occurrence of the disease and was puzzled by what happened to the worm between the middle of March and the middle of November, when no emerging worms were seen.60 However, he could not appreciate that the seasonality of transmission was a clinching argument for local transmission. He identified Guinea worm as a disease of the dry and cool season in Grenada, together with pleurisies and catarrhal fevers; the rainy warm season being characterized by remittent fevers (i.e. malaria) and cholera morbus.61

59 Chisholm, ‘On the malis dracunculus’, op. cit., note 58 above, p. 150 and passim.
60 Ibid., p. 154.
61 Chisholm, An essay, op. cit., note 58 above, vol. 1, pp. 56, 57n.
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Brazil

Two doctors, Manuel Victorino Pereira and José Francisco da Silva Lima, provided evidence for the endemicity of dracunculiasis in the 1850s in north-east Brazil, in the hinterland of Salvador da Bahia, the former capital of Brazil. As a result of their interviews with local people a number of years after the cases occurred, they concluded that the infection was transmitted at certain ponds from which affected individuals had taken drinking water.

The work of these two doctors is also significant in the light of the prominent role they played in the Tropicalista School of Medicine which flourished between 1860 and 1890 in Salvador da Bahia. Dr José Francisco da Silva Lima (1826–1910) was born in Portugal and graduated in 1851 from the University of Bahia medical school, the oldest in Brazil. He began to practise medicine in the capital in 1853, and during the next thirty years made five trips to Europe. He is also known for important work on yellow fever and beriberi. Silva Lima’s younger colleague, Manuel Victorino Pereira (1853–1902), worked with Wucherer on hookworm. He made the control of this disease, and the improvement in rural health conditions, part of a political programme, thus combining his medical research and practice with political concerns, especially the struggle against slavery and the monarchy. Pereira became vice-president of Brazil after the Republic was established in 1889.

Other well known physicians in the Tropicalista group included Dr Otto Wucherer (1820–1873), who gave his name to the filarial worm, Wuchereria bancrofti, and Dr John L Paterson (1820–1882). Paterson, a Scottish-born physician and graduate of Aberdeen University practising in the town of Salvador, translated Silva Lima’s paper on dracunculiasis from Portuguese and sent it to T Spencer Cobbold, professor of helminthology at the Royal Veterinary College in London, who had it published in the *Veterinarian* in 1879; it was published in French in 1881.

Paterson and Wucherer had been the first to identify cases of yellow fever in 1849 in Bahia. Members of the medical elite in Rio de Janiero, who did not wish to recognize the return of the disease to Brazil, accused these doctors of being “meddlesome foreigners”. The rivalry which developed between the two groups of physicians arose from different views of disease causation. The medical establishment, based in the capital Rio de Janeiro, clung to older generalized environmental ideas of disease

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62 Julyan G Peard, ‘Tropical disorders and the forging of a Brazilian medical identity, 1860–1890’, *Hisp. Am. hist. Rev.*, 1997, 77: 1–44; *idem*, ‘Tropical medicine in nineteenth century Brazil: the case of the “Escola Tropicalista Bahiana”, 1860–1890’, in David Arnold (ed.), *Warm climates and western medicine: the emergence of tropical medicine in 1500–1900*, Amsterdam, Rodopi, 1996, pp. 108–32; *idem*, ‘Medicina tropical en el Brazil del siglo XIX: la “Escuela Tropicalista Bahiana”, 1860–1890’, in Marcos Cueto (ed.), *Salud, cultura y sociedad en América Latina*, Lima, Instituto de Estudios Peruanos and PAHO, 1996, pp. 31–52; Nancy Stepan, *Beginnings of Brazilian science: Oswaldo Cruz, medical research and policy, 1890–1920*, New York, Science History Publications, 1976, esp. ch. 3, ‘Medicine in Brazil: nineteenth century background’. 63 Silva Lima, op. cit., note 37 above, p. 395, note 1. 64 Donald B Cooper, ‘Brazil’s long fight against epidemic disease, 1849–1917, with special emphasis on yellow fever’, *Bull. N. Y. Acad. Med.*, 1975, 51: 672–96.
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causation related to swamps, bad air, and the direct rays of the sun. The doctors in Salvador rejected this view and began to study the pattern of occurrence of specific diseases, especially those they recognized as characteristic of the tropical environment of north-east Brazil. They gradually began to use the most up-to-date techniques in haematology, microscopy and autopsy, keeping up with European research through visits and correspondence. Their work on yellow fever, beriberi and hookworm was of global, as well as national importance. They published their findings, and those of European researchers, in the *Gazeta Médica da Bahia*. However, ultimately, their work contributed little to the modern foundations of Brazilian medicine, established in the 1880s and 1890s by Oswaldo Cruz who, unlike the Tropicalistas, was able to set up a government funded research institution. By then, as Peard points out in a recent paper, “the Tropicalistas' attempt to define the idea of the tropics and tropical disorders had been eclipsed by a tropical medicine and a racial determinism constructed by the colonial nations of the more developed world.”

In 1850, as a young student, Silva Lima was consulted by Antonio Francisco d'Oliveira, a Portuguese lawyer living in Juazeiro, on the São Francisco River, about 500 km inland from Salvador, for treatment of a pre-emergent Guinea worm. In the same year, Silva Lima treated Antonio's brother, Manoel, for an emerging worm. In 1852 Antonio wrote to Silva Lima that he was convinced that he and several of his companions had been infected in April 1849 on a journey from Salvador to Juazeiro, after drinking water from a pond near the town of Feira de Santa Anna, about 50 km from the coast.

These cases of dracunculiasis occurred in 1850, soon after the outbreak of yellow fever in Salvador in September 1849, which caused at least 3,000 deaths in the city. At that time, Silva Lima was still a student, and was probably busy treating yellow fever patients, which may explain why he was unable to follow up the Guinea worm cases. However, in 1869, after the death of Antonio and the departure of his brother Manoel for Portugal, he discovered that there were still five people living in Juazeiro who had made the fateful journey from the coast in 1849. In an interview in 1869, he discovered that six of the nine people in that caravan, and three others in a later convoy, had been infected at the same pond. Both caravans had been the first to use the route through the area after the first rains of the year.

Francisco, "un Africain", travelling with the d'Oliveira brothers, told Silva Lima that, according to tradition, the Guinea worm was known to have existed near Feira de Santa Anna, in two ponds along the route to Jacuipe, at São José, and at Pojuca. The convoy had stopped and drunk the water at Pojuca, where d'Oliveira thought

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65 Peard, 'Tropical disorders', op. cit., note 62 above, p. 44 and passim; see also Peard, 'Medicina tropical', op. cit., note 62 above.  
66 Silva Lima, op. cit., note 37 above, pp. 397–8. Julyan Peard has also identified further papers by Silva Lima: 'Pathologica historica e geographica nos nosologia das boubas, do maculo e dascontiasse no Brasil: causas de sua atual raridade ou extinção', *Gazeta Médica da Bahia*, 1890–91, 22: 297–305, 337–45, 385–96, 433–44, 481–91, 533–40.  
67 Cooper, op. cit., note 64 above, p. 676.
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he had contracted the disease. Francisco had advised the travellers not to bathe in either ponds, or drink the water unless it had first been boiled or filtered. Other informants gave similar testimony.

Silva Lima had observed only 3 cases of dracunculiasis during 26 years of practice, and he considered Guinea worm by then a pathological curiosity. Although over 20 years separated the events of the journey and the time they were recalled, he found the evidence convincing and argued for local transmission as a result of drinking water at certain sites around the town of Feira de Santa Anna, most commonly during the first rains, immediately after the dry season. He stated that the disease was first brought to coastal Brazil by slaves, and that the parasites must have moved from the coast to "poison the waters" around Feira de Santa Anna. The leading British parasitologist, Cobbold, noted that the Brazilian findings were compatible with the definitive study recently published by Fedchenko.68

In 1877, Victorino Pereira mentioned in an article the findings of his colleague, Silva Lima, and provided information about other cases. He noted one case, in 1871, of a man who had recently arrived from West Africa in a secret cargo of slaves. In 1876, in Feira de Santa Anna, he saw a dracunculiasis scar on the heel of a free mulatto woman. His informants told him that local people still recommended that travellers crossing the River Pojuca, three leagues from Feira de Santa Anna, or the River Jacuhy (Jacuipe) a short distance to the east, should not drink that water.

Pereira discovered a report by Dr Cabussú, who had practised in Feira de Santa Anna, of fifty cases of Guinea worm in 1865–6 in the town and in the nearby parish of São José. Cabussú wrote that the disease attacked mainly Blacks, but without distinction of sex or nationality (i.e. place of origin in Africa), and that since that year there had been few cases. He identified certain ponds in the area which were locally reputed to be sources of infection, and he believed that the infection was transmitted by drinking the water.69

Pereira and Silva Lima both found that Cabussú's findings were convincing, and that they reinforced their own argument for local transmission of the worm through drinking the water at certain sources.70 It seems reasonable that transmission could have been sustained, through one or more visits by infected travellers, during the fifteen year period from about 1849 to the mid-1860s at certain points along the well-travelled land route between the provincial capital of Salvador and the important area of inland settlement along the São Francisco River.

With hindsight, we can also recognize the seasonality of the occurrence of the disease, noted by Silva Lima, as evidence for transmission. Transmission in north-east Brazil appeared to occur during the wet season when ponds were flooded by

68 Silva Lima, op. cit., note 37 above, pp. 400–3; Cobbold's comment is appended as a footnote on p. 397.
69 Pereira, op. cit., note 51 above, pp. 301–2.
70 Silva Lima, op. cit., note 37 above, pp. 402–3; Pereira, op. cit., note 51 above, p. 302.
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the first rains, in contrast to the situation in Grenada, where transmission occurred in the dry season.

Possible Transmission Sites

There are a number of accounts which suggest the possibility of local transmission in other areas. Compared to those above, they are brief and incomplete and thus do not provide conclusive evidence.

In Barbados, William Hillary (d. 1763), who practised medicine there between 1752 and 1758, noted the many authorities who claimed that:

This disease proceeds from drinking the water of stagnating ponds, in hot countries, after droughts and sultry hot seasons, wherein the ova or animacula of this worm are contained . . . I am informed that there are some stagnating ponds, in this island, the washing in or drinking the waters of which is subject to generate this worm.71

Unfortunately, he did not take the trouble to investigate these suspect ponds, so his suggestion remained unverified. He saw cases in Barbados, and described the extraction of the worm around a stick.72

Pereira mentioned a statement about the endemicity of Guinea worm in Demerara, now known as Georgetown, the capital of Guyana. This was taken from the section on Guinea worm written by G Busk in A system of surgery, edited by Timothy Holmes; it does not occur in Busk’s other papers.73

In 1881, August Hirsch reported observations by Ferg, in 1801, concerning nearly 200 cases of “filariae” on a coffee plantation in Surinam. Ferg noted that the only thing all sufferers had in common was that they drew drinking water from a spring “which had been the true cause of the outbreak”. Presumably this evidence is the reason why Hirsch, who knew of Fedchenko’s recent work, accepted the disease as dracunculiasis.74

The Twentieth Century

In the early twentieth century, British colonial records reported scattered cases of Guinea worm among immigrants and travellers in the Caribbean and Guyana, but provided little supporting evidence for the diagnosis. In Jamaica, seven cases were listed in 1911–12.75 Returns from British Guiana (now Guyana), in 1910–11, record 152 cases of dracunculiasis, but no cases of hookworm, which was next on the list

71 William Hillary, Observations on the changes of the air, and the concomitant epidemic diseases in the island of Barbados (lst ed. 1759), Philadelphia, B and T Kite, 1811, p. 230.
72 Ibid., p. 229.
73 Pereira, op. cit., note 51 above, p. 298.
74 Hirsch, op. cit., note 13 above, p. 354; Ferg, ‘Remarques sur les insectes de Surinam dont la piqûre est nuisible’, Bibliothèque Médicale, 1814, 43: 100. The original source by Ferg has not been examined.
75 Colonial Medical Reports—No. 31—Jamaica. J E Ker, ‘Medical report for the year 1912’, J. trop. Med. Hyg., 1914, 17: 31–40, table showing ‘Return of diseases and deaths in 1911–12 in the various hospitals of Jamaica’, p. 35.
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and discussed in the text. This report was probably a clerical error, in which the figures for Guinea worm and hook worm were transposed. The physician A T Ozzard wrote that in British Guiana, in 1904, “a few cases have been described, but it is decidedly rare”.

A source for imported cases was suggested in a report from Trinidad, in 1901, of dracunculiasis in “two newly arrived E. Indian immigrants”. It is likely that some Indian immigrants to British Guiana and to British West Indian islands came from areas of the Indian subcontinent endemic for dracunculiasis. Beginning in the late nineteenth century, indentured labourers were brought from the Indian subcontinent to work on plantations; following the revolt of compulsorily apprenticed freed slaves in Jamaica, in 1865, plantation owners did not see Black labour as reliable and sought for suitable, malleable replacements.

Later twentieth-century cases were noted in Cuba among students from endemic areas of Africa: 25,000 students from Africa studied in Cuba between 1979 and 1991. A thorough surveillance of the students identified fifteen cases between 1980 and 1992 among males aged between 13 and 37 from the endemic countries of Ghana, Sudan, Ethiopia, Burkina Faso, Mali and Benin. The infected immigrants’ worms appeared in Cuba at the same time of year as they would have appeared in Africa, depending on whether they were from the coastal zone or the interior. In January 1997, the WHO certification committee assessed the risk of the establishment of local transmission in Cuba as “almost zero” because of the effective system of surveillance for immigrants, the network of primary healthcare facilities, and protected rural water supplies.

Guinea Worm Disease north of the Rio Grande

The WHO list of group C countries, those with a possible history of endemic Guinea worm, does not include the USA. However, in the light of our discussion of records in South and Central America, it is worth examining the few existing US records in more detail. A recent report, in 1997, of a worm in a nine-year-old Sudanese girl in Nashville, who had recently left the war-torn endemic area of southern Sudan, is believed to be the first reported case in an immigrant in recent years.

76 Colonial Medical Reports—No. 32—British Guiana. J E Godfrey, ‘Medical report for the year 1910–1911’, J. trop. Med. Hyg., 1914, 17: 41–50, table showing ‘Return of diseases and deaths in 1910 in the public hospitals, British Guiana’, p. 42.
77 A T Ozzard, ‘Notes on the tropical diseases of British Guiana’, J. trop. Med. Hyg., 1904, 7: 357–9.
78 ‘Trinidad—Annual report of the Surgeon-General, for 1900’, J. trop. Med., 1901, 4: 207.
79 James Walvin, Black ivory: a history of British slavery, London, Fontana, 1993, pp. 331–2.
80 Burbano Diago, et al., op. cit., note 6 above, pp. 4–5.
81 WHO, ‘Dracunculiasis’, op. cit., note 2 above.
82 WHO, Criteria, op. cit., note 2 above, p. 14.
83 Michele Spring and Paul Spearman, ‘Dracunculiasis: report of an imported case in the United States’, Clin. Infect. Dis., 1997, 25: 749–50.
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B G Chitwood, writing in 1933, examined ten reported cases of dracunculiasis in humans in the USA, of which he considered that only four were actually dracunculiasis. All four cases originated overseas; a white sailor en route to New York (reported in 1860); two seamen, one English and one Danish, visiting Charleston, South Carolina (reported in 1874), and the fourth, an African from the Gold Coast, reported in 1901.84 In 1682, Dampier and fellow seamen suffered from Guinea worms on their arrival in Virginia.85 In the light of the above examples, it is likely that only occasional cases of dracunculiasis occurred in and around ports on the Atlantic littoral of North America which imported slaves from Africa and the Caribbean.

The major factor militating against the actual transmission of the disease north of the Rio Grande was cooler climatic conditions which would be very unlikely to allow the survival of the larvae in drinking water sources, even if cyclops were present. The number of slaves, possible sources of infection, imported into what is now the USA, was much lower than in lands to the south; but it would have taken only one infected person to release thousands of larvae into a water source.86

Conclusion

The primary sources, originally in English, French, Spanish and Portuguese, indicate a number of places in which dracunculiasis was recorded among slaves brought from Africa, and more rarely, among seamen. The records provide evidence for the survival of African knowledge of the infection and methods of treatment, which were spread through the Black diaspora. There were frequent comments about Black healers who wound the emerging worm slowly around a stick, and were careful not to break it; this method of treatment is widely recorded in endemic areas in Africa and India.

As a result of the effective enforcement of laws against the trade in slaves with Brazil and later with Cuba, the supply of new infections in these two countries gradually dried up and there is no further evidence for local, indigenous transmission. It thus seems that Pereira and Silva Lima, in Brazil, writing originally in the late 1870s, and Cuban epidemiologists writing in 1905, were broadly correct in stating that the infection disappeared from their countries with the end of the slave trade.87

A few locations where the infection was actually transmitted at local water sources, though perhaps for only a short period of time, can be identified. In

84 B G Chitwood, ‘Does the Guinea-worm occur in North America?’, J. Am. med. Ass., 1933, 100: 802–4; see also Muller, op. cit., note 5 above, pp. 104–5.
85 Dampier, op. cit., note 4 above, vol. 2, pt 2, p. 90.
86 Between 1500 and 1870, slaves imported into the USA comprised 6 per cent of the total number brought into the Americas; Robert William Fogel and Stanley L Engerman, Time on the cross: the economics of negro slavery, Boston, Little Brown, 1974, p. 14.
87 Pereira, op. cit., note 51 above, pp. 296–7; Silva Lima, op. cit., note 37 above, p. 396; Certification, op. cit., note 38 above, p. 5.
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drifting in these places, transmission occurred at suitable unprotected ponds or shallow wells containing host cyclops which were used as drinking water sources at a particular season. In north-east Brazil, the reputation of affected water ponds as sources of the Guinea worm, which local people therefore considered should not be used, is likely to have hastened the end of transmission. In Grenada and St Vincent, Chisholm reported that the disease disappeared on two plantations after the owners constructed rainwater cisterns as an alternative to wells. As demonstrated by the documentation of recent cases in Cuba, the arrival in tropical America of a small number of infected travellers from endemic areas is not sufficient, of itself, to establish a new focus of infection.

For its local transmission, Guinea worm requires very specific and environmental and human settings, compared to the settings which facilitated the transmission of smallpox, malaria, yellow fever, syphilis and measles in the Americas, after their transfer across the Atlantic. Today, the focality of Guinea worm disease is a key factor in ensuring rapid progress towards the global eradication of this painful and debilitating affliction.

The physicians who wrote the most extensive reports on the disease in South America and the Caribbean islands carefully observed symptoms of the infection and emerging worms, and noted treatment and possible means of transmission. As independent practitioners, they treated a wide range of patients, slaves, recently emancipated Africans and free citizens of European origin. As physicians working outside the social and climatic context to which they were accustomed, their empirical observations were likely to be guided by a consideration of the actual environmental conditions they observed, rather than by abstract concepts of "miasma" or "contagion".

Some of the doctors whose reports are discussed here later became well known for their contributions to medicine, or other scientific endeavours: for example, among British physicians Colin Chisholm, William Hillary, Edward Bancroft, and perhaps best known of all, Sir Hans Sloane, founder of the botanic garden at Chelsea, whose collection of books, manuscripts and natural history specimens became the nucleus of the British Museum. For Chisholm and Silva Lima concern for dracunculiasis came second to their interest in more lethal diseases, especially yellow fever. Chisholm's careful study resulted in an interpretation of dracunculiasis which appears surprisingly modern for the 1790s, and it became widely known among physicians in Britain and in India. However, during the following eighty years, his findings on the role of drinking water in Guinea worm transmission remained only one of several hypotheses about the transmission of the disease. The later work of Silva Lima and Pereira was readily accepted by researchers in Europe as it supported the recent findings of Fedchenko. The contribution of the doctors in Salvador da Bahia can also be seen as an aspect of the concern of members of the Bahian Tropicalista School of Medicine to

88 Pereira, op. cit., note 51 above, p. 301; Silva Lima, op. cit., note 37 above, p. 402.
89 Chisholm, 'On the malis dracunculus', op. cit., note 58 above, p. 148.
90 Chisholm, DNB, vol. 5, p. 261; Hillary, DNB, vol. 9, p. 880; Bancroft, DNB, vol. 1, pp. 1025-6; Sloane, DNB, vol. 18, pp. 378-80.
improve the health of all the population, and to build up Brazil's reputation as a modern society, rather than a poor, tropical backwater characterized by devastating tropical diseases. Thus, this study of the end of dracunculiasis in the Caribbean and South America can be seen in the context of the history of health in the tropics, as well as an early step towards the global eradication of the disease.