

Lymphatic Filariasis in the Americas



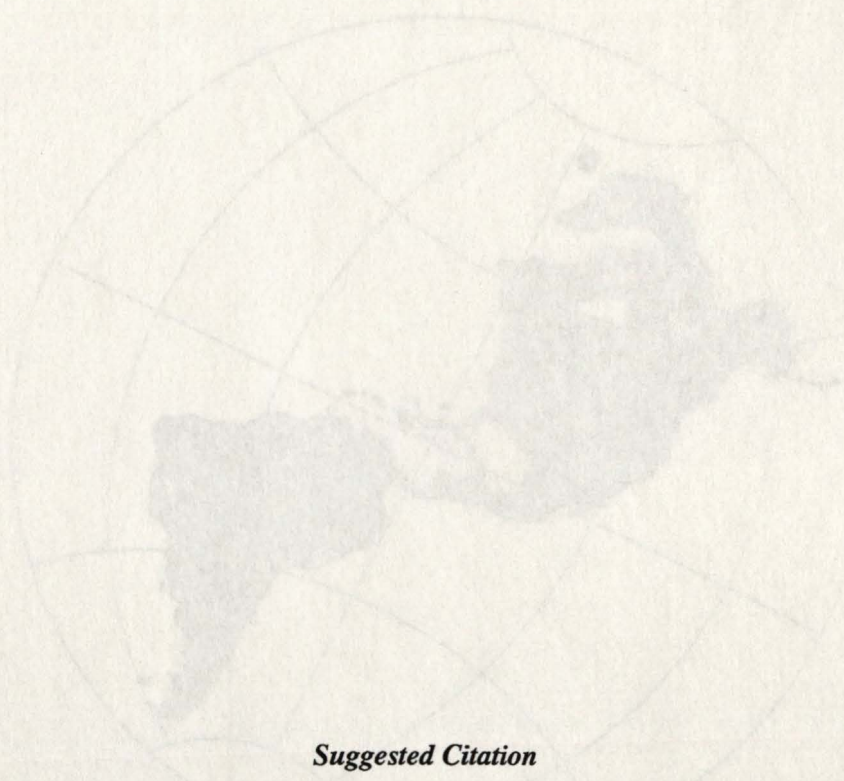
• *An Epidemiologic History* •

Lymphatic Filariasis in the Americas



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Preface

THE EPIDEMIOLOGY of lymphatic filariasis in the Americas is intimately linked to the social, political, and economic history of the region. After becoming established in Asia centuries ago, the filarial parasite *Wuchereria bancrofti* spread westward to Africa and the Middle East as a consequence of human migration. Forced migration of slaves from Africa first brought *W. bancrofti* to the Americas during the 1600s, where it flourished under conditions of human misery, poverty, and environmental degradation. Extensive migration, both forced and voluntary, facilitated transmission of the parasite throughout the Caribbean and along the Atlantic coast. Later immigrants from other filariasis-endemic areas, particularly Asia, were most certainly infected with *W. bancrofti*, although the degree to which they introduced or reintroduced transmission of the parasite in the Americas is unclear.

From the perspective of the parasitologist, *W. bancrofti* is an elegant and fascinating parasite. However, the total human suffering that results from infection with this organism can scarcely be imagined. In the Americas alone, up to 1 million people are infected in four countries: Haiti, the Dominican Republic, Guyana, and Brazil. If global estimates hold true in the Americas, then approximately 200,000 men in this region suffer from filarial urogenital disease (hydrocele, chylocele, lymphedema of the penis and scrotum) and 100,000 persons, most often women, suffer from lymphedema or elephantiasis of the leg.

Thanks to recent developments in diagnosis and antifilarial drug therapy, as well as advances in managing the chronic manifestations of lymphatic filariasis, the World Health Assembly passed a resolution in May 1997 that called for the "global elimination of lymphatic filariasis as a public health problem." In December of that same year, the World Health Organization (WHO) and the Pan American Health Organization (PAHO) designated the U.S. Centers for Disease Control and Prevention as a Collaborating Center for Control and Elimination of Lymphatic Filariasis in the Americas. As a Collaborating Center, CDC was charged with providing information to WHO, PAHO, and member countries on the distribution and epidemiology of lymphatic filariasis in the Americas. This book is a result of that effort.

We began our search with known published and unpublished literature on the epidemiology of lymphatic filariasis in the Americas. For each article we found, we searched for any cited references that seemed relevant. Our efforts received a tremendous boost when we discovered the website <http://nema.cap.ed.ac.uk/fgn/BiblioFil/biblioFil.html>, an exhaustive database on the published literature on human filariasis, originally assembled by Ralph Muller of the International Filariasis Association and Christian Back, and continued by Mark Blaxter and Chris Booth, from the Institute of Cell, Animal, and Population Biology (ICAPB), Ashworth Laboratories, at the University of Edinburgh, Scotland, U.K. Their efforts were sponsored by the Edna McConnell Clark Foundation, and the database they produced is a veritable treasure trove for anyone interested in lymphatic filariasis. We also received assistance from numerous colleagues in filariasis-endemic countries in the Americas, and from Dr. Eric Ottesen at WHO, who provided us with unpublished documents on the subject.

In all, we identified approximately 700 references that we consider relevant to the epidemiology of lymphatic filariasis in the Americas. These are on file at the Division of Parasitic Diseases, National Center for Infectious Diseases, CDC. An electronic copy of the citation database is available on request, and paper copies of the individual documents and papers are also available to interested persons. Requests for information should be sent to WHO/PAHO Collaborating Center for the Control and Elimination of Lymphatic Filariasis in the Americas, Division of Parasitic Diseases (DPD), Mailstop F22, Centers for Disease Control and Prevention (CDC), 4770 Buford Highway NE, Atlanta, GA, USA 30341.

This book is intended for public health officials, lymphatic filariasis program directors, and others who are interested in the epidemiology or history of lymphatic filariasis. It is also intended to be a reference for persons actively involved in the elimination of lymphatic filariasis in the Americas. Knowledge of the distribution of *W. bancrofti* in the past is needed not only for countries with ongoing transmission of the parasite, but also for those in which transmission is currently considered unlikely.

To certify that *W. bancrofti* has indeed been eliminated from this hemisphere, all countries in which transmission has occurred will have to document that transmission has ceased. The most fruitful areas to search for ongoing – yet unrecognized – transmission are likely to be those where transmission was known to occur previously.

Of the seven human filarial parasites, four currently are transmitted in the Americas, including *W. bancrofti*, *Onchocerca volvulus*, *Mansonella ozzardi*, and *Mansonella perstans*. This review focuses on *W. bancrofti*. The relevance of these other parasites for the purpose of this book is limited to the potential for diagnostic errors, in which the microfilariae of *M. ozzardi*, for example, may have been confused by some authors for those of *W. bancrofti*. The microfilariae of both species are found in the peripheral blood, and look similar to the untrained eye.

We are acutely aware that this book is a work in progress. We have been surprised by the wealth of literature, both published and unpublished, on lymphatic filariasis in the Americas. Much of the unpublished literature remains in the form of doctoral dissertations in university libraries throughout the Caribbean. We have been unable to obtain many of these valuable references, as well as some of the more obscure published articles; an asterisk identifies these reference citations at the end of each chapter. We would be most grateful for information that might lead us to copies of these articles, or for copies of the articles themselves. Our intent is to create a comprehensive reference library at the Collaborating Center that will be useful in the effort to rid the western hemisphere of lymphatic filariasis. We encourage your comments, criticism, and suggestions for future revisions of this document.

In keeping true to the character of the material, virtually no attempt has been made to change or correct misspellings or grammar from original texts.

We wish to thank our many colleagues at CDC and throughout the Americas who provided helpful comments on earlier drafts of this book. We especially thank Mary Bartlett for her encouragement, excellent editorial guidance, and tireless efforts to ensure that this document was published. We would also like to thank Kimberly Eastham for technical assistance and for graphics that further elucidated our work. We are grateful for the opportunity to have gotten acquainted with the work of many of the pioneers of lymphatic filariasis through the process of researching this book.

In particular, we are indebted to the work of two pioneers in the history and geography of lymphatic filariasis: Drs. Brian R. Laurence and Manabu Sasa.

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PART 1

Arrival and Spread of Lymphatic Filariasis in the Americas

LYMPHATIC FILARIASIS is a debilitating parasitic disease^{1,2} that, in the Americas, is caused by infection with *Wuchereria bancrofti*.¹ The disease is also known as bancroftian filariasis.

W. bancrofti has a life cycle that involves a mosquito vector and only one definitive host – humans.^{1,3} In the Americas, the vector is *Culex quinquefasciatus* (*Culex pipiens fatigans*).^{1,4-8} The parasite is of the nocturnal periodic form,⁹ meaning that microfilariae appear in the bloodstream primarily at night. This facilitates the process of transmission, since *Culex* is a night-biting mosquito.⁹

Life cycle of *Wuchereria bancrofti*

Infection is acquired by persons who have been bitten by infective mosquitoes.¹⁰⁻¹³ When the mosquito takes a blood meal, embryonic larvae (microfilariae) enter the mosquito via its proboscis and migrate to the stomach (Figure 1).¹⁴ Some larvae are expelled from the mosquito through the rectum, while others die from suffocation as the blood clots.¹⁴ Some exsheath to become shorter, broader, sluggish, and sausage-shaped, second-stage (L2) larvae.¹⁴ The L2 larvae pierce the stomach wall and migrate to the thorax via the anterior midgut.¹⁴ In the musculature of the thorax, the larvae develop to become the infective larval form, third-stage (L3) larvae.^{5,14-16} This maturation process takes about 10 days or longer.^{5,16-18}

After development, the infective L3 larvae migrate to the head and then to the proboscis of the mosquito.^{14,19-21} They are released onto the skin during subsequent blood meals.^{14,18,19,21} The L3 larvae enter the skin and make their way to the lymphatic system,²² where they develop into adult worms,^{3,22} a process that takes approximately 1 year.^{23,24} After maturity, the adult worms copulate and the females produce millions of embryonic microfilariae,^{3,22,23} which are released into the bloodstream.^{3,22}

Within 3 to 5 months of exposure to infective L3 larvae, the classical early signs and symptoms of lymphatic filariasis may appear in humans.^{23,25} Infected persons may have lymphadenitis followed by retrograde (i.e., centrifugal) lymphangitis,²⁵ characterized by pain along the course of the lymphatic vessel, with overlying cutaneous erythema.²⁶

Lymphangitis also causes transient and painful swellings of the genitalia, which may present as funiculitis, epididymitis, orchalgia, or edema of the scrotum.^{23,25} Lymphadenopathy of the epitrochlear, cervical, axillary, femoral, and inguinal lymph nodes may also occur.^{17,25,26}

The adult filarial worms are relatively long-living, surviving for 5 to 10 years or more in humans.²⁷ The presence of adult worms induces lymphatic vessel dilation (lymphangectasia), which leads to lymphatic dysfunction.²⁸ Dysfunction of the lymphatic vessels leads to hydrocele and predisposes to lymphedema.²⁸ Secondary bacterial infections are associated with progressive lymphedema of the leg, arm, breast, scrotum, and penis.²⁸ Elephantiasis usually appears only after several years.²⁹

When patients exhibit elephantiasis, microfilariae are often not detectable in the peripheral blood.²⁹ Other chronic conditions such as chyluria and chylous ascites occur when lymph vessels rupture into the urinary tract and the peritoneum, respectively.^{1,4,14,17,26,30-36}

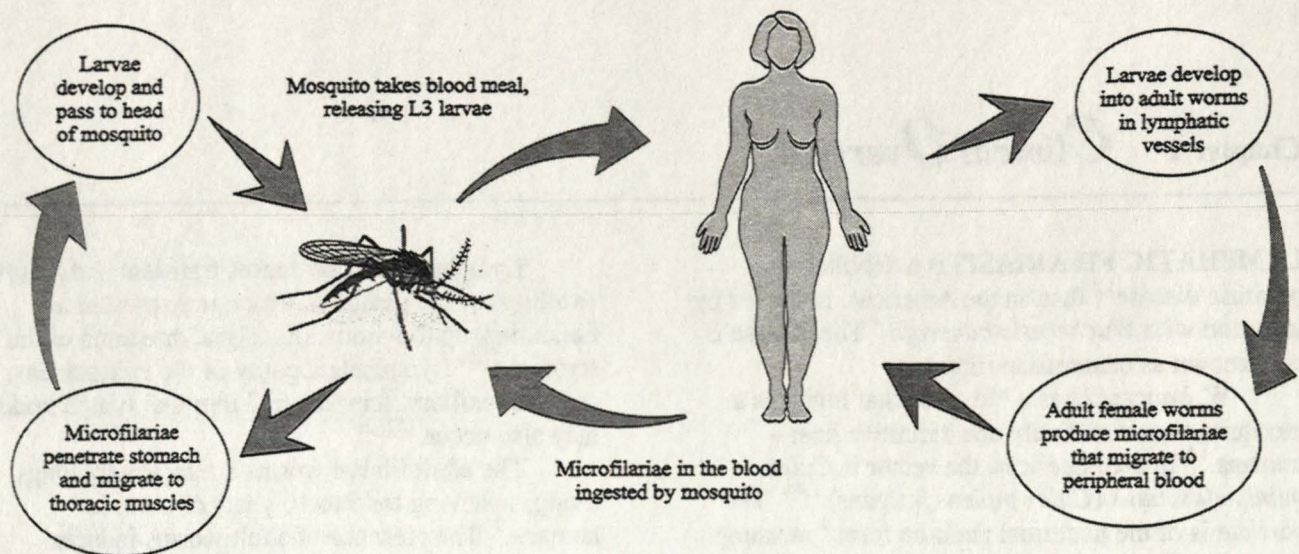


Figure 1. The life cycle of *Wuchereria bancrofti*

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Chapter 2 *Routes of Global Transmission*

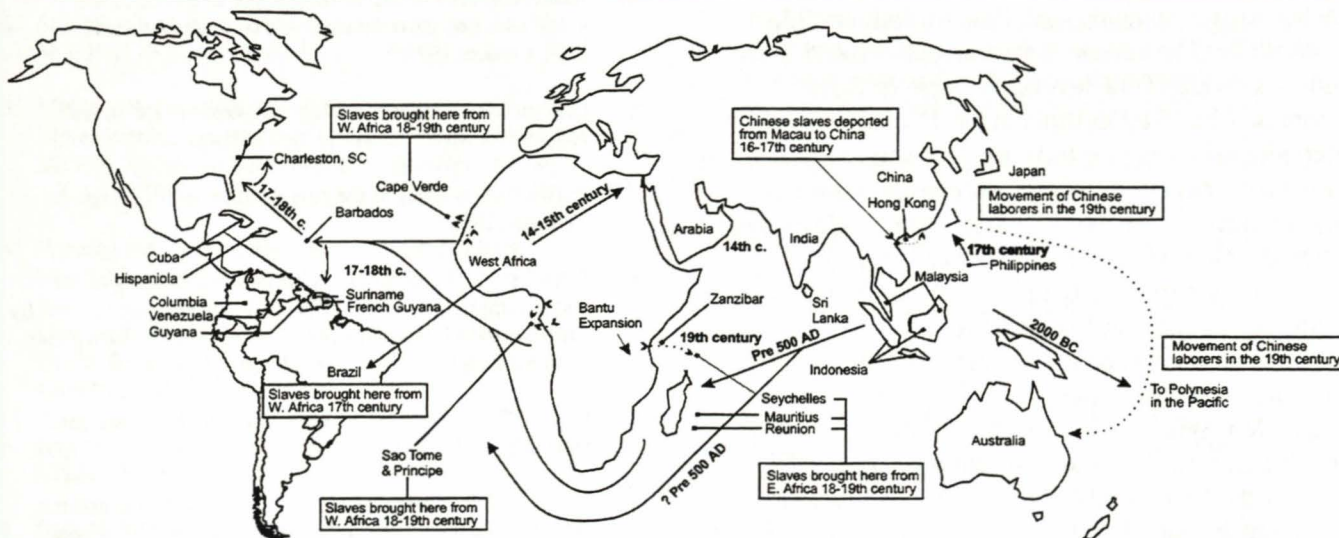


Figure 1. Map of possible routes of migration of *Wuchereria bancrofti* with its human host

Reprinted from Laurence BR. The global dispersal of bancroftian filariasis. *Parasitol Today* 1989; 5(8):260-4, with permission from Elsevier Science (map was modified).

Possible origins

IT IS BELIEVED that the parasite *Wuchereria bancrofti* originated in the rainforests of Southeast Asia, most likely in Indonesia, Malaysia, or Melanesia (Figure 1).¹⁻⁴ Residents of this area, the proto-Polynesians, are believed to have migrated by sea eastward to the Polynesian Islands in the second millennium B.C., carrying the parasite with them.^{3,4} *W. bancrofti* readily adapted to the day-biting mosquitoes *Aedes* (*Stegomyia*) *polynesiensis* and *Aedes variegatus* in the South Pacific Islands.^{3,4}

During the first 5 centuries A.D., people from the Indo-Malayan region are believed to have also migrated westward³ to the uninhabited island of Madagascar and the African mainland, rounding the Cape to West Africa.^{3,5} The parasite readily adapted to transmission by the *Anopheles* mosquito found in Africa.^{3,6} During the 14th and 15th centuries, several thousands of people made pilgrimages from the empires of Mali and Songhai in mainland Africa to Mecca, through Cairo.³ Lymphatic filariasis may have been spread by this route to the Middle East.³

One of the earliest records of a possible depiction of elephantiasis appears as a sculpture, the

“Queen of Punt” stele found in the funerary temple of Queen Hatsheput of Dier-al Bahari of Egypt (18th Dynasty, circa 1500 B.C.).⁷⁻⁹ It shows involvement of all limbs with absence of swelling in the feet, which is not characteristic of bancroftian filariasis, and has therefore provoked much controversy.¹⁰ Another opinion is that the disease depicted was hereditary.^{10,11}

Other early references to elephantiasis were documented by the Greeks and Romans;¹² by Indian scholars in such works as Samhita of Susruta (the period between the Buddha and Asoka, 600-250 B.C.);^{13,14} by Pliny in his Natural History (Book XXVI, 7-8), where he states that elephantiasis did not occur in Italy before the time of Pompey the Great (61 B.C.);¹⁰ by Celsus in the 1st century;¹⁰ by Paul Aegineta in the early 7th century;¹⁰ and by Arabian scholars in Avicenna, who together with their Latin translators are said to have confused the description of elephantiasis with leprosy.¹⁵

The earliest most descriptive record of elephantiasis as a probable clinical manifestation of lymphatic filariasis comes from the Tonga Group of

Islands in the South Pacific.^{2-4,16,17} Abel Janszoon Tasman wrote in his journal about meeting a man with Saint Thomas' arm, while his ship was anchored at Tongatapu and 'Eua, on January 22, 1643.^{2-4,16,17} The author had previously visited India and was aware of the legendary curse of Saint Thomas, who lived in India in the 1st century.^{4,17} Elephantiasis was said to be the punishment of the supposed murderers of Saint Thomas and their descendants.^{4,17} It is likely that the elephantiasis that Tasman saw was as a result of *Brugia malayi* infection; this form of lymphatic filariasis still exists in southwest India in Kerala State.^{17,18}

With the forced movement of thousands of Africans as slaves during the 17th and 18th centuries to the New World, it is likely that many infected persons carried the parasite to the Americas.^{3,4,6,19-34} Here in the New World, the parasite readily adapted from transmission by the *Anopheles* mosquitoes in Africa to the *Culex* mosquitoes found in the heavily populated and unsanitary towns in the American colonies.^{3,6,28,30,35}

Large numbers of slaves were first brought to Barbados^{6,28} and Brazil.³⁹ They were subsequently sent to other islands in the Caribbean,^{6,28} to the American colonies, especially South Carolina and Virginia,^{6,19,28,32,36,37} and to Guyana, Suriname, French Guiana, Brazil, and Venezuela.^{6,28} Barbados appears to have played an important role in the distribution of *W. bancrofti* throughout the West Indies.^{6,28}

The major factors contributing to the spread of lymphatic filariasis include mass migration of infected humans, whether forced or voluntary; high densities of efficient mosquito vectors; the adaptability of the parasite to different vectors (or the susceptibility of these vectors to the parasite); and climatic and living conditions that provided a hospitable environment for the breeding of the vector and transmission of the parasite.^{3,6,21,38}

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Chapter 3 *History of Lymphatic Filariasis in the Americas*

Earliest records

THE EARLIEST RECORD of elephantiasis in the Americas comes from the island of Barbados, where in 1726, Richard Towne, a local doctor, published his treatise on the diseases of the West Indies, especially Barbados.¹ The author stated that “Elephantiasis as a disease ... has a great affinity to the best accounts we have of the Leprae of Arabians.” In addition, William Hillary, a medical practitioner in Barbados from 1747-1758, included a section on elephantiasis in his published work “Observations on the changes of the air, and the concomitant epidemical diseases of the Island of Barbadoes.”² In this book, Hillary stated that elephantiasis occurred among slaves and settlers.

The Reverend Griffith Hughes, a rector of Saint Lucy’s Parish in Barbados, recorded in 1750, “The Elephantiasis is likewise a disease very common..... and hitherto incurable.”³ James Hendy, physician to His Majesty’s Naval Hospital at Barbados and Physician General to the Militia of the island,⁴ wrote about elephantiasis in his “Treatise on the glandular disease of Barbados, proving it to be seated in the lymphatic system,” which was published in 1784. The author stated that “The Glandular disorder was unnoticed in the Island till eighty years ago,” affecting both settlers and slaves alike, thereby dating the appearance of clinical manifestations of this disease in Barbados to about 1704.

In 1625, explorers came upon the island of Barbados^{5,6} and in 1627, it was first settled by about 80 people, including 10 slaves.⁵ Seventy years of extensive deforestation, establishment of settlements, and importation of large numbers of slaves onto the 166-square mile island led to overcrowding.⁵ During this period, elephantiasis probably became firmly entrenched on the island.⁷ Elephantiasis was so common in Barbados that in the English-speaking New World, it was known as “Barbados leg.”⁷⁻¹⁴

In Haiti around 1759, elephantiasis was referred to as “pied-botte.”¹⁵⁻²⁰ In Suriname, it was called “foto footoo,” which means “legs of the town,” a reflection of the urban rather than rural clustering of the disease.²¹⁻²³

Earliest detection of the parasite *Wuchereria bancrofti*

Microfilariae of *Wuchereria bancrofti* were first described by Jean-Nicholas Demarquay.²⁴ On July 24, 1862, an 18-year-old man, originally from Havana, Cuba, was seen in Paris by Demarquay.²⁴ During surgery to remove a tumor in the left scrotal sac, the physician aspirated a milky fluid.²⁴ On August 28 of the following year, the patient returned with a similar problem on the right side of the scrotum and a milky fluid was also aspirated.²⁴ The aspirate, a bluish-white fluid, was examined microscopically by a “Dr. Lemoine,” who observed “a little elongated and cylindrical creature” that “had extremely rapid movement of coiling and uncoiling in its different parts, especially its terminal extremity.”²⁴

On August 4, 1866, Otto Wücherer, at the Hospital da Santa Casa Misericordia in Salvador, Bahia, Brazil, found microfilariae in the urine of a female patient, who was believed to have urinary bilharziasis.²⁵ This discovery, along with that of Demarquay, helped establish the prominence of the Americas in the early investigations into lymphatic filariasis.⁹

Subsequently, other scientists made similar observations, including, J.F. da Silva Lima, who in 1868 found microfilariae in the blood-tinged urine of a patient in Bahia, Brazil.²⁶ On July 27, 1870, Jules Crévaux, a French Naval Surgeon, also found microfilariae in the urine (hematochyluria) of a young Creole in Guadeloupe.²⁷ These discoveries further expanded the known geographic distribution of *W. bancrofti* in the Americas.²⁸

In the Americas, the female adult worm was first identified on October 16, 1877 by A.J.P. da Silva Araujo.²⁹ Working in Rio de Janeiro, da Silva Araujo found microfilariae in the peripheral blood.²⁹ He named the genus of the parasite *Wuchereria*, in honor of Otto Wücherer.²⁹ On November 12, 1877, Felicio dos Santos and Julio de Moura, also in Brazil, extracted female adult worms from infected patients.³⁰ These findings suggested that adult worms, later known as *Wuchereria bancrofti*, were responsible for the microfilariae that had been seen in peripheral blood, hydrocele fluid, and urine.²⁹

Present Status of Transmission

- Countries with current active transmission
- Countries with transmission that apparently stopped after 1980
- Countries with transmission that apparently stopped before 1980
- Countries with possible but not well-documented transmission
- Countries with imported cases but no apparent transmission
- Countries with no documented cases or transmission



Figure 1. Lymphatic filariasis in the Americas (map not drawn to scale).

Table 1. Countries in the Americas categorized based on their status of transmission

Countries with current active transmission	Countries with transmission that apparently stopped after 1980	Countries with transmission that apparently stopped before 1980	Countries with possible but not well-documented transmission	Countries with imported cases but no apparent transmission	Countries with no documented cases or transmission
Brazil	Costa Rica	Antigua and Barbuda	Aruba and Bonaire	Canada*	Anguilla
The Dominican Republic	Trinidad & Tobago	Barbados	Belize	Chile	Argentina
Guyana	Suriname	Colombia	Curaçao		Bermuda*
Haïti		Cuba	Grenada		Bolivia
		Dominica	Honduras		British Virgin Islands
		French Guiana	Jamaica		Ecuador
		Guadeloupe	Mexico		El Salvador
		Martinique	Nicaragua		Greenland*
		Montserrat	Saba & Saint Eustatius (Statia)		Guatemala
		Panama	Saint Martin/Sint Maarten		Paraguay
		Puerto Rico			Peru
		Saint Kitts & Nevis			Saint Barthélemy
		Saint Lucia			Saint Pierre de Miquelon*
		Saint Vincent & the Grenadines			The Cayman Islands
		United States			The Islands of the Bahamas
		U.S. Virgin Islands			Turks & Caicos Islands
		Venezuela			Uruguay

*not included in map

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PART 2

Countries with Current Active Transmission

THE ORIGINAL INHABITANTS of Brazil, thought to have been Amerindians, are believed to have numbered about 2-5 million when the Portuguese arrived.^{1,2} In 1500, Admiral Pedro Alvares Cabral arrived on the Brazilian coast from Lisbon, Portugal on a trip ostensibly meant for India.¹⁻³ In 1531, King João III of Portugal sent the first settlers to Brazil and in 1534 he divided the coast into 12 hereditary captaincies, which were given to friends of the Crown.^{1,2}

In the mid-1600s, Amerindians worked in the sugarcane plantations.^{1,2} They were replaced by slaves in the latter part of the 17th century.^{1,2} In the 1690s, gold was discovered in Minas Gerais.^{1,2} During this gold rush, more slaves were brought in to work the mines.^{1,2} In 1807, two days after the army of Napoleon marched into Lisbon, the Portuguese Prince Regent set sail for Brazil.^{1,2} On September 7, 1822, Don Pedro, the son of the Prince Regent, having been left behind to rule the colony after the return of his father to Portugal, declared independence for Brazil from Portugal.¹⁻³ During the 19th century, coffee replaced sugar as the major export, and after emancipation was declared in 1888, thousands of European immigrants replaced slave labor.^{1,2} In 1899, Brazil became a republic.^{1,2}

The earliest reference to lymphatic filariasis in Brazil comes from the reports of Rego, who in 1843 wrote that elephantiasis was common in Rio de Janeiro, and cited Souts-Ammarel, who reportedly performed surgical repairs on both women and men who had elephantiasis of the genitalia.^{4,5}

On August 4, 1866, Otto Wücherer, at the Hospital da Santa Casa Misericórdia in Salvador, Bahia first observed microfilariae in urine from a female patient.⁶ He was looking for eggs of *Schistoma haematobium*, for the patient was believed to have urinary bilharziasis.⁶ On October 9 of the same year, Wücherer observed similar microfilariae in the urine of another female patient, who was a patient of Santos Pereira, and later, in the urine of a male patient of J.F. da Silva Lima.⁶

In September 1878, da Silva Lima informed T.S. Cobbold that da Silva Araujo had verified the presence of the larval stage of the parasite in mosquitoes in Bahia.⁷ During the same year, from

October 25 to November 25, J.L. Paterson and a "Dr. Hall Sr." conducted the first survey for microfilaremia in the Americas.^{8,9} They found that 26/309 (8.4%) persons sampled in Bahia were positive for microfilariae.^{8,9}

In 1904, Moncorvo stated that filariasis occurred less frequently in Brazil at that time than previously.^{5,10} In 1908 and 1909, Oliveira conducted studies in Salvador, Bahia, where he observed microfilaremia prevalences of 9.5% and 10.5%, respectively.^{5,11,12} The latter study was presented as a thesis to the medical faculty of Bahia.¹¹ From 1918 to 1919, Afrânio Amaral conducted a study on filariasis in Bahia, where he observed a microfilaremia prevalence of 9.0%.¹³

In 1922, B. Coqueiro conducted a study in Maceio, Alagoas and observed that 295/634 (46.5%) persons were microfilaria-positive.^{14,15} In 1928, G.V. de P. Magalhães conducted a study in São Salvador, Mont Serrat District of Bahia, and observed that 42/350 (12.0%) persons were microfilaria-positive.^{16,17} In 1933, H.P. Froes examined a patient who had ascites; microfilariae were found in the ascitic fluid.^{18,19}

In 1935, at the Yellow Fever Laboratory of the International Health Division of the Rockefeller Foundation in São Salvador, Mont Serrat District of Bahia, N.C. Davis conducted a study of laboratory employees, members of their immediate families, a few other relatives, and friends.¹⁷ Of 80 persons, three (3.8%) had microfilaremia.¹⁷

From November 1942 to December 1943, studies on random samples of the human and mosquito populations of Belém, Pará were conducted to investigate the prevalence and distribution of filariasis and the prevalence of infected mosquitoes.^{20,21} The investigators observed that 541/5,000 (10.8%) persons were positive for microfilariae and 64/5,000 (1.3%) persons had elephantiasis.²⁰ Of 1,014 *Culex fatigans* mosquitoes collected from homes in Belém, 118 (11.6%) were found to be infected, while 8 (1.8%) harbored infective larvae.^{20,21}

In 1944, Viana Martins caught and dissected 81 *C. fatigans* mosquitoes at the Military Hospital in Belém, where she found 19 (23.5%) to be infected

with larvae.²² In 1948, M.P. Deane conducted a study in Manaus, Amazonas, where 48/2,405 (2.0%) persons were positive for microfilariae.²³ In 1949, S.B. Pessôa conducted a study in various barrios of Salvador, Bahia.²⁴ In the bairro of Uruguai, 43/934 (4.6%) persons were found to be positive for microfilariae; 7/56 (12.5%) *Culex* mosquitoes were

infected and two (3.6%) harbored mature larvae.²⁴

Table 1 lists studies on lymphatic filariasis conducted in Brazil from the latter half of the 19th century to the middle of the 20th century. Table 2 shows mosquito surveys conducted in Belém, Pará and in the bairro of Uruguai in Salvador, Bahia.

Table 1. Studies on lymphatic filariasis in Brazil, 1878-1950

Year	Location	No. of persons examined	No. of persons microfilariae - positive	Microfilaremia prevalence (%)	Author and year of publication
1878	Bahia	309	26	8.4	J. L. Paterson, 1878 ⁸ ; J. L. Paterson, 1880 ⁹
1908	Salvador, Bahia	—	—	9.5	Oliveira, 1909 ¹¹ *; Sasa, 1976 ²²
1909	Salvador, Bahia	—	—	10.5	Oliveira, 1909 ¹¹ ; Padilla, 1915 ¹² ; Smith, 1917 ⁵
1918 - 1919	Bahia	—	—	9.0	A. Amaral, 1918-1919 ¹³
1922	Maceio, Alagoas	634	295	46.5	B. Coqueiro, 1922 ¹⁴ *; R. G. Rachou and L. M. Deane, 1952 ²⁵ ; Sasa, 1976 ²²
1928	São Salvador in Mont Serrat District, Bahia	350	42	12.0	G. V. de P. Magalhães, 1928 ¹⁶ *; N. C. Davis, 1935 ¹⁷ ; Sasa, 1976 ²²
1935	Yellow Fever Laboratory of the International Health Division of the Rockefeller Foundation in São Salvador, Mont Serrat District, Bahia	80	3	3.8	N. C. Davis, 1935 ¹⁷
1942 - 1943	Belém, Pará	5,000	541	10.8	O. R. Causey et al., 1945 ²⁰
1948	Manaus, Amazonas	2,405	48	2.0	M. P. Deane, 1949 ²³
1949	Salvador, Bahia	934	43	4.6	S. B. Pessôa and Z. A. Andrade, 1950 ²⁴

Table 2. Entomologic surveys in Brazil, 1942-1949

Year	Location	No. of mosquitoes dissected	No. of mosquitoes infected	Infection rate (%)	No. of mosquitoes infective	Infective rate (%)	Author and year of publication
1942 - 1943	Belém, Pará	1,014	118	11.6	8	1.8	O. R. Causey, 1945 ²⁰
1944	Military Hospital, Belém, Pará	81	19	23.5	—	—	Viana Martins, 1944, cited by Sasa, 1976 ²²
1949	The bairro of Uruguai, Salvador, Bahia	56	7	12.5	2	3.6	S. B. Pessôa, 1950 ²⁴

On August 28, 1951, the National Filariasis Control Program was formed as a project of the "Departamento Nacional de Doenças Endêmicas Rurais" of the Brazilian Ministry of Education and Health.^{25,26} Its purpose was to determine filariasis-endemic areas in Brazil through surveys for microfilaremia and clinical disease; determine the local vectors; control infection in human carriers of *Wuchereria bancrofti*; and conduct the basic research necessary for control.²² From 1951 to 1965, more than 551 blood surveys were carried out in 852 localities in 24 states; 16 states were found to be *W. bancrofti*-endemic (Table 3).^{22,27,28} Overall, 4,310 (0.5%) persons were found to be positive for microfilariae.^{22,27-29} As filariasis was distributed in scattered foci,^{30,31} the average (low)

microfilaremia prevalence for entire states obscure the fact that transmission was intense in some foci.³⁰

From 1951 to 1965, specimens of *C. fatigans* were collected from 12 states and dissected.^{22,27} Of 120,399 mosquitoes, 2,720 (2.3%) were infected with larvae and 258 (0.2%) harbored mature larvae (Table 4).^{22,27}

Between 1966 and 1996, more blood surveys were carried out in several states: Alagoas, Bahia, Paraíba, Pernambuco, and Santa Catarina.²⁸ Microfilariae were found predominantly in urban areas (Table 5).²⁸ Between 1991 and 1996, samples of *Culex quinquefasciatus* were collected in two states, dissected, and examined for *W. bancrofti* (Table 6).²⁸

Table 3. Microfilaremia surveys in Brazil, 1951-1965^{2,22}

State	Number of localities surveyed	Number of persons examined	Number of persons microfilaria-positive	Microfilaremia prevalence (%)
Acre	15	6,438	2	0.03
Alagoas	10	15,809	19	0.12
Amapá	24	44,686	190	0.43
Amazonas	104	43,583	40	0.09
Bahia	56	66,258	200	0.30
Ceará	2	16,504	0	0.00
Espírito Santo	42	31,114	0	0.00
Federal Distrito	1	1,274	2	0.16
Fernando de Noronha	1	809	0	0.00
Goiás	35	15,662	0	0.00
Guanabara	1	3	0	0.00
Maranhão	25	22,910	55	0.24
Mato Grosso	45	6,568	1	0.02
Pará	96	98,955	1,600	1.62
Paraíba	16	32,326	6	0.02
Paraná	28	13,389	0	0.00
Pernambuco	36	66,280	1,763	2.66
Piauí	14	9,805	0	0.00
Rio Grande do Norte	10	14,455	0	0.01

Table 3. (Cont.) Microfilaremia surveys in Brazil, 1951-1965^{22,27,28}

State	Number of localities surveyed	Number of persons examined	Number of persons microfilaria-positive	Microfilaremia prevalence (%)
Rio Grande do Sul	60	102,950	29	0.03
Rondônia	13	6,833	8	0.12
Roraima	5	1,534	0	0.00
Santa Catarina	178	167,310	391	0.23
Sergipe	35	26,003	3	0.01
Total	852	811,361	4,310	0.53

Table 4. Study on lymphatic filariasis in Brazil, 1977-1984²⁴

State	No. of entomologic surveys conducted	No. of <i>C. fatigans</i> dissected	No. of <i>C. fatigans</i> infected	Infection rate (%)	No. of <i>C. fatigans</i> that harbored mature larvae	Infectivity rate (%)
Alagoas	1	4,975	5	0.10	1	0.02
Amapá	5	2,971	11	0.37	0	0.00
Amazonas	8	18,613	155	0.83	5	0.03
Bahia	2	7,990	32	0.40	1	0.01
Fernando de Noronha	1	120	0	0.00	0	0.00
Maranhão	1	1,369	0	0.00	0	0.00
Pará	8	33,587	155	0.83	5	0.03
Paraíba	1	10,577	1	0.01	0	0.00
Pernambuco	1	14,158	1,036	7.32	136	0.96
Rio Grande do Norte	1	211	0	0.00	0	0.00
Rio Grande do Sul	1	14,832	7	0.05	0	0.00
Santa Catarina	21	10,996	133	1.21	19	0.17
Total	51	120,399	2,720	2.26	258	0.21

Table 5. Microfilaremia surveys in Brazil, 1966-1996

City and State	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
Castro Alves, Bahia	23,871	414	1.73	I. A. Sherlock and E. M. Serafim, 1967 ³²
Salvador, Bahia	2,036	127	6.24	I. A. Sherlock and E. M. Serafim, 1967 ³²
João Pessoa, Paraíba	8,836	8	0.10	J. S. Rached, H. Rodrigues, D. Santos, D. Monteiro, 1974 ³³
Castro Alves, Bahia	15,220	18	0.12	Ministério da Saúde, 1985 ³⁴
Castro Alves, Bahia	207	0	0.00	Ministério da Saúde, 1985 ³⁴
Belém, Pará	347,605	705	0.20	Ministério da Saúde, 1985 ³⁴
Recife, Pernambuco	295,392	4,293	1.45	Ministério da Saúde, 1985 ³⁴
Belém, Pará	266,884	296	0.11	Ministério da Saúde, 1985 ³⁴
Recife, Pernambuco	282,311	2,136	0.76	Ministério da Saúde, 1985 ³⁴
Olinda, Pernambuco	754	53	7.03	A. Rangel and G. Dreyer, 1988 ³⁵
Maceió, Alagoas	731	2	0.27	G. Dreyer et al., 1991 ³⁶
Maceió, Alagoas	10,450	69	0.66	G. Fontes et al., 1994 ³⁷
Recife, Pernambuco	466	63	13.52	M. A. V. Maciel et al., 1994 ³⁸
Olinda, Pernambuco	685	84	12.26	M. A. V. Maciel et al., 1994 ³⁸
Ponta Grossa, Santa Catarina	206	0	0.00	B. R. Schlemper Jr et al., 1994 ³⁹
Barra de Laguna, Santa Catarina	1,154	0	0.00	B. R. Schlemper Jr et al., 1994 ³⁹
Recife, Pernambuco	4,597	460	10.01	M. F. P. M. Albuquerque et al., 1995 ⁴⁰
Maceió, Alagoas	10,857	73	0.67	G. Fontes, 1996 ⁴¹
Recife, Pernambuco	10,581	683	6.46	A. Maciel et al., 1996 ⁴²

Table 6. Entomologic surveys in Brazil, 1991-1996

City and State	Number of <i>C. quinquefasciatus</i> dissected	Number of <i>C. quinquefasciatus</i> infected	Infection rate (%)	Author and year of publication
Recife, Pernambuco	7,856	48	0.61	Medeiros et al., 1992 ⁴³
Olinda, Pernambuco	8,003	105	1.31	Medeiros et al., 1992 ⁴³
Jaboatão, Pernambuco	8,010	97	1.21	Medeiros et al., 1992 ⁴³
Maceió, Alagoas	1,321	28	2.12	Fontes et al., 1994 ³⁷
Maceió, Alagoas	529	2	0.38	Fontes et al., 1994 ³⁷
Maceió, Alagoas	529	2	0.38	Fontes et al., 1994 ³⁷
Maceió, Alagoas	467	16	3.43	Calheiros, 1996 ⁴⁴
Maceió, Alagoas	1,426	21	1.47	Calheiros, 1996 ⁴⁴
Maceió, Alagoas	564	6	1.06	Calheiros, 1996 ⁴⁴
Maceió, Alagoas	1,403	4	0.29	Calheiros, 1996 ⁴⁴

Based on studies conducted in the 1990s, filariasis continues to exist in a few urban foci, along the coast of northeast Brazil.^{28,31} Presently, the National Health Foundation (FUNASA- Fundação Nacional de Saúde) of the Ministry of Health (Ministério da Saúde) considers three foci as having active transmission: Belém (Pará), Maceió (Alagoas), and Recife (Pernambuco).^{28,31}

In the Recife focus, *W. bancrofti* appears to be emerging: prevalence of infection is high (up to 15.0%) in several barrios,⁴⁵ and transmission has recently been shown in several areas of the metropolitan area that were not previously known to be *W. bancrofti*-endemic.⁴⁶

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THE DOMINICAN REPUBLIC shares the Island of Hispaniola with Haïti. It gained its independence from Haïti on February 27, 1844.¹ Hispaniola was the first place of settlement by Europeans (the Spanish) in the Americas.¹ Apparently, it was also the first area in the region to receive slaves, who were brought to work as sugarcane cutters in 1503.²⁻⁴ There are no available reports of either elephantiasis or chyluria from the time of the Taino Indians, who were the inhabitants of the island at the time of the arrival of Columbus in 1492 until the mid-20th century.^{1,5}

The earliest record (known to us) of lymphatic filariasis in the Dominican Republic is from 1947, when 7.0% of patients at the Hospital Padre Billini in Santo Domingo were found to be positive for microfilariae.⁵ Between October 2, 1953 and May 8, 1954, 896 patients at the Doctor William A. Morgan Hospital gave blood for a systematic search for microfilariae.^{6,7} The authors observed that 43/896 (4.8%) patients, many of who lived on the outskirts of Santo Domingo, had microfilaremia.^{6,7}

In 1956, of 895 asymptomatic adult patients examined by C.A. Aquino in Santo Domingo, 40 (4.5%) were observed to be positive for microfilariae.^{8,9} Within the same year, another study was conducted by Rosario, who showed that 39/157 (24.8%) persons, nonrandomly selected in San Cristóbal, had microfilaremia.^{9,10}

In 1969, 8.5% of the children and adolescents living in marginal communities in San Pedro de Macorís were found to be microfilaria-positive.¹¹ In 1970, 26/342 (7.6%) children and adolescents in these same communities, aged 3 to 14, were found to be positive for microfilariae.^{4,12} In 1976, a third and similar study was conducted; the authors observed that 6.6% of children and adolescents had microfilaremia.¹³

In September 1975, a Dominican immigrant to Russia, who had only lived in the Dominican Republic, was found to be microfilaria-positive on routine blood examination.^{14,15}

In 1977, 2.0% of parasitology students at the Universidad Autónoma de Santo Domingo were positive for microfilariae and all of these students were asymptomatic.^{9,10,16} Within the same year, 55/1,520 (3.6%) patients from various institutions

were found to be microfilaria-positive.^{10,17} In 1978, 3.1% persons from the capital city, Santo Domingo, were microfilaria-positive, with prevalence higher in men than in women.^{9,18} In 1979, of 233 persons who lived in Santo Domingo but originally came from various areas of the country, 8 were found to be microfilaria-positive.^{9,10,19}

In 1980, 149 persons from three communities, Caei, Pajarito, and Cabria, all located near Yaguata in San Cristóbal Province (south-central region), were examined for microfilariae.^{9,10,20} The study showed microfilaremia prevalences of 9.5%, 13.8%, and 26.3%, respectively.^{9,10}

In May 1981, 100 night blood samples were each collected from hospitals in Barahona and La Romana.¹⁰ All 100 samples from La Romana were negative, while 8 (8.0%) samples from Barahona were microfilaria-positive.^{4,10} Within the same year, daytime blood samples were collected from 417 persons originally from Batey Ocho, which is located in Central Barahona (southwest region).⁹ The authors found that 29/417 (7.0%) persons from Batey Ocho had microfilaremia.⁹

Between March 3, 1981 and June 11, 1985, A.L. Vincent and colleagues conducted a door-to-door survey in Santo Domingo and other places in south-central Dominican Republic.³ Of 2,876 persons examined, 110 (3.8%) were found to be positive for microfilariae.³ The authors also observed that the age ranged from 6 to 74 years, and that the proportion of positive samples was significantly higher in persons aged 10 to 19 (9.0%) than all the other groups combined.³

In addition, significant areas of transmission were found among some of the impoverished barrios of Santo Domingo.³ These areas were located west of the Ozama River and south of the Isabella River.³ The barrios included Barrio Villa Francisca, an established barrio located above the Ozama River channel, with a microfilaremia prevalence of 29/751 (3.9%), and Barrio La Ciénaga, a recently established (1960s) shantytown on the floodplain of the Ozama, with a microfilaremia prevalence of 51/522 (9.8%).^{3,4} Bancroftian filariasis was observed in three rural communities west of Santo Domingo, where many sugarcane workers who were associated with

the Ingenio Mill in Caei resided.³

Of 1,007 adult mosquitoes collected by A.L.Vincent and colleagues from 11 filariasis-affected houses in La Ciénaga, most were *Culex quinquefasciatus*.³ The authors reported that 144/543 (26.5%) female *C. quinquefasciatus* dissected were infected with 867 larvae; 8/867 (0.9%) larvae were in the infective stage.³ Thus, while more than a quarter of all mosquitoes were infected, only one of 543 (0.2%) was infective, with 8 third-stage larvae.³ The authors encountered a few typical cases of filarial disease, apparently acquired in the central valley (the Cibao) and on the north coast.³

In 1987, Vincent and colleagues reviewed five theses published at the Universidad Autónoma de Santo Domingo, which documented that 3,566 blood samples were obtained from residents of the following interior cities: Santiago (north-central), La Vega (north-central), Baní (south-central), San Juan (west-central), and Pedernales (southwest).^{3,4} No microfilariae were found.^{3,4}

In 1991, a meeting on filariasis was held in Santo Domingo.²¹ At this conference, other barrios of Santo Domingo were reported to be filariasis-endemic, with microfilaremia prevalences of 18.2% in María Auxiliadora; 1.4% in San Carlos; 33.3% in Los Guandules; 8.0% in Gualey; and 4.0% in Capotilo.⁴ Some reports at this meeting indicated the possible occurrence of filariasis in "sugarcane country" particularly, Vicente Noble in Batey Central (southwest region).⁴ Other investigators reported negative blood examination for microfilariae in the La Vega Real Valley, La Vega, Santiago, and San Francisco de Macorís (all in the north-central region),

despite the observation of elephantiasis in the region.⁴

Data presented at the same conference were collected from June 1988 to June 1989, in which 22/254 (8.7%) persons from the urban community of Cabral in Barahona Province (southwest) were observed to be positive for microfilariae, using both nucleopore and Knott's techniques on blood samples.^{22,23} Twenty infected female *Culex quinquefasciatus* collected from Cabral were dissected.²⁴ Of 98 larvae, 14 were in the infective stage.²⁴ Another presentation showed that 12/472 (2.5%) university students who had microfilaremia were also symptomatic.²⁵ These students were from Barahona and its neighboring communities of Tamayo, Cabral, and Vicente Noble.²⁵

A pilot study for a national seroprevalence survey of lymphatic filariasis was conducted between March 22 and April 10, 1998, in two cities: La Ciénaga (a barrio of Santo Domingo) and Barahona city.²⁶ The participants were preschool and primary schoolchildren between the ages of 4 and 19.²⁶ A total of 470 students were tested, 227 in La Ciénaga and 243 in Barahona city.²⁶ The authors observed that 29/227 (12.8%) children in La Ciénaga and 10/243 (4.1%) children in Barahona city were positive for circulating *Wuchereria bancrofti* antigen (in blood samples), using the ICT card test (a rapid immunochromatographic assay).²⁶

In summary, lymphatic filariasis is endemic in the Dominican Republic, with known foci located in sugarcane areas of the populous south, as well as in urban-coastal port cities of Santo Domingo, San Pedro de Macorís, San Cristóbal, and Barahona.^{3,4,10,27,28}

Table 1. Studies on lymphatic filariasis in The Dominican Republic, 1947-1998

Year	Area	No. of persons examined	No. of microfilaria-positive persons	Microfilaremia prevalence (%)	Author and year of publication
1947	Hospital Padre Billini, Santo Domingo	—	—	7.0	C. Inchaustegui, 1947 ⁵
1953 - 1954	Doctor William A. Morgan Hospital, Santo Domingo	896	43	4.8	E. A. Alvarez and A. G. Olivier, 1954 ⁷
1956	Santo Domingo	895	40	4.5	C. A. Aquino, 1956 ⁸ ; Vargas et al., 1981 ⁹ ; Vincent et al., 1981 ¹⁰
1956	San Cristóbal	157	39	24.8	Rosario, 1956 cited in Vargas, et al., 1981 ⁹
1969	San Pedro de Macorís	—	—	8.5	A. T. Ponce Ravelo, 1969 ¹¹

Table 1. (Cont.) Studies on lymphatic filariasis in The Dominican Republic, 1947-1998

Year	Area	No. of persons examined	No. of microfilaria-positive persons	Microfilaremia prevalence (%)	Author and year of publication
1970	San Pedro de Macorís	342	26	7.6	A. T. R. Ravelo and A. M. P. Pinedo, 1970 ¹²
1976	San Pedro de Macorís	—	—	6.6	A. Ponce Ravelo and A. Ponce Pinedo, 1976 ¹³
1977	Autonomous University, Santo Domingo	—	—	2.2	E. Vallejo de Saint-Amand, 1977 ¹⁶
1977	Patients from various institutions	1,520	55	3.6	M. Vargas et al., 1977 ¹⁷
1978	Santo Domingo	—	—	3.1	B. Santos et al., 1978 ¹⁸
1979	Santo Domingo	233	8	3.4	D. Roldan et al., 1979 ¹⁹
1980	Caei	149	—	9.5	C. Ascuasiati de Alfau et al., 1980 ²⁰
	Pajarito	(total)	—	13.8	
	Cabria	—	—	26.3	
1980	Barahona	100	8	8.0	A. L. Vincent et al., 1981 ¹⁰
1980	La Romana	100	0	0.0	A. L. Vincent et al., 1981 ¹⁰
1980	Batey Ocho	417	29	7.0	M. Vargas et al., 1981 ⁹
1981- 1985	Santo Domingo	2,876	110	3.8	A. L. Vincent et al., 1987 ³
1988- 1989	Cabral	254	22	8.7	M. Vargas et al., 1991 ²²
1991	María Auxiliadora	—	—	18.2	M. González De Pena, 1991 ⁴
	San Carlos	—	—	1.4	
	Los Guandules	—	—	33.3	
	Gualey	—	—	8.0	
	Capotilo	—	—	4.0	
1998	La Ciénaga (a barrio of Santo Domingo)	227	29	12.8	S. Y. Huang et al., 1998 ²⁶
1998	Barahona city	243	10	4.1	S. Y. Huang et al., 1998 ²⁶

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THE ORIGINAL INHABITANTS of Guyana are believed to have been primarily the Arawaks and the Caribs.¹ The name Guyana was derived from an Amerindian word meaning "land of many waters."² In 1604, the first attempt at settlement was made by the English, led by Captain Leigh, on the Oiapoque River (which is now in present-day French Guyana).^{3,4} This settlement proved unsuccessful.^{1,4} Between 1615 and 1624, the region was partially settled by the Dutch West Indian Company,^{1,3,4} which erected a fort and depot at Fort Kyk-over-al, on the lower Essequibo River in the County of Essequibo.^{1,3}

During the 17th century, the English established sugarcane and tobacco plantations on the west bank of the Suriname River,^{1,3,4} while the Dutch established posts on the coast, up-river, and in the hills, mostly as trading points with the original inhabitants.^{3,4} In 1663, Lord Willoughby of Barbados founded a settlement in Suriname, which was captured by the Dutch in 1667 and was later ceded to the Dutch at the "Treaty of Breda."³ In exchange, the English received Nieuw Amsterdam, which later became New York.³ Slaves were brought to work on the plantations, but their success was limited by the poor nature of the soil, and so settlers and slaves alike retreated to the coastal areas in the mid-18th century.^{3,4} Coffee and cotton remained the main crops up to the end of the 18th century, but by 1820, sugar had become the dominant crop.^{1,3,4}

The Dutch held the three colonies (Guyana, Suriname and French Guyana) until 1796, when the colonies were captured by a British fleet.^{1,3} In 1802, ownership of the territory was restored to the Dutch, but was retaken the following year by the British, who finally gained Guyana in 1814, when the three counties of Essequibo, Berbice, and Demerara were merged to form British Guiana.³ After emancipation in 1834, the plantation industry was sustained by indentured workers from the Far East, the Azores, the Canaries, and Madeira, many of whom settled in Guyana after their service.^{1,3,5} During the period 1846 to 1917, about 250,000 workers entered Guyana.¹

On May 26, 1966, independence was gained, and the country changed its name from British Guiana to Guyana.^{1,3,4} On February 23, 1970, Guyana became a Cooperative Republic within the Commonwealth,

adopting a new constitution.^{1,3,4} A major economic activity is mining, principally bauxite, but gold and diamonds are often sifted from the riverbeds.^{1,3} In addition, the sugar industry was nationalized and the economic base of the country has diversified to include the production of rice.³

The earliest reference to lymphatic filariasis appears in the reports of a "Dr. Hillis," who in about 1877, recognized *Wuchereria bancrofti* microfilariae in blood obtained from patients with clinical manifestations of lymphatic filariasis.⁶ Elephantiasis and chyluria were also seen and described in detail.⁶ On May 15, 1883, a "Dr. Grieve" conducted a study on the inmates of the Lunatic Asylum. He observed that 21/400 (5.3%) patients had elephantiasis; 10/252 (4.0%) in males, and 11/148 (7.4%) in females.⁷

In 1896, C.W. Daniels and J.H. Conyers conducted a study on patients at the Public Hospital in Georgetown, and observed that 52/348 (15.0%) were positive for microfilariae.^{7,8} The authors also reported that 69/752 (9.2%) persons had elephantiasis; 30/549 (5.5%) in males, and 39/203 (19.2%) in females.⁷ In 1897, A.T. Ozzard examined 100 patients at the Public Hospital in Berbice and found 28 patients to be positive for microfilariae.⁶ Within the same year, P. Manson and Ozzard conducted independent studies that showed that 9/61 (14.8%) and 24/83 (28.9%) persons, respectively, were microfilaria-positive.⁸

In 1908, G.C. Low conducted a survey in Guyana, where he observed that 25/150 (16.7%) persons were positive for microfilariae.⁹⁻¹¹ In 1908, K.S. Wise conducted studies on lymphatic filariasis in patients at the Public Hospital in Georgetown.¹² Here, he saw three isolated cases with signs and symptoms suggestive of lymphatic filariasis; only two patients were microfilaria-positive.¹² He also observed adult filariae and enlarged lymphatic vessels as incidental findings on autopsy of two patients: one died of lobar pneumonia, the other of heart disease (at age 67).¹² In an autopsy study of 250 persons, adult filariae were found as incidental findings in the pelvis of the kidneys in 10 (4.0%) persons.¹² In other studies conducted by Wise in Guyana, 21/95 (22.1%) patients had signs or symptoms suggestive of lymphatic filariasis; 66/493 (13.4%) persons were found to be

microfilaria-positive,¹² and 5.2% of males and 15.4% of females had elephantiasis.¹³

Records from the Georgetown Registration District indicate that in 1917, 1918, and 1919, 50, 71, and 82 persons, respectively, died from lymphatic filariasis.¹⁴ In 1918, F.G. Rose conducted studies in Georgetown and in the rural districts of the County of Demerara, where he observed that 846/3,383 (25.0%) and 200/1,001 (20.0%) children, respectively, had signs and symptoms suggestive of lymphatic filariasis.^{13,15} In November and December of the same year, the author dissected mosquitoes from various houses in Georgetown; 84/1,131 (7.4%) *Culex fatigans* mosquitoes were found to be infected with *W. bancrofti* larvae.^{13,15}

From January 1919 to March 1920, Rose conducted a small study on patients at the Public Hospital in Georgetown, and found that 36/60 (60.0%) males, who were from various parts of the country unknown to be affected by lymphatic filariasis, had microfilaremia.¹³ In a larger study at the same hospital and within the same period, 73/351 (20.8%) patients were found to be positive for microfilariae.¹³ In addition, 3.4% of males and 7.1% of females had elephantiasis, while 18.6% of males and 23.1% of females had signs or symptoms suggestive of lymphatic filariasis.¹³

In 1921, J. Anderson, M. Khalil, C.U. Lee, and R.T. Leiper conducted a series of studies throughout Guyana.^{14,16} The participants in these surveys included blacks, East Indians, Amerindians, Portuguese,

Chinese, Europeans, and multiracial people.^{14,16} Four surveys were conducted in Georgetown with the following groups: a randomly selected sample of people from the city; patients at the Public Hospital night clinic; persons from the Chinese community; and children from the Christchurch School.^{14,16} The investigators reported that 512/2,434 (21.0%) persons from a randomly selected sample in the city; 192/865 (22.2%) patients at the Public Hospital night clinic; 31/431 (7.2%) persons from the Chinese community; and 23/196 (11.7%) children from the Christchurch School were positive for microfilariae, respectively.^{14,16}

Outside Georgetown, the investigators reported that 102/785 (13.0%) persons in rural districts, 1/131 (0.8%) Amerindians, 14/100 (14.0%) patients from New Amsterdam Hospital, 33/442 (7.5%) persons from the Chinese communities of Charlestown, Lacytown, and Werkenrust, 157/515 (30.5%) persons from Albertown, 80/180 (44.4%) prisoners originally from Georgetown, and 36/244 (14.8%) prisoners from other country districts were positive for microfilariae.^{14,16}

In total, these investigators conducted 10 separate studies, in which 19.5% of persons examined were found to be positive for microfilariae (Table 1).^{14,16} In Albert Town, mosquitoes were collected from residential homes and dissected; 34/141 (24.1%) *C. fatigans* mosquitoes were found to be infected with *W. bancrofti* larvae.^{14,16}

Table 1. Results of 10 studies in Guyana by J. Anderson et al., 1919-1921^{14,16}

Population	No. of persons examined	No. of microfilaria-positive persons	Microfilaremia prevalence (%)
A randomly selected sample in Georgetown	2,434	512	21.0
Patients at the Public Hospital night clinic in Georgetown	865	192	22.2
Chinese community in Georgetown	431	31	7.2
Children at the Christchurch School, Georgetown	196	23	11.7
A randomly selected sample in rural districts	785	102	13.0
A group of Amerindians (Aboriginal Indians)	131	1	0.8
Patients from New Amsterdam Hospital	100	14	14.0
Chinese community in Georgetown and in country districts of Charlestown, Lacytown, and Werkenrust	442	33	7.5
Random sample in Albert Town	515	157	30.5
Prisoners from H. M. Penal settlements in Bartica and Georgetown	424	115	27.1
Total	5,892	1,149	19.5

In 1923, C.U. Lee reported the results of studies conducted in Georgetown and Albertain, where 65/585 (11.1%) and 95/223 (42.6%) persons, respectively, were microfilaria-positive.^{17,18} During 1924-1933, Cesare Romiti conducted a study at the Mackenzie Hospital located in the coastal region, and found that 31.5% persons were positive for microfilariae.^{19,20} From December 1927 to September 1928, A.W. Grace and Feiga B. Grace tested 1,000 patients from the Public Hospital in Georgetown.²¹ Of these, 231 (23.1%) were positive for microfilariae.^{21,22}

During 1934-1937, on the estates of Blairmont and Bath in Berbice, 14/642 (2.2%) persons were observed to be positive for microfilariae, while 21/3,848 (0.6%) had signs and symptoms suggestive of clinical lymphatic filariasis.^{23,24} In 1959, Subryan found that 9/561 (1.6%) persons on the Blairmont estate were positive for microfilariae.²⁴

In 1942 and 1943, and from 1947 to 1951, clinical and laboratory investigations of patients were conducted at the Filariasis Clinic and the Central Medical Laboratory, both of which were located in Georgetown.²⁵ During this period, 628 patients were seen; 54/278 (19.4%) patients were microfilaremic, while 467/628 (74.4%) reportedly had elephantiasis.²⁵

Between 1945 and 1948, George Giglioli showed that, in addition to *C. fatigans*, *Anopheles darlingi* was a vector of *W. bancrofti* in Guyana.^{23,26,27} From 1945 to 1946, numerous mosquitoes were collected during a series of field investigations.²³ In Lodge Village, a suburb of Georgetown, 303/12,438 (2.4%) *C. fatigans* mosquitoes, dissected immediately and also after 7-day and 12-day intervals, were found to be infected; 131 (1.1%) harbored mature larvae.²³ A total of 588 *A. darlingi* mosquitoes from Lodge Village were also collected and dissected.²³ Of these, 24 (4.1%) were infected; 2 (0.3%) harbored mature larvae.²³ On Plantation Lusignan, 58/5,921 (1.0%) *A. darlingi* mosquitoes dissected immediately after capture were infected; none harbored mature larvae.²³ *A. darlingi* was an important vector of malaria in Guyana.²⁴ Both malaria and the vector were eliminated from Guyana in 1960, after more than 15 years of a DDT (dichloro-diphenyl-trichloroethane) residual spraying campaign.²⁴

Between August 1946 and March 1947, after Giglioli had assessed microfilaremia prevalence in five study areas, DDT-residual spraying was conducted.²³ Between January and October 1947, on the Lusignan Plantation, mosquitoes were again collected and 65/1,288 (5.1%) *C. fatigans* mosquitoes,

dissected immediately and also after a 7-day interval, were found to be infected, while 10 (1.9%) dissected after a 7-day interval were found to harbor mature larvae.²³ From 1945 to 1946, Giglioli conducted a study near Georgetown, where he observed a microfilaremia rate of 21.4%, while the reported prevalence of elephantiasis was 5.3%.²⁷

Between 1946 and 1947, during the same major project that involved the mosquito collection, Giglioli and collaborators examined persons from Georgetown City and suburbs; New Amsterdam; the Village of Buxton and Friendship; and Plantation Lusignan,²³ located east of Georgetown.²⁸

In Georgetown City and its suburbs, 1,572/10,000 (15.7%) persons were microfilaria-positive, while 276/10,000 (2.8%) persons had elephantiasis.²³ In New Amsterdam, 126/549 (23.0%) persons were microfilaria-positive, while 31/549 (5.7%) persons had elephantiasis.²³ In the Village of Buxton and Friendship, which lies on the Demerara Coast 12 miles east of Georgetown, 257/1,206 (21.3%) persons were microfilaria-positive, while 44/1,206 (3.7%) persons had elephantiasis.²³ At the Plantation Lusignan, 33/846 (4.5%) persons were microfilaria-positive; 15/846 (1.8%) persons had elephantiasis.²³ Giglioli observed that microfilaremia prevalence had fallen since the late 1920s, and suggested that the sewer systems built between 1926-1930 may have been responsible.²³

In conclusion, Giglioli and colleagues conducted 5 separate studies from which 1,993/12,601 (15.8%) persons from urban areas, suburban areas, villages, and sugarcane estates were microfilaria-positive, while 366/12,601 (2.9%) persons from the same areas had elephantiasis (Table 2).²³ Giglioli maintained that pit latrines provided the ideal environment for the breeding of *C. fatigans*, and reported high prevalence of microfilaremia in areas with latrines – the Georgetown suburbs of Kitty, Lodge Village, and La Penitence; New Amsterdam; Buxton village; and Friendship.²³ These findings were confirmed in other studies.^{24,29-33}

Table 2. Studies of microfilaremia conducted by G. Giglioli, 1945-1948²³

Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Four wards in Georgetown: Kingston, Cummingsburg, Albouystown, and Wortmanville	6,329	876	13.8
Three suburbs of Georgetown: Kitty, Lodge Village, and La Penitence	3,671	696	19.0
New Amsterdam	549	126	23.0
The Village of Buxton and Friendship	1,206	257	21.3
Plantation Lusignan	846	38	4.6
Total	12,601	1,993	15.8

In 1946, 1950, 1953, and 1954, the Annual Reports of the Director of Medical Services indicated that filariasis was common in Guyana.³⁴⁻³⁷

In November 1947 and February 1948, M. Kenney and R. Hewitt conducted studies with Hetrazan (diethylcarbamazine - DEC) in Kwakwani, Georgetown, and New Amsterdam.³⁸ In Kwakwani, a small bauxite mining community located 140 miles inland from the mouth of the Berbice River, 57/400 (14.3%) persons were found to be positive for microfilariae, based on a review of patient records of the Berbice Company Limited Hospital.³⁸ All patients were treated with 0.5 mg/kg of Hetrazan, three times per day for 21 days.³⁸

A more extensive trial was conducted in both Georgetown and New Amsterdam, where participants were enrolled from the Forte Canje Mental Hospital in Berbice, the Public Hospital in New Amsterdam, and the Public Hospital in Georgetown.³⁸ The authors found that 255/1,241 (20.6%) persons were positive for microfilariae, while 49/1,241 (4.0%) persons had elephantiasis.³⁸ All patients were treated with 0.5 mg/kg of Hetrazan, three times per day for 21-28 days.³⁸ Eight months after treatment, 26/41 (63.5%) patients from Kwakwani were still microfilaremic.³⁹ In addition, 30/83 (36.2%) patients from Georgetown and New Amsterdam remained microfilaria-positive 14 months after treatment with Hetrazan.³⁹

According to published official national reports, beginning in 1951, there were 350 cases of filariasis; in 1952 (1,221); 1953 (1,166); 1954 (1,279); 1955 (1,495); 1956 (1,886); 1957 (1,053); and 1958 (1,053).⁴⁰ In a similar document for 1959, filariasis was reported, but recorded as a plus sign (+), rather than in figures.⁴⁰

From 1955 to 1961, W.E. Adams and colleagues conducted a series of studies along the

Guyana coastlands, extending eastward from Queenstown in Essequibo County to Nabacilis in Demerara County.⁴¹⁻⁴⁶ The authors reported that 17,698/223,166 (7.9%) persons were microfilaria-positive.⁴⁶

From 1956 to mid-1959, H.B. Edghill and colleagues conducted a study at Port Mourant, a sugarcane estate located on the Corentyne coast, 14 miles east of the Berbice River estuary and at Rose Hall, a large village that adjoined the western part of the main residential area of the estate.³⁰ Among all the estate village sections - Ankerville, Phoebe, and Tain-Clifton, and at Rose Hall - 399/3,539 (11.3%) persons were found to be positive for microfilariae, while 174/2,815 (6.2%) had lymphedema.³⁰ Two treatment groups were studied: 180 persons were treated with Banocide (diethylcarbamazine) 2 mg/kg body weight for 10 days while 312 persons were treated with the same daily dose of DEC for 21 days.³⁰ Both groups included some amicrofilaremic patients with clinical signs or symptoms of filariasis.³⁰ Thirty months after treatment, 197 of the original microfilaria-positive persons were re-examined; 61 (31.0%) still had microfilaremia.³⁰ After four courses of treatment, only one patient of 197 remained positive for microfilariae.³⁰

In 1960, Giglioli and H.M.S.G. Beadnell reviewed the status of lymphatic filariasis in Guyana, and observed that there was a great reduction, at least in recent years, in the microfilaremia prevalence in Georgetown and its environs.²⁴ Like Eli Chernin, who reviewed the status of lymphatic filariasis in the United States,⁴⁷ the authors attributed the fall in microfilaremia to the installation of a modern sewer plant between 1926 and 1930 and possibly the treatment with DEC.²⁴ Another school of thought believed that the short-lived DDT residual spraying

effort conducted in the late 1940s caused the reduction in microfilaremia prevalence.²⁴ However, the importance of the spraying effort in this regard, particularly in rural areas, was questioned.²⁴

Between April 1961 and 1963, G.J. Burton and colleagues collected and dissected female *Culex pipiens fatigans* mosquitoes from houses in the Buxton "intervention" area (Buxton, Friendship and Annadale; villages of Vigilance, Bladen Hall, Strath Spey; and Lusignan) where lymphatic filariasis infection had been reported, and in the "non-intervention" peripheral Georgetown villages (Kitty, Campbellville, Lodge, La Penitence) and the Golden Grove coastal area (Golden Grove, Cove and John, Victoria, Nabaclis, and Plantation Hope).^{31,32} Filariasis control in the Buxton area was achieved by a combination of anti-mosquito measures of once-monthly latrine spraying with gas-oil for 2 years and anti-parasitic measures of a single course of diethylcarbamazine over a 7-day period.^{31,32} At the beginning of the study, the microfilaremia prevalence in the Buxton "intervention" area was 17.2%, and at the end of the 2-year period it had reduced to 2.2%.³² For the duration of the study, 1,514/15,622 (9.7%) *C. pipiens fatigans* mosquitoes collected from the "non-intervention" areas of Georgetown peripheral villages and the Golden Grove coastal area were found to be infected, while 80/15,622 (0.5%) mosquitoes harbored infective larvae.^{31,32}

In 1962, 1 (0.1%) of 946 persons from the interior of the country were found to be positive for microfilariae.³³ In 1963, 1964, 1965, and 1966 the Filariasis Control Unit conducted surveys in the Demerara County area, and observed that 762/6,192 (12.3%), 1,985/18,465 (10.8%), 1,278/19,755 (6.5%), and 1,873/20,292 (9.2%) persons, respectively, were positive for microfilariae.^{33,48} The Filariasis Control Unit conducted further studies: 496/9,018 (5.5%) persons in four suburbs of Georgetown in 1967 and 177/3,696 (4.8%) persons in one suburb of Georgetown from January-September 1968 were positive for microfilariae.^{33,48} In October 1968, J.F.B. Edeson and F. Kuhlrow conducted impromptu investigations in New Amsterdam and Rose Hall.³³ They observed that 31/192 (16.2%) and 21/188 (11.2%) persons, respectively, were microfilaria-positive.^{33,48}

In 1981, a limited microfilariae survey in Georgetown was conducted by V. Stroom, during which 56/1,365 (4.1%) persons were found to be positive for microfilariae.⁴⁹ In 1984, the Greater

Georgetown area, including the city center and suburbs, was subdivided into nine areas for survey purposes.⁵⁰ Each area included between two to eight municipal units or wards from which the authors randomly selected a sample.⁵⁰ From the nine areas, 182/2,818 (6.5%) persons were found to be microfilaria-positive.⁵⁰ Survey area VIII – which included La Penitence, Alexander Village, and Ruimveldt (only the west and east portions of Ruimveldt) had the highest prevalence, with 67/547 (12.3%) persons positive for microfilariae.⁵⁰

Filariasis has been reported in immigrants from Guyana in other countries.⁵¹⁻⁵⁵ In 1914, in Montreal, Canada, a 13-year-old girl from Guyana had a painful swelling of lymph glands in the left groin for 2 years.⁵¹ She presented with fever and vomiting and was delirious.⁵¹ The symptoms exacerbated, resulting in death of unknown cause the following evening.⁵¹ A diagnosis of lymphatic filariasis was suggested and confirmed on autopsy, when microfilariae were demonstrated.⁵¹ In 1954, microfilariae were observed during a routine blood examination of a 34-year-old male immigrant in Toronto, Canada, who was treated for filariasis 2 months after emigrating from Guyana.⁵² In mid-October 1964, a 55-year-old female immigrant in Montreal noticed a small lump in her left breast, which on biopsy, contained fragments of worm identifiable as an adult male *W. bancrofti*.⁵³

In 1979, in New Nickerie, located on the western border of Suriname, the thick blood smear technique and the membrane filter technique were both used to detect microfilariae.⁵⁴ The thick blood smear technique detected 10/5,886 (0.2%) persons with microfilaremia, while the membrane filter technique detected 7/567 (1.2%) persons with microfilaremia.⁵⁴ A total of 17 infected persons were found to be microfilaria-positive by either method.⁵⁴ Of these, 16 (94.1%) were originally from Guyana, while one person (5.9%) was from Suriname, but had made frequent visits to Guyana.⁵⁴

From 1979 to 1981 during a massive survey in Suriname, specifically Paramaribo, 27/1,601 (1.7%) Guyanese (of 51,097 persons) were found to be microfilaria-positive on thick blood smears and 10/132 (7.6%) Guyanese (of 3,168 persons), were microfilaria-positive on the membrane filter technique.⁵⁵ In the District of Suriname, 18/1,200 (1.5%) Guyanese were microfilaria-positive on thick blood smears and 2/62 (3.2%) Guyanese were microfilaria-positive by the membrane filter technique.⁵⁵

On the sugarcane estate of Marienburg in Commewijne District, Suriname, 26/410 (6.3%) Guyanese laborers and their families were microfilaria-positive on thick blood smears and 12/410 (2.9%) of the same group were microfilaria-positive by the membrane filter technique.⁵⁵ Here, all the workers were originally from Guyana.⁵⁵ On the palm-oil estate of Victoria, in Brokopondo District, Suriname, 16/180 (8.9%) Guyanese were found to be microfilaria-positive using the membrane filter technique.⁵⁵

Investigations that began a few years before the turn of the 20th century confirm that Georgetown, for many years, was a primary focus of filariasis.^{6,7,12-14,16,21,23,28,31,56,57}

Based on studies conducted in the 1990s, filariasis still exists, primarily along the coast, south of Georgetown and close to the city of New Amsterdam.⁵⁸ In 1998, S.C. Rawlins and colleagues

conducted a survey of schoolchildren (aged 8-11) and adults, to determine the prevalence of circulating *W. bancrofti* antigen using the ICT card test (a rapid immunochromatographic assay) in five of the ten regions in Guyana.⁵⁹ Before the survey, a screening test was conducted at the Georgetown Hospital Filariasis Clinic.⁵⁹ The investigators reported that 37/119 (31.1%) asymptomatic patients and 22/407 (5.4%) patients with lymphedema were microfilaria-positive.⁵⁹ The results of the survey showed that 126/575 (21.9%) schoolchildren from three of the five regions and 39/133 (29.3%) adults from four of the five regions had circulating *W. bancrofti* antigen (Table 3).⁵⁹

Tables 4-7 summarize studies on lymphatic filariasis and mosquito investigations conducted in Georgetown and other communities in Guyana.

Table 3. *Wuchereria bancrofti* antigen prevalence in five of the ten regions of Guyana, S.C. Rawlins et al, 2000⁵⁹

Location (setting)	No. of persons examined	No. of persons positive for circulating <i>W. bancrofti</i> antigen	Prevalence of circulating <i>W. bancrofti</i> antigenemia (%)
Schoolchildren in Bartica, Suddie in the Cuyuni-Mazaruni region	40	1	2.5
Schoolchildren in Vreed-en-Hoop in the West Coast Demerara region	106	31	29.3
Schoolchildren in Georgetown in the Demerara-Mahaica region	196	65	33.2
Adults in Georgetown in the Demerara-Mahaica region	24	4	16.7
Schoolchildren in Fort Wellington in the Mahaica-Berbice region	60	1	1.7
Schoolchildren in New Amsterdam in the East Berbice-Corentyne region	110	22	20.0
Adults in New Amsterdam in the East Berbice-Corentyne region	109	35	32.1
Schoolchildren in Linden in the Upper Demerara-Berbice region	63	6	9.5

Table 4. Microfilaremia in the city of Georgetown, 1896-2000

Year	Location (setting)	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1896	Georgetown Public Hospital	348	52	15.0	C. W. Daniels and J. H. Conyers, 1896 ⁷
1908	Georgetown Public Hospital	493	66	13.4	K. S. Wise, 1909 ¹²

Table 4. (Cont.) Microfilaremia in the city of Georgetown, 1896-2000

Year	Location (setting)	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1919-1920	Georgetown Public Hospital	351	73	20.8	F. G. Rose, 1921 ¹³
1921	Inhabitants of Georgetown, randomly selected	2,434	512	21.0	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Night Clinic for Filariasis, Georgetown Public Hospital	865	192	22.2	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Chinese community in Georgetown	431	31	7.2	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Children between 4 and 16 years old from Christchurch School, Georgetown	196	23	11.7	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1923	General population in Georgetown	585	65	11.1	C. U. Lee, 1923 ¹⁷
1927-1928	Georgetown Public Hospital	1,000	231	23.1	A. W. Grace and F. B. Grace 1931 ²¹
1942-1943; 1947-1951	A filariasis clinic and Central Medical Laboratory, Georgetown	278	54	19.4	B. B. G. Nehaul, 1956 ²⁷
1945-1946	Near Georgetown	—	—	21.4	Giglioli, 1948 ²⁶
1946-1947	Georgetown wards and suburbs (Kingston, Cummingsburg, Albouystown, Wortmanville; Kitty, Lodge Village, La Penitence)	10,000	1,572	15.7	Giglioli, 1948 ²³
1948	Georgetown and New Amsterdam (before Hetrazan treatment)	1,241	255	20.6	M. Kenney and R. Hewitt, 1949 ³⁸
1949	Georgetown and New Amsterdam (14 months after Hetrazan treatment)	83	30	36.2	R. Hewitt, M. Kenney, A. Chan and H. Mohamed, 1950 ³⁹
1955-1961	Northeastern coastlands, extending from Queenstown in Essequibo County through Georgetown, Nabaclis, which are both in Demerara County	223,166	17,698	7.9	W. E. Adams, 1961 ⁴⁶
1967	Four suburbs of Georgetown	9,018	496	5.5	[Study conducted by the Filariasis Control Unit], J. F. B. Edeson and F. Kuhlow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1967	A suburb of Georgetown	3,696	177	4.8	[Study conducted by the Filariasis Control Unit], J. F. B. Edeson and F. Kuhlow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1981	Georgetown	1,365	56	4.1	V. Stroom, 1981 ⁴⁹ ; M. B. Nathan and V. Stroom, 1990 ⁵⁰
1984	The city center and suburbs of Georgetown	2,818	182	6.5	M. B. Nathan and V. Stroom, 1990 ⁵⁰
1998	Georgetown Hospital Filariasis Clinic (asymptomatic patients)	119	37	31.1	S. C. Rawlins et al., 2000 ⁵⁹
1998	Georgetown Hospital Filariasis Clinic (patients with lymphedema)	407	22	5.4	S. C. Rawlins et al., 2000 ⁵⁹

Table 5. Microfilaremia in Guyana, outside the city of Georgetown, 1897-1968

Year	Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1897	Public Hospital, Berbice	100	28	28.0	A. T. Ozzardi, 1897 ⁶
1897	Location uncertain	83	24	28.9	A. T. Ozzardi, 1897 cited from P. Manson, 1897 ⁸
1897	Demerara littoral	61	9	14.8	P. Manson, 1897 ⁸
1921	Country districts selected at random	785	102	13.0	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Aboriginal Indians	131	1	0.8	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Albert Town (a municipal district of Georgetown)	515	157	30.5	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Prisoners from H. M. Penal Settlement at Bartica and Georgetown	424	115	27.1	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	New Amsterdam Hospital	100	14	14.0	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Chinese community in Country districts (Charlestown, Lacytown and Werkenrust)	11	2	18.2	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1923	General population of Albertytown	223	95	42.6	C. U. Lee, 1923 ¹⁷
1934-1937	Estates of Blairmont and Bath, on the Berbice estuary in Berbice Country	642	14	2.2	Giglioli, 1948 ²³
1935	Coastal districts of Guyana	—	—	31.5	C. Romiti, 1935 ¹⁹ ; Romiti, 1936 ²⁰
1947	Persons in New Amsterdam	549	126	23.0	Giglioli, 1948 ²³
1947	Village of Buxton and Friendship	1,206	257	21.3	Giglioli, 1948 ²³
1947	Plantation Lusignan	846	38	4.5	Giglioli, 1948 ²³
1947	Kwakwani	400 [approx.]	57	14.3	M. Kenny and R. Hewitt, 1949 ³⁸
1949	Kwakwani (8 months after Hetrazan treatment)	41	26	63.5	R. Hewitt, M. Kenney, A. Chan and H. Mohamed, 1950 ³⁹
1956-1959	Port Mourant and its environs on the Corentyne Coast	3,539	399	11.3	H. B. Edghill, 1961 ³⁰
1956-1959	Port Mourant and its environs on the Corentyne Coast (30 months after Banocide treatment [DEC]; many positives lost to follow-up)	197	61	31.0	H. B. Edghill, 1961 ³⁰
1959	Estates of Blairmont, on the Berbice estuary in Berbice Country	561	9	1.6	Study conducted by Subryan] and cited by Giglioli, 1948 ²³
1962	In the Interior, actual location unknown	946	1	0.1	Cited by J. F. B. Edeson and F. Kuhlow, 1969 ³³
1963	Demerara County	6,192	762	12.3	Cited by J. F. B. Edeson and F. Kuhlow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸

Table 5. (Cont.) Microfilaremia in Guyana, outside the city of Georgetown, 1897-1968

Year	Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1964	Demerara County	18,465	1,985	10.8	Cited by J. F. B. Edeson and F. Kuhlrow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1965	Demerara County	19,755	1,278	6.5	Cited by J. F. B. Edeson and F. Kuhlrow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1966	Demerara County	20,292	1,873	9.2	Cited by J. F. B. Edeson and F. Kuhlrow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1968	New Amsterdam	192	31	16.2	J. F. B. Edeson and F. Kuhlrow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸
1968	Rose Hall	188	21	11.2	J. F. B. Edeson and F. Kuhlrow, 1969 ³³ ; R. S. Muller and E. S. Tiksingh, 1975 ⁴⁸

Table 6. Prevalence of "elephantiasis" (lymphedema of the leg) in studies conducted in Guyana, 1883-1959

Year	Location	No. of persons examined	No. of persons with "elephantiasis"	"Elephantiasis" prevalence (%)	Author and year of publication
1883	Berbice Lunatic Asylum, near New Amsterdam	400	21	5.3	[Study conducted by Dr. Grieve] cited by C. W. Daniels and J. H. Conyers, 1896 ⁷
1896	Georgetown Public Hospital	752	69	9.2	C. W. Daniels and J. H. Conyers, 1896 ⁷
1920	Georgetown Public Hospital	371	20	5.4	Rose, 1920 ¹⁵ ; Rose, 1921 ¹³
1921	Night Clinic for Filariasis, Georgetown Public Hospital	709	434	61.2	J. Anderson, M. Khalil, C. U. Lee, R. T. Leiper and G. M. Vevers, 1924 ¹⁴
1921	Children between age 4-16 from Christchurch School, Georgetown	196	5	2.6	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1921	Albert Town	515	14	2.7	Anderson et al., 1924 ¹⁴
1942-1943; 1947-1951	A Filariasis Clinic and Central Medical Laboratory, Georgetown	628	467	74.4	B. B. G. Nehaul, 1956 ²⁷
1945-1946	Near Georgetown	—	—	5.3	Giglioli, 1948 ²⁶
1946-1947	Georgetown wards and suburbs (Kingston, Cummingsburg, Albouystown, Wortmanville; Kitty, Lodge Village, La Penitence)	10,000	276	2.8	Giglioli, 1948 ²³
1947	A convenience sample of persons in New Amsterdam	549	31	5.7	Giglioli, 1948 ²³
1947	Village of Buxton and Friendship	1,206	44	3.7	Giglioli, 1948 ²³

Table 6. (Cont.) Prevalence of “elephantiasis” (lymphedema of the leg) in studies conducted in Guyana, 1883-1959

Year	Location	No. of persons examined	No. of persons with “elephantiasis”	“Elephantiasis” prevalence (%)	Author and year of publication
1947	Plantation Lusignan	846	15	1.8	Giglioli, 1948 ²³
1948	Georgetown and New Amsterdam	1,241	49	4.0	M. Kenney and R. Hewitt, 1949 ³⁸
1956-1959	Port Mourant and its environs on the Corentyne Coast	2, 815	174	6.2	H. B. Edghill, 1961 ³⁰

Table 7. *Wuchereria bancrofti* infection in *Culex fatigans* and *Anopheles darlingi* in Guyana, 1918-1963

Year	Town	No. of mosquitoes dissected	No. of mosquitoes infected	Infection rate of mosquitoes (%)	No. of mosquitoes infective	Infectivity rate of mosquitoes (%)	Author and year of publication
1918	Georgetown	1,131	84	7.4	—	—	F. G. Rose, 1921 ¹³
1921	Albert Town	141	34	24.1	—	—	Anderson et al., 1923 ¹⁶ ; Anderson et al., 1924 ¹⁴
1945-1946	Lodge Village, a suburb of Georgetown	12,438 [both immediate and delayed dissection]	303	2.4	131	1.1	G. Giglioli, 1948 ²³
1945-1946	Lodge Village, a suburb of Georgetown	588 [A. darlingi]	24	4.1	2	0.3	G. Giglioli, 1948 ²³
1945-1946	Plantation Lusignan	5,921 [A. darlingi]	58	1.0	—	—	G. Giglioli, 1948 ²³
1947	Lodge Village, a suburb of Georgetown (After DDT residual spraying)	1,288 [both immediate and delayed dissection]	65	5.1	10	1.9	G. Giglioli, 1948 ²³
1961-1963	From the non-intervention areas of Georgetown, peripheral villages and Golden Grove	15,622	1,514	9.7	80	0.5	G. J. Burton, 1967 ³²

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HAÏTI SHARES THE ISLAND of Hispaniola with the Dominican Republic.¹ The original inhabitants are believed to have been the Siboneys, who migrated from the North American continent around 450 A.D.¹ The next group of people to colonize the island were the Tainos, who arrived circa 900 A.D. from the Amazon valley.¹ The Tainos called the island “Ayiti” which meant “land of mountains.”¹ The third group were the Caribs, who migrated in the late 15th century.¹

Christopher Columbus arrived on the island on December 5, 1492, at the point currently called Mole Saint Nicolas, located on the western tip of the Northern Peninsula of Haïti.¹ The ship *Santa Maria* was wrecked near the coast of present-day Cap-Haïtien, where apparently the first European settlement – “La Navidad” – was established, but this settlement was later destroyed.¹ Although officially under Spanish rule, no other settlements were established for about 75 years, until the English arrived.¹

By 1630, the French and British had established settlements on the island of Tortuga and, by 1644 had also established a settlement near Port-de-Paix.¹ Control of the island alternated among the Spanish, the French, and the English on numerous occasions, which eventually led the Crown of Spain, in 1687, to cede the western third of the island to France through the “Treaty of Ryswick.”¹ This portion of the island was renamed Saint Domingue and later became “La République d’Haïti.”¹ Saint Domingue flourished economically under French rule, where slaves were brought to cultivate land for sugarcane, coffee, cotton, indigo, cocoa, and ebony, until emancipation on August 23, 1793.¹ During this period, Toussaint Louverture fought the British under the French flag.¹ In 1801, he gained control of the eastern part of the

island and became the governor of the entire island, Hispaniola.¹ In 1802, he relinquished control of the island to the French, who were led by Leclerc Bonaparte (brother of Napoleon Bonaparte).¹ On November 18, 1803, an ally of Louverture, Jean-Jacques Dessalines, defeated Leclerc’s successor, Rochambeau, at the famed “Battle of Vertieres,” outside of what is today Cap-Haïtien.¹ Dessalines proclaimed the independence of the colony at Gonaïves on January 1, 1804.¹

Elephantiasis of the leg has been known in Haïti since 1759, where it was referred to as “*pied-botte*.”²⁻⁶ In 1786, Gauche saw six cases of elephantiasis and in 1788, he again observed similar cases.^{7,8} Reports from 1894, 1896, 1904, 1909, and 1910 also show filariasis did occur in Haïti.⁹⁻¹³ In 1894, Audain stated that Haïti “has probably been infested for many years, perhaps in no higher ratio than Cuba.”^{9,14} Later reports indicate that “Filariasis is quite common as evidenced by the many cases of elephantiasis seen among the natives and the finding of *Microfilariae bancrofti* in the blood.”¹⁵

Between November 30, 1927 and January 30, 1928, at the Emigration Bureau of the Service d’Hygiène in Port-de-Paix, 11,000 laborers bound for Cuban sugarcane fields were examined by doctors.¹⁶ Blood samples were taken from which blood smears were made and checked for malaria parasites as well as microfilariae of *Wuchereria bancrofti*.¹⁶ Of the 11,000 laborers, 187 (1.7%) from the 10 sanitary districts of Haïti were found to be microfilaria-positive (Table 1).¹⁶

During this period Wilson also examined 2,007 children under age 14 from each of the 10 sanitary districts of Haïti and took blood samples for smears.¹⁶ Of these children, 12 (0.6%) had microfilaremia (Table 2).¹⁶

Table 1. Microfilaremia prevalence in laborers examined at Port-de-Paix, 1927-1928¹⁶

Sanitary district	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Cap-Haïtien	2,303	115	5.0
Port-de-Paix	5,927	41	0.7
Gonaïves	1,977	6	0.3
Port-au-Prince	251	12	4.8
Petit-Goave	106	6	5.7
Saint Marc	117	5	4.3

Table 1. (Cont.) Microfilaremia prevalence in laborers examined at Port-de-Paix, 1927-1928¹⁶

Sanitary district	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Jacmel	224	2	0.9
Aux Cayes (Les Cayes)	39	0	0.0
Hinche	19	0	0.0
Jeremie	37	0	0.0
Total	11,000	187	1.7

Table 2. Microfilaremia prevalence in children from each of Sanitary Districts of Haïti, 1928¹⁶

Sanitary district	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Cap-Haïtien	173	3	1.7
Port-de-Paix	424	4	0.9
Gonaïves	149	0	0.0
Port-au-Prince	179	3	1.7
Petit-Goave	103	0	0.0
Saint Marc	130	0	0.0
Jacmel	100	0	0.0
Aux Cayes (Les Cayes)	100	0	0.0
Hinche	549	0	0.0
Jeremie	100	2	2.0
Total	2,007	12	0.6

According to published official national reports, beginning in 1951, there were 5 cases of filariasis; in 1952 (56); 1953 (76); and 1954 (123).¹⁷ In similar reports from 1955 to 1959, filariasis was found, but recorded as plus signs (+) rather than in figures.¹⁷ The data were collected from both inpatient and outpatient records of hospitals and dispensaries.¹⁷

In 1963, Cavalier, Gentilini, and coworkers reported that lymphatic filariasis probably extended throughout the country, with two important foci.² One was around the city of Les Cayes in the south; the other, in the north, formed a triangle, with Port-de-Paix, Cap-Haïtien, and Gonaïves as its apices and Pilate as its center.² The same year, preliminary investigations were conducted at the laboratory of the Service National d'Éradication de la Malaria en Haïti (SNEM).² Of 1,000 daytime thick-film blood smears made for a malaria survey, 8 (0.8%) blood smears were found to be positive for microfilariae.²

In December 1969, a 44-year-old Haïtian-born man presented at the Boston City Hospital, Boston, with bilateral hydrocele and epididymo-orchitis.¹⁸ The left hydrocele and epididymis were surgically removed.¹⁸ On histologic examination, the epididymis exhibited adult worms; blood smears were positive for microfilariae, and the patient was treated with a course

of diethylcarbarmazine (DEC) for 3 weeks.¹⁸

In April of 1976, 90 patients from Cap-Haïtien, Limbé, and Gonaïves were selected, examined, and screened at the outpatient clinic of L'Hôpital Bon Samaritain, located in Limbé, Département du Norde.¹⁹ Of these, 6 (6.7%) patients were found to be microfilaria-positive in diurnal blood smears.¹⁹ Quantitative studies of four of the patients indicated that all infections showed a definite and marked nocturnal periodicity.¹⁹ This confirmed earlier reports of *W. bancrofti* microfilariae being present in diurnal blood smears from Haïti and suggested that the true infection prevalence and density were probably much greater than reported in the two earlier studies.^{2, 19}

In February 1977, nocturnal blood smears were made from blood collected from persons in a random sample of the population in Limbé.³ C. Raccurt and W. Hodges observed that 203/1,135 (17.9%) persons were microfilaria-positive.³ Of the infected persons, three were 4-year-olds; 22% of the children aged 10-14 years had microfilaremia.³ Males were more often infected than females; persons who lived close to the canal were more likely to be infected.³

This study showed that there was extensive transmission of filariasis in Limbé, affecting children at an early age.³ Clinical manifestations included

lymphadenitis of the inguinal nodes in almost all microfilarial carriers, engorged lymph vessels, edema of the extremities, and hydrocele.³ However, no elephantiasis or chyluria was seen.³ W. Hodges, who directed L'Hôpital Bon Samaritain in Limbé, reported that cases of elephantiasis were seen infrequently, but that hydrocele was relatively common in the outpatient clinic in Limbé.¹⁹

From February to November 1977, C. Raccurt, W. Hodges, and J. Bony conducted another filariasis survey of patients hospitalized at the L'Hôpital Bon Samaritain, Limbé.²⁰ They observed that 243/1,450 (16.8%) patients were positive for microfilariae.²⁰ Within the same year, a case of filariasis was diagnosed and reported when the blood smear from a 30-year-old woman from Plaisance was examined at SNEM.⁴ Two men over the age of 60, from Croix-des-Missions, which is located some distance north of Port-au-Prince, had microfilariae observed in their blood during a routine blood examination at the Hôpital de l'Université d'Etat in Port-au-Prince.⁴

During the period 1979-1981, 12 workers from Haïti residing in Victoria, Brokopondo District, in Suriname were examined for microfilariae, and two (16.7%) were positive by the membrane filter technique.²¹

In 1980-1981, Raccurt and colleagues conducted another survey in the southern community of Léogane and its environs.²² The area has been known as a major center for sugarcane cultivation.²² Participants were randomly selected from households located in four areas: Léogane city proper; the southeastern suburb of Dampus, located near sugarcane fields and a major cane-crushing factory; Chassagne, a southwestern suburb, where numerous distilleries are located; and Ca-Ira, a northwestern suburb, located on the coast, adjacent to a large

mangrove swamp where *Culicoides furens*, a vector of *Mansonella ozzardi* breeds.²² The participants were examined at the Saint Croix Hospital in Léogane, where the authors observed that 140/421 (33.3%) persons had microfilaremia.²² This study showed that filariasis was not restricted to northern Haïti or to urban settings, and suggested an association with areas of sugarcane farming and refineries (Table 3).²²

Between January 1981 and March 1982, a filariasis survey was conducted in six counties located in central and south Florida.⁶ The researchers observed that of 668 recent Haïtian immigrants, 45 (6.7%) were microfilaria-positive.⁶ Some showed varying degrees of inguinal lymphadenopathy, but no definitive signs of filarial disease were seen.⁶ Most of the microfilaria-positive participants came from five coastal urban areas in Haïti: Port-au-Prince, Duvalierville, Gonaïves, Lèogane, and Cap-Haïtien.⁶

During this survey, a Haïtian with an enlarging scrotal mass was referred to the investigators from a hospital in Naples, Florida.⁶ Degenerating female adult worms were observed in histologic sections of the epididymis.⁶ In 1985, another Haïtian immigrant presented with scrotal swelling of 4 years' duration in Charleston, South Carolina.²³ Excision revealed microfilariae of *W. bancrofti*.²³

In 1986, Raccurt published a review of studies conducted from 1977 to 1984 on the extent and distribution of filariasis in urban communities and their suburban environs, as well as in rural communities.²⁴ He observed that microfilaremia had been reported from nine urban and suburban areas and two groups of rural areas.²⁴ Of 6,779 persons from these areas, 667 (9.8%) had microfilaremia (Table 4).²⁴

Table 3. Study on lymphatic filariasis in Leogane and its environs, 1980-1981²²

Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Léogane city proper	200	54	27.0
Dampus	82	32	39.0
Chassagne	61	27	44.3
Ca-Ira	78	28	35.9
Total	421	140	33.3

Table 4. Study on lymphatic filariasis in Haïti, 1977-1984²⁴

Community	Number of persons examined	Number of persons microfilaria-positive	Microfilaremia prevalence (%)
Duvivier	532	162	30.5
Lèogane faubourg [suburb]	221	87	39.4
Lèogane [town]	200	53	26.5
Arcahaie	525	104	19.8
Limbé	1,450	243	16.8
Croix-des-Bouquets	319	9	2.8
Petite-Rivière-de-l'Artibonite	673	1	0.2
Miragoane	847	0	0.0
Aquin	170	0	0.0
Bayeux [North]	1,165	8	0.7
7 communes [South]	677	0	0.0
Total	6,779	667	9.8

In summary, lymphatic filariasis is assumed to occur throughout the country,^{4,24,26} with important foci in coastal areas of the north.^{3,4,24,25} The prevalence appears to be increasing^{24,25} in the south^{2,4,5,19,27} and along the Gulf of La Gonave.²⁴ Haïti has some

hyperendemic foci, other areas with low rates of infection,^{23,24,26,29,30} and yet other areas where a few sporadic cases occur.²⁴ A national elimination program is presently being developed.³⁰

Table 5. Studies on lymphatic filariasis in Haïti, 1927-1984

Year	Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1927 - 1928	Laborers at Port-de-Paix	11,000	187	1.7	P. W. Wilson, 1929 ¹⁶
1927 - 1928	Children from the 10 sanitary districts of Haïti	2,007	12	0.6	P. W. Wilson, 1929 ¹⁶
1963	Laboratory of the Service National d'Éradication de la Malaria en Haïti (SNEM) (from daytime blood smears)	1,000	8	0.8	M. Gentilini et al., 1964 ²
1976	Adult from Cap-Haïtien, Limbé and Gonaïves at the outpatient clinic of L'Hôpital Bon Samaritain, Limbé (from daytime blood smears)	90	6	6.7	M. J. Miller et al., 1976 ¹⁹
1977	General population, Limbé	1,135	203	17.9	C. P. Raccurt, 1977 ²⁰
1977	Patients at L'Hôpital Bon Samaritain, Limbé	1,450	243	16.8	C. P. Raccurt et al., 1983 ²¹
1977 - 1984	A compilation of studies on filariasis conducted in 11 communities	6,779	667	9.8	C. P. Raccurt, 1986 ²⁴
1980 - 1981	Léogane city proper, Dampus, Chassagne and Ca-Ira	421	140	33.3	C. P. Raccurt et al 1988 ²²
1981 - 1982	Haïtian immigrants in 6 counties in central and south Florida	668	45	6.8	B. G. Yangco et al 1984 ²⁵

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PART 3

Countries with Transmission That Apparently Stopped after 1980

EVIDENCE SUGGESTS that the culture of Costa Rica on the eve of the Spanish arrival was quite similar to that of the Incas and Mayas.¹ The indigenous people include the Chorotegas of Guanacaste, who have gradually assimilated into the mainstream population; the Bribri; the Boruca; and the Cabecar people, all who reside in the valleys of the Talamanca Mountains of southern Costa Rica.²

Christopher Columbus came upon Costa Rica during his fourth voyage, where he arrived on September 18, 1502 and stayed for 17 days.^{1,2} He named it Costa Rica, which means "the rich coast" because he was impressed by the gold decorations worn by the friendly indigenous people.¹ Although the Spanish laid claim to the area, settlements were never established until the 1560s, when Cartago was established in the fertile central highlands.¹ The search for gold proved unsuccessful, and so additional settlements were not established.¹

In the 18th century, settlements such as Heredia, San José, and Alajuela were established.¹ In 1723, much of Cartago was destroyed by the eruption of "*Volcán Irazú*," but was subsequently rebuilt by its survivors.¹ In 1808, coffee was introduced and this spurred further development, which apparently invigorated the country and led to its independence from Spain on September 15, 1821.^{1,2}

The earliest reference to lymphatic filariasis is found in the reports of William F. Thornton, who in 1908, observed a worker with lymphatic filariasis

from a nearby banana plantation who had lived along the coastal region of Costa Rica for more than a year, before moving to Nicaragua.³ The patient was found to be positive for *Wuchereria bancrofti* microfilariae.³ In 1912, 1914, and 1917, other investigators made general references to the occurrence of lymphatic filariasis in Costa Rica.⁴⁻⁶

The first study on lymphatic filariasis was conducted in 1946 by D.C.A. Butts and covered four areas: one along the Pacific coast (Quepos) and the other three (Puerto Limón, Bataan, and Cahuita) along the Caribbean seaboard.⁷⁻⁹ The microfilaremia prevalence was 1/100 (1.0%) in Quepos; 2/100 (2.0%) in Cahuita; 3/100 (3.0%) in Bataan; and 101/1006 (10.0%) in Puerto Limón.⁷⁻⁹ In Puerto Limón, the survey was conducted in two subdivisions of the city: Cristóbal Colón (Cieneguita) and Barrio Roosevelt (Jamaica Town) (Tables 1-3).⁷⁻⁹

Of the 107 microfilaria-positive patients, 58 were either born or had lived more than 3 years in Puerto Limón; 48 lived elsewhere before coming to Puerto Limón; and one patient (a 25-year-old woman from Cahuita) had never been to Puerto Limón, but lived in close proximity to the other patient from Cahuita (a 40-year-old woman, who had once lived in Puerto Limón for 23 years).⁷⁻⁹ Infection prevalence was similar in males (8.2%) and females (8.2%); and was higher in blacks (100/935, 10.7%) than in whites (7/371, 1.9%).⁷⁻⁹

Table 1. Microfilaremia prevalence in a study on lymphatic filariasis conducted in 1946⁷⁻⁹

Region	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Quepos	100	1	1.0
Cahuita	100	2	2.0
Bataan	100	3	3.0
Puerto Limón	1,006	101	10.0
Total	1,306	107	8.2

Table 2. Distribution of microfilaremia prevalence by region and gender, a study by D. C. A. Butts, 1946⁷⁻⁹

Region	No. of males examined	No. of males microfilaria-positive	Microfilaremia prevalence (%)	No. of females examined	No. of females microfilaria-positive	Microfilaremia prevalence (%)
Quepos	50	1	2.0	50	0	0.0
Cahuita	59	0	0.0	41	2	4.9
Bataán	62	1	1.6	38	2	5.3
Puerto Limón	466	50	10.7	540	51	9.4
Total	637	52	8.2	669	55	8.2

Table 3. Distribution of microfilaremia prevalence by region and race, a study by D. C. A. Butts, 1946⁷⁻⁹

Region	No. of blacks examined	No. of blacks microfilaria-positive	Microfilaremia prevalence (%)	No. of whites examined	No. of whites microfilaria-positive	Microfilaremia prevalence (%)
Quepos	3	0	0.0	97	1	1.0
Cahuita	92	2	2.2	8	0	0.0
Bataán	42	3	7.1	58	0	0.0
Puerto Limón	798	95	11.9	208	6	2.9
Total	935	100	10.7	371	7	1.9

In 1953, another study was conducted in Barrio Roosevelt (Jamaica Town).¹⁰ The investigators observed that 21/137 (15.3%) persons were microfilaria-positive.¹⁰

In 1976, Weinstock and colleagues conducted two studies similar to those of Butts (1946), to determine the microfilaremia prevalence in Puerto Limón.¹¹ The studies involved four subdivisions of Puerto Limón, namely Roosevelt, Cuartel, Cementerio, and Pueblo Nuevo.¹¹ A total of 64/2,338 (2.7%) persons from the four subdivisions had

microfilaremia (Tables 4-7).¹¹

In one study, a random sample of 1,142 persons from all four subdivisions was conducted.¹¹ Of these, 25 (2.2%) were microfilaria-positive (Table 4).¹¹ In the second study, a total of 1,196 persons were selected from blocks of houses where one or more infected persons resided ("positive blocks of houses").¹¹ Of these, 39 (3.3%) were from three subdivisions; Roosevelt, Cuartel, and Cementerio, had microfilaremia (Table 6).¹¹

Table 4. Microfilaremia prevalence in a randomly selected sample in Puerto Limón, Weinstock et al., 1976¹¹

Subdivision of Puerto Limón	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Roosevelt	406	14	3.5
Cuartel	275	3	1.1
Cementerio	159	3	1.9
Pueblo Nuevo	302	5	1.7
Total	1,142	25	2.2

Table 5. Microfilaremia prevalence for "positive blocks of houses" in Puerto Limón, Weinstock et al., 1976¹¹

Subdivision of Puerto Limón	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Roosevelt	931	35	3.8
Cuartel	158	4	2.5
Cementerio	107	0	0.0
Total	1,196	39	3.3

Table 6. Distribution of microfilaremia prevalence by gender, in Puerto Limón, Weinstock et al., 1976¹¹

Study	No. of males examined	No. of males microfilaria-positive	Microfilaremia prevalence (%)	No. of females examined	No. of females microfilaria-positive	Microfilaremia prevalence (%)
Randomly selected sample	490	15	3.1	652	10	1.5
"Positive blocks of houses"	503	24	4.8	693	15	2.2
Total	993	39	3.9	1,345	25	1.9

Table 7. Distribution of microfilaremia prevalence by race in Puerto Limón, Weinstock et al., 1976¹¹

Study	No. of blacks examined	No. of blacks microfilaria-positive	Microfilaremia prevalence (%)	No. of whites examined	No. of whites microfilaria-positive	Microfilaremia prevalence (%)
Randomly selected sample	604	20	3.3	538	5	0.9
"Positive blocks of houses"	682	33	4.8	514	6	1.2
Total	1,286	53	4.1	1,052	11	1.1

The infection was more frequent in males (39/998, 3.9%) than in females (25/1,345, 1.9%), in blacks (53/1,236, 4.1%) than in whites (11/1,052, 1.1%).¹¹ It was highest in persons aged 50 and above (13/273, 4.8%), and in those aged 10 to 19 years (26/651, 4.0%).¹¹

In 1979, a fourth study was conducted in Roosevelt, where the authors observed that 21/119 (17.7%) persons were positive for microfilariae.¹² Of the 21 patients, 19 were black, and two were white.¹²

Between 1979 and 1980, a study was done in the remaining four subdivisions in Puerto Limón, (Limón Centro, Santa Eduvigis, Barrio Quinto and

Cristóbal Colón), where the researchers observed that 53/2,879 (1.8%) persons had microfilaremia (Tables 8-11).¹³ The infection was more frequent in males (31/1,276, 2.4%) than in females (22/1,603, 1.4%), in blacks (36/663, 5.4%) than in whites (17/2,216, 0.8%), and in persons aged 10 to 19 years (31/876, 3.5%).¹³

This study was extended to include the remainder of the province of Limón (13 rural communities), where 3/3,438 (0.1%) persons were found to be positive for microfilariae.¹³ The three infections were presumed to have been acquired in Puerto Limón.¹³

Table 8. Microfilaremia prevalence in Puerto Limón, Paniagua et al., 1979-1980¹³

Subdivision of Puerto Limón	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Cristóbal Colón	743	26	3.5
Santa Eduvigis	533	12	2.3
Limón Centro	914	8	0.9
Barrio Quinto	689	7	1.0
Total	2,879	53	1.8

Table 9. Distribution of microfilaremia prevalence by gender in Puerto Limón, Paniagua et al., 1979-1980¹³

Gender	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Female	1,603	22	1.4
Male	1,276	31	2.4
Total	2,879	53	1.8

Table 10. Distribution of microfilaremia prevalence by race in Puerto Limón, Paniagua et al., 1979-1980¹³

Race	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Black	663	36	5.4
White	2,216	17	0.8
Total	2,879	53	1.8

Table 11. Microfilaremia prevalence by age in Puerto Limón, Paniagua et al., 1979-1980¹³

Age group (years)	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
1-9	787	2	0.3
10-19	876	31	3.5
20-29	432	4	0.9
30-39	292	3	1.0
40-49	199	6	3.0
50 and above	293	7	2.4
Total	2,879	53	1.8

Table 12. Summary of studies on lymphatic filariasis, in Puerto Limón, 1946-1980

Year	Subdivision(s)	No. of persons examined	No. of persons microfilariae positive	Microfilaremia prevalence (%)	Author and year of publication
1946	Cristóbal Colón and Roosevelt	1,006	101	10.0	D. C. A. Butts, 1947 ⁷
1953	Roosevelt	137	21	15.3	H. Lieske, 1954 ¹⁰
1976	Roosevelt, Cuartel, Cementerio and Pueblo Nuevo	2,338	64	2.7	H. Weinstock et al, 1977 ¹¹
1979	Roosevelt	119	21	17.7	R. Brenes Madrigal et al, 1979 ¹²
1979 - 1980	Limón Centro, Santa Eduvigis, Barrio Quinto and Cristóbal Colón	2,879	53	1.8	F. Paniagua et al, 1983 ¹³

The results from all these studies indicate that Puerto Limón was endemic for lymphatic filariasis from 1946 to 1980 (Table 12).⁷⁻¹⁶ A limited focus of continuing low-level transmission may still remain.¹⁷ In the 1980s, microfilariae were observed in all age groups and the microfilaremia prevalence had been reported to range from 1.8% to 3.3%.¹⁶

In Costa Rica, lymphatic filariasis is believed to have been limited to Puerto Limón.¹⁶ The origin of filariasis in Puerto Limón, based on limited data, is attributed to immigration from Jamaica circa 1871.^{2,7-9,13} In the late 19th century, immigrants came to provide labor for industrialist Minor Keith of Brooklyn, New York, in the construction of the Northern Atlantic Railroad from Puerto Limón to San José and for the banana plantations.^{2,7-9,13} For many of these immigrants, movement throughout the country was restricted until 1949.² This may be one reason for the circumscribed focus of filariasis in Puerto Limón.¹³

The literature provides additional reports of isolated cases of filariasis in Costa Rica. In 1951, H. Paez documented a case of hematochyluria caused by *W. bancrofti* infection.¹⁸ In 1974, Brenes Madrigal reported a case of filariasis, in which microfilariae were reportedly observed in the spermatic artery.¹⁹ In both instances, the patients were presumed to have acquired the infection in Puerto Limón.^{18,19} In 1978, Rodríguez et al reported an unusual case of filariasis in a young Costa Rican woman, who had a subcutaneous nodule in the left arm.²⁰

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THE ORIGINAL INHABITANTS of Suriname are believed to have been primarily the Arawaks and the Caribs.¹⁻⁴ Christopher Columbus came upon the area during his third voyage in 1498.² In 1593, Spain officially claimed the area, even though Portuguese and Spanish explorers of the time paid little attention to it.² In 1616, Dutch settlements were established at the mouths of several rivers between present-day Georgetown in Guyana and Cayenne in French Guiana.² In 1650, the English established sugarcane and tobacco plantations on the west bank of the Suriname River and a settlement, which is now known as Paramaribo.^{1,3,4} The settlers were sent by Lord Willoughby, who was at that time Governor of Barbados.¹ In 1667, Zealander Abraham Crijnsen invaded this settlement.¹⁻⁴ Following this, the Dutch gained Suriname from the English, in exchange for their North American colony Nieuw Amsterdam, present-day New York City, at the "Treaty of Breda."¹⁻⁴

Upkeep of the plantations was expensive, and so in 1683, the Zealanders sold Suriname to the Dutch West Indian Company and the Van Sommelsdijck family.¹ In 1683, the head of the family, Cornelis van Aerssen heer van Sommelsdijck, became governor of Suriname and during his term improved upkeep and therefore the security of the plantations.¹ However, he was killed in 1688 during a revolt.¹

Throughout the late 1600s and early 1700s, agricultural yields were low.¹⁻⁴ To expand the plantations, slaves were imported in the late 1700s until emancipation on July 1, 1863.¹⁻⁴ During the late 19th century, the slaves were replaced by laborers

from the former Dutch East Indies.^{1,3,4}

Circa 1919, bauxite was discovered, and became an important contribution to both the economy of Suriname and to the World War II effort, as aluminum made from bauxite was used in the construction of airplanes.^{1,2} In 1954, Suriname became a self-governing state and on November 25, 1975, it gained its independence.¹⁻⁵

The earliest reference to lymphatic filariasis in Suriname was obtained from the reports of P.C. Flu, who from 1909 to 1910, conducted investigations on *Wuchereria bancrofti* infection in different racial groups in Paramaribo, the capital, which is located on the northeastern coast.⁶⁻⁸ He observed that in low-income Creoles, 50% of males and 60% of females were positive for microfilariae.⁶⁻⁸ In addition, 25% of Jews, 23% of high-income Creoles, 6% of East Indians, 1.2% of Europeans, and 0.7% of Indonesians had microfilaremia.⁶⁻⁸

In 1927, P.C. Flu conducted another study in Paramaribo, in which 64/224 (28.6%) persons were positive for microfilariae.^{7,8} In 1947, A.E. Wolff examined 4,851 persons (children under age 5 years were excluded).⁹ Of these, 1,067 (22.0%) had microfilaremia.⁹ On December 31, 1948, a sample of 1,214 persons was randomly selected from the population in Paramaribo.¹⁰ Of these, 345 (28.4%) were microfilaria-positive.¹⁰

Several studies on lymphatic filariasis were conducted in the city of Paramaribo from 1927 to 1981 (Tables 1 and 2).

Table 1. Studies on lymphatic filariasis in Paramaribo, 1927-1981

Year	Population/Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1927	Weidstreet in Paramaribo	224	58	25.9	P. C. Flu, 1928 ⁷
1940	Conscripts	—	—	30.0	Cited from J. F. B. Edeson and F. Kuhlow
1947	General population (children under 5 years were excluded)	4,851	1,067	22.0	A. E. Wolff, 1948 ⁹
1948	Creoles	1,214	345	28.4	P. H. J. Lampe, 1950 ¹⁰

Table 1. (Cont.) Studies on lymphatic filariasis in Paramaribo, 1927-1981

Year	Population/Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1949-1951	First large-scale survey	50,861	8,857	17.4	J. Fros, 1953 ¹¹
1954-1956	General population	45,000	2,745	6.1	E. Van der Kuyp in 1959, cited from C. F. A. Bruyning, 1961 ¹²
1959-1961	Second large-scale survey	39,167	3,525	9.0	E. Van der Kuyp, 1965 ²¹
1969-1971	Third large-scale survey	79,613	1,672	2.1	B. F. J. Oostburg, 1974 ²²
1979-1981	Fourth large-scale survey	51,097	33	0.1	B. J. F. Oostburg, 1985 ⁵

Table 2. Clinical manifestations of lymphatic filariasis in Paramaribo, 1949-1981

Year	Population/Setting	No. of persons examined	No. of persons with elephantiasis	Elephantiasis prevalence (%)	No. of persons with lymphangitis	Lymphangitis prevalence (%)	Author and year of publication
1949	General population	1,214	—	—	230	19.0	P. H. J. Lampe, 1950 ¹⁰
1949-1951	First large-scale survey	50,861	469	5.3	—	—	J. Fros, 1953 ¹¹
1959-1961	Second large-scale survey	39,167	431	1.1	—	—	E. Van der Kuyp, 1965 ²¹
1969-1971	Third large-scale survey	79,613	801	1.0	436	0.6	B. F. J. Oostburg, 1974 ²²
1979-1981	Fourth large-scale survey (group 1)	51,097	316	0.6	271	0.5	B. J. F. Oostburg, 1985 ⁵
1979-1981	Fourth large-scale survey (group 2)	3,168	15	0.5	10	0.3	B. J. F. Oostburg, 1985 ⁵

In 1949, the Bureau of Public Health established the Section of Filariasis Control.⁸ The first large-scale survey of Paramaribo was conducted from 1949 to 1951; investigators found that 8,857/50,861 (17.4%) persons were positive for microfilariae.¹¹ Of the microfilaria-positive persons, 7,112 (80.3%) were treated with diethylcarbamazine (DEC).^{8,12} In 1952, 1 year after treatment, 5,690 (80.0%) of these persons were microfilariae-negative.^{8,12}

In 1967, records from the Filariasis Control Clinic show that 95/1,687 (5.6%) patients were positive for microfilariae.¹³ During the same year, 70/3,347 (2.1%) hospital patients were found to be positive for microfilariae.¹³ Between January and

September 1968, 63/2,558 (2.5%) blood films taken from patients at the same hospital were microfilaria-positive.¹³ The prevalence of microfilariae remained low (less than 1.0%) in the large-scale surveys for the next 2 decades (Table 3).

Between November 20, 1979 and October 20, 1981, Oostburg identified 33 persons who had microfilaremia.⁵ Of these, 27 (81.8%) were from Guyana.⁵ Several studies were also conducted in areas outside the city of Paramaribo from 1940 to 1981 (Tables 3 and 4).

Table 3. Lymphatic filariasis in areas outside the city of Paramaribo, 1940-1981

Year	Town and District or population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1940-1945	Conscripts from rural districts	—	—	15.0 (includes <i>M. ozzardi</i>)	Cited from J. F. B. Edeson and F. Kuhlrow, 1969 ¹³
1947	Nieuw Nickerie, Nickerie District	100	—	17.0 (includes <i>M. ozzardi</i>)	A. E. Wolff, 1948 ⁹
1950	Moengo, a bauxite mining village along Cottica River in Marowijne District	1,716	256	14.9	B. S. Blumberg, J. McGiff and I. Guicherit, 1951 ²³
1950	Nieuw Nickerie, Nickerie District	—	—	10.0	Cited from E. Van der Kuyp, 1955 ⁸
1951	Nieuw Nickerie, Nickerie District	—	—	10.0	J. Fros, 1953 ¹¹ ; J. F. B. Edeson and F. Kuhlrow, 1969 ¹³
1953	Kwatta, in the District of Wanica	—	—	2.5	E. Van der Kuyp, 1953, cited from E. Van der Kuyp, 1955 ⁸
1953	Para villages, in the District of Para	353	12	3.4	E. Van der Kuyp, 1953, cited from E. Van der Kuyp, 1955 ⁸
1953	Coronie, in the District of Coronie	—	—	4.6	E. Van der Kuyp, 1953, cited from E. Van der Kuyp, 1955 ⁸
1953	Coronie, in the District of Coronie	601	23	3.8	Cited from J. F. B. Edeson and F. Kuhlrow, 1969 ¹³
1953	Lelydrop, in Wanica District	266	12	4.5	E. Van der Kuyp, 1965 ²¹
1954	Weg naar Zee (Fernandesweg), District of Suriname	243	6	2.5	E. Van der Kuyp, 1965 ²¹
1954-1956	Eerste Rijkweg, Tweede Rijkweg, and Uitvlugt (all in District of Suriname)	—	—	1.2	E. Van der Kuyp, 1965 ²¹
1956	Bigi Poika, near the Saramacca River (Lowland Amerindians in the Savannah Belt)	173	1	0.6	J. Fros, 1956 ²⁴
1956	Areas near the Suriname River (Bushnegroes and Creoles in the Savannah Belt)	232	9	3.9	J. Fros, 1956 ²⁴ ; WHO, 1961 ²⁵
1956	Areas near the Coppename River (Hindustanis in the Savannah Belt)	7	1	14.3	J. Fros, 1956 ²⁴ ; WHO, 1961 ²⁴⁵
1957	Nieuw Nickerie, Nickerie District	2,919	111	3.8	E. Van der Kuyp, 1965a; J. F. B. Edeson and F. Kuhlrow, 1969 ¹³
1962	Pad van Wanica, Disitric of Suriname	3,810	46	1.3	B. F. J. Oostburg, 1962 ²⁶
1963	Albina, Marowijne District	312	8	2.6	Cited from J. F. B. Edeson and F. Kuhlrow, 1969 ¹³

Table 3. (Cont.) Lymphatic filariasis in areas outside the city of Paramaribo, 1940-1981

Year	Town and District or population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1963	Nieuw Nickerie, Nickerie District	2,619	92	3.5	E. Van der Kuyp, 1965a; J. F. B. Edeson and F. Kuhlow, 1969 ¹³
1963	Rural Nickerie, Nickerie District	1,382	6	0.4	Cited from J. F. B. Edeson and F. Kuhlow, 1969 ¹³
1963	Moengo, Marowijne District	1,834	11	0.6	Cited from J. F. B. Edeson and F. Kuhlow, 1969 ¹³
1963	Surroundings of Moengo, Marowijne District	1,145	8	0.7	Cited from J. F. B. Edeson and F. Kuhlow, 1969 ¹³
1964	Coronie, in the District of Coronie	1,677	29	1.7	Cited from J. F. B. Edeson and F. Kuhlow, 1969 ¹³
1969-1971	Various towns across the country (excluding Paramaribo)	41,132	170	0.4	B. F. J. Oostburg, 1974 ²²
1969-1971	Various towns in District of Suriname	12,616	88	0.7	B. F. J. Oostburg, 1974 ²²
1970	Nickerie, in Nickerie District	—	—	1.0	B. F. J. Oostburg, 1974 ²²
1979	New Nickerie, in Nickerie District [++]	5,886	10	0.2	B. F. J. Oostburg, 1983 ¹⁴
1979	New Nickerie, in Nickerie District [**]	567	7	1.2	B. F. J. Oostburg, 1983 ¹⁴
1979-1981	Various towns in District of Suriname [++]	56,318	21	0.03	B. F. J. Oostburg, 1985 ⁵
1979-1981	Various towns in District of Suriname [**]	3,034	10	0.1	B. F. J. Oostburg, 1985 ⁵
1979-1981	Marienburg, Zoelen, and Backdam, District of Commwijnine [++]	410	12	2.9	B. F. J. Oostburg, 1985 ⁵
1979-1981	Marienburg, Zoelen, and Backdam, District of Commwijnine [**]	410	26	6.3	B. F. J. Oostburg, 1985 ⁵
1979-1981	Victoria, District of Brokopondo [++]	192	12	6.3	B. F. J. Oostburg, 1985 ⁵
1979-1981	Victoria, in the District of Brokopondo [**]	192	18	9.4	B. F. J. Oostburg, 1985 ⁵

++ thick blood smear technique

** membrane filter technique

Where no technique is specified, the thick blood smear technique was used.

Table 4. Clinical manifestations of lymphatic filariasis in areas outside the city of Paramaribo, 1969-1981

Year	Town and District	No. of persons examined	No. of persons with elephantiasis	Elephantiasis prevalence (%)	No. of persons with lymphangitis	Lymphangitis prevalence (%)	Author and year of publication
1969-71	Various towns in District of Suriname	12,616	38	0.3	13	0.1	Oostburg, 1974 ²²
1979	New Nickerie in Nickerie District	5,886	16	0.3	222	3.8	Oostburg, 1983 ¹⁴
1979	New Nickerie in Nickerie District	567	1	0.2	14	2.5	Oostburg, 1983 ¹⁴
1979-81	Various towns in District of Suriname	56,318	117	0.2	97	0.2	Oostburg, 1985 ⁵
1979-81	Various towns in District of Suriname	3,034	10	0.3	6	0.2	Oostburg, 1985 ⁵

In 1979, in New Nickerie, located on the western border of Suriname, the thick blood smear technique and the membrane filter technique were used to detect microfilariae.¹⁴ By using the thick blood smear, 10/5,886 (0.2%) persons had microfilaremia, while 7/567 (1.2%) other persons were microfilaria-positive by the membrane filter technique.¹⁴ Thus, 17 persons were infected with *W. bancrofti*.¹⁴ Of these, 16 (94.1%) were originally from Guyana while one (5.9%) was from Suriname, but had made frequent visits to Guyana.¹⁴

Between November 20, 1979 and October 20, 1981, Oostburg identified 21 (0.03%) persons in the District of Suriname who were positive for microfilariae, using the membrane filter technique.⁵ Of these, 18 (85.7%) were originally from Guyana.⁵ In Marienburg, Commewijne District, of 410 workers originally from Guyana, 26 (6.3%) and 12 (3.0%) persons were positive for microfilariae, using thick blood smear and membrane filtration, respectively.⁵

In Victoria, Brokopondo District, of 192 workers screened using the membrane filter technique, 18 persons had microfilaremia.⁵ Of these, 16 (88.9%) were from Guyana and the remaining two (11.1%) were from Haïti.⁵ Of the entire group of workers, 180 (93.8%) were from Guyana, thus 16/180 (8.9%) workers from Guyana were microfilaria-positive.⁵ In addition, 12/192 (6.3%) workers were from Haïti and therefore, 2/12 (16.7%) workers from Haïti were infected.⁵

In 1956, the peak biting density of the vector was found to be 20 *Culex fatigans* per person/hour, from hand catches in houses of residents of Paramaribo.¹³ Mosquito density was highest during the major rainy season from April-June and lowest

from September-October.¹³ Other reports document an infective rate of up to 0.7% and an infection rate of 8.3% in *Aedes aegypti*.¹³ Between 1979 and 1981, Oostburg and collaborators collected and dissected mosquitoes from Marienburg, District of Commewijne and Victoria, District of Brokopondo (Table 5), but no such work was conducted for Paramaribo or the rest of Suriname.⁵

Table 5. *Wuchereria bancrofti* infection in *Culex quinquefasciatus* in Paramaribo and other towns in Suriname, 1927-1981

Year	Town and District	Number of mosquitoes dissected	Number of mosquitoes infected	Infection rate of mosquitoes (%)	Author and year of publication
1927	Paramaribo	638	182	28.5	P. C. Flu, 1928 ⁷
1950	The Old City Center of Paramaribo (during 1 st large-scale survey just before treatment with DEC)	1,187	304	25.6	G. F. A. Bruyning, 1953 ²⁷
1950	The Old City Center of Paramaribo (1 year after 1 st large-scale survey and treatment with DEC)	875	55	6.3	G. F. A. Bruyning, 1953 ²⁷
1950	From 6 central areas of Paramaribo (after large-scale survey and treatment with DEC)	4,437	426	9.6	G. F. A. Bruyning, 1953 ²⁷
1951	From 6 central areas of Paramaribo (after large-scale survey and treatment with DEC)	3,670	319	8.7	G. F. A. Bruyning, 1953 ²⁷
1957	Nickerie, in Nickerie District	201	7	3.5	T. W. Reyenga, 1958 ²⁸
1959-1961	Paramaribo	—	—	0.5	Cited from E. Van der Kuyp, 1965 ²¹
1969-1971	Paramaribo	5,239	2	0.04	B. F. J. Oostburg, 1974 ²²
1979	New Nickerie in Nickerie District	1,416	0	0.0	B. F. J. Oostburg, 1983 ¹⁴
1979-1981	Marienburg, District of Commewijne	48	0	0.0	B. F. J. Oostburg, 1985 ⁵
1979-1981	Victoria, District of Brokopondo	61	0	0.0	B. F. J. Oostburg, 1985 ⁵

In 1942, E.P. Snijders described a case of chyluria in a Surinamese male patient with filariasis, who at that time lived in the Netherlands.^{15,16} In the late 1960s, others investigators (F.P. Bol, A.C. Klinkhamer, G. Bosman, and H.C.E.M. Houtappel) reported six additional cases of chyluria in Surinamese immigrants, which were presumed to be lymphatic filariasis.¹⁷⁻¹⁹

Between 1961 and 1980, 15 patients with chyluria were seen at the Clinic for Tropical Diseases, Royal Tropical Institute, Amsterdam.¹⁶ The authors observed that 14/15 (93.3%) patients came from Suriname, 11 of whom had lived in Paramaribo, while the remaining one patient came from Guyana, but had lived in Suriname for many years.¹⁶ The clinical findings were suggestive of lymphatic filariasis in

7/15 (46.7%) patients; 4/15 (26.7%) patients had hydrocele and one had painful inguinal adenopathy.¹⁶

The results of the 1979-1981 mass survey of lymphatic filariasis and previous reports suggest that lymphatic filariasis is disappearing in Suriname (Table 1).^{5,20}

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THE ORIGINAL INHABITANTS of Trinidad are believed to have included at least seven groups: the Arawaks, Chaimas, Tamanagues, Salives, Chaguanes, Quaquas, and Caribs, while Tobago was inhabited only by the Caribs.^{1,2} It is said that Christopher Columbus came upon both islands during his third voyage in 1498.^{1,2}

In 1530, Don Antonio Sedeño became the first Spanish governor, but was unable to establish a permanent settlement because of numerous attacks by the original inhabitants.¹ In 1592, another governor, Don Antonio de Berrio y Oruna, founded the town of San José de Oruna (present-day Saint Joseph), but in 1595, it was destroyed by Sir Walter Raleigh and not rebuilt until 1606.¹ The Caribs aggressively prevented successful colonization in Trinidad, until the end of the 17th century when Catalan Capuchin missionaries arrived.¹ During this period, the two islands were a source of contention amongst the French, the Dutch, the British, and the Spanish.¹

In 1783, a policy known as the "Royal Cedula of Population" was introduced to encourage immigration of Roman Catholics to Trinidad.^{1,2} The implementation of this policy created organized settlements, which began with the influx of mostly French-speaking immigrants, particularly after the French Revolution.¹ Other immigrants came from Saint Lucia and Dominica, when these islands were ceded to Britain in 1784.¹ Others came with their slaves from the French Caribbean, when slavery was abolished from Saint Domingue (Haïti), after the country declared its independence.¹

In 1797, the British captured Trinidad.¹ In 1802, Spain ceded Trinidad to Britain under the "Treaty of Amiens."¹ In 1763, Tobago was ceded by France to Britain under the "Treaty of Paris," but it was not until 1802, after further invasions by the French and subsequent recapture by the British, that it was once again ceded to Britain, becoming a Crown Colony in 1877.¹ In 1888 it amalgamated politically with Trinidad.¹

During the period between 1791 and 1802, slaves were imported to work in the sugarcane fields, which were initially planted by the French, until emancipation in 1807.¹ In 1834, as labor became scarce, the British government encouraged

immigration by offering higher wages.¹ Several thousand immigrants came to Trinidad from neighboring islands, from Baltimore, Maryland and Philadelphia, Pennsylvania in the United States, and from England, Scotland, Ireland, France, Germany, and Switzerland in Europe.¹

In 1844, the British government approved the importation of indentured laborers from India, but by 1866, there was a dwindling influx of East Indians.¹ In 1849, the British government also approved the importation of indentured laborers from China, but by 1917, Chinese immigration ceased.¹

Trinidad and Tobago became an independent member of the British Commonwealth on August 31, 1962, and became a republic within the Commonwealth on August 1, 1976.¹

The earliest reference to lymphatic filariasis appears in annual reports of the Surgeon General of Trinidad and Tobago between 1893 and 1937.³ An average of 24 persons with elephantiasis and 54 persons with hydrocele received hospital treatment each year.³ In 1902, George A. Vincent visited Trinidad and examined patients at the Colonial Hospital in Port-of-Spain, Saint Ann's Lunatic Asylum, and from private medical practices.^{4,5} He observed that 25/500 (5.0%) patients were positive for microfilariae and 33 (6.7%) patients had elephantiasis.⁴ During the same year, a medical student from Trinidad at Howard University, Washington D.C., was found to be microfilaria-positive during a routine blood examination.⁶

In 1908, George C. Low visited Trinidad, and examined a number of patients.⁷⁻⁹ He observed that 83/400 (20.8%) had elephantiasis and 43 (10.8%) were positive for microfilariae.⁷⁻⁹ Of these, 19/43 (44.2%) patients had signs or symptoms of lymphatic filariasis and 24/43 (55.8%) were asymptomatic.⁷⁻⁹ In 1909, A.B. Herrick reported a case of chyluria and microfilaremia in a patient who was originally from Trinidad, but had lived in Panama for 22 years.¹⁰

In 1928, the report of the Surgeon General of Trinidad and Tobago stated that "the disease (filariasis) occurs, but is not an important or prominent cause of sickness."³ In 1945, at a naval base located in Trinidad, 33,970 employees were examined for microfilariae.^{11,12} The investigators

found that 35 Trinidadian employees and one Tobagoan employee were microfilaria-positive and that most of them had never left Trinidad.^{11,12} In 1955, reports document that lymphatic filariasis was uncommon as a cause of death¹³⁻¹⁵ and, in 1965, M.T. Ashcroft stated that lymphatic filariasis was uncommon in Trinidad.¹⁶

In 1976, G. Nelson and J.B. Davis observed that 19/300 (6.3%) day blood samples were positive for microfilariae in a village near the north coast of Trinidad.¹⁷ They also identified a few cases of mild lymphedema and one case of hydrocele during the investigation.¹⁷

Between May 1978 and September 1979, M.B.Nathan and colleagues made efforts to determine the microfilaremia prevalence and the infection and infectivity rates of *Culex quinquefasciatus* mosquitoes in Blanchisseuse in Northern Trinidad.^{3,18} Of 562 persons examined, 86 (15.3%) had microfilaremia.^{3,18} Twelve (2.1%) persons, who were amicrofilaremic, had lymphedema of the leg.³ Of 2,006 resting mosquitoes collected, 141 (7.0%) were infected, while

2 (0.1%) mosquitoes harbored mature larvae.¹⁸

The aim of another study conducted between February 1980 and April 1981 was to determine the effects of mass chemotherapy, using spaced doses of diethylcarbamazine citrate (DEC) on microfilariae in the population and on vector infection rates.¹⁹ The treatment regimen was 6 mg/kg body weight given at monthly intervals for 1 year.¹⁹

Before treatment, 76/525 (14.5%) persons living in Blanchisseuse were microfilaria-positive.¹⁹ Of 699 *C. quinquefasciatus* mosquitoes, 45 (6.4%) were infected and one (0.1%) was infective with *Wuchereria bancrofti*.¹⁹ Twelve months after treatment began, 9/525 (1.7%) persons were still microfilaria-positive, but the geometric mean microfilariae density in microfilaria-positive persons was reduced from 19.7 per ml to 4.6 per ml.¹⁹ The post-treatment mosquito infection prevalence was zero in 2,211 *C. quinquefasciatus* mosquitoes.¹⁹

Several studies on microfilaremia prevalence and mosquito infection were conducted in Trinidad and Tobago (Tables 1 and 2).

Table 1. Studies on lymphatic filariasis in Trinidad and Tobago, 1902-1992

Year	Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	No. of persons with lymphedema	Lymphedema prevalence (%)	Author and year of publication
1902	Patients from Colonial Hospital, Port-of-Spain, the Saint Ann's Lunatic Asylum, and from private medical practices	500	25	5.0	33	6.6	G. A. Vincent, 1902 ⁴
Circa 1907	Unknown	400	43	10.8	83	20.8	G. C. Low, 1908 ⁷ ; Low, 1908 ⁸ ; Low, 1908 ⁹
1943-1945	At a naval base in Trinidad	—	36	—	—	—	R. F. Platzer and W. K. A. Lawlor, 1945 ¹¹
1976	A village near the north coast of Trinidad	300	19	6.3	—	—	G. S. Nelson and J. B. Davis, 1976 ¹⁷
1978-1979	Blanchisseuse, located on the northern coast of Trinidad	562	86	15.3	—	—	M. B. Nathan et al., 1982 ³

Table 1. (Cont.) Studies on lymphatic filariasis in Trinidad and Tobago, 1902-1992

Year	Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	No. of persons with lymphedema	Lymphedema prevalence (%)	Author and year of publication
1980-1981	Pre-treatment survey conducted in Blanchisseuse	525	76	14.5	—	—	M. B. Nathan et al., 1987 ¹⁹
1981	1-year post-treatment survey in Blanchisseuse	525	9	1.7	—	—	M. B. Nathan et al., 1987 ¹⁹
1992	12 years after mass chemotherapy, Blanchisseuse	348	0	0.0	—	—	D. D. Chadee et al., 1995 ²⁰

In 1992, 12 years after mass chemotherapy with diethylcarbamazine (DEC) in Blanchisseuse, Chadee and colleagues examined 348/520 (66.9%) of the total population for the presence of microfilaremia.²⁰ None

of the 348 persons were microfilaria-positive.²⁰

At present, transmission, if it exists, is believed to be limited to the northern coast of Trinidad.²¹

Table 2. *Wuchereria bancrofti* infection in *Culex quinquefasciatus* in Blanchisseuse, Trinidad, 1978-1981

Year	Location and Setting	No. of mosquitoes dissected	No. of mosquitoes infected	Infection rate of mosquitoes (%)	No. of mosquitoes with L3	Infectivity rate of mosquitoes (%)	Author and year of publication
1978-1979	Indoor resting collection	2,006	141	7.0	2	0.1	M. B. Nathan, 1981 ¹⁸
1980-1981	Pre-treatment baseline	699	45	6.4	1	0.1	M. B. Nathan, 1987 ¹⁹
1981	1-year post-treatment	2,211	0	0.0	—	—	M. B. Nathan, 1987 ¹⁹

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PART 4

Countries with Transmission That Apparently Stopped before 1980

THE ORIGINAL INHABITANTS of these islands were the Sibonay, known as the “stone people,” who are believed to have arrived here around 10,000 B.C.^{1,2} The next group of people to colonize the islands were the Arawaks, who came from South America about 2,000 years ago.^{1,2} The third group were the Caribs, who were initially based in Dominica.²

Christopher Columbus came upon Antigua during his second voyage, in November 1493.^{2,4} He named it after a Cathedral in Seville, Spain, which was called “*Santa Maria de la Antigua*.”^{2,3} Although the Spanish laid claim to the islands, settlements were never established until the arrival of the English.⁴

In 1632, English settlers and their leader Edward Warner (son of Sir Thomas Warner of Saint Kitts), arrived from Saint Kitts to establish small farms of tobacco, ginger, and indigo.^{2,4} The French landed at Deep Bay in 1666 and occupied Antigua for 8 months until it was given back to the English in the “Treaty of Breda.”^{2,4} In 1674, Colonel Christopher Codrington arrived from Barbados and introduced the latest sugarcane planting techniques.⁴

The sugarcane plantations became so successful during the 17th and 18th centuries that, in order to protect their interests, in 1672 the English built several forts, the ruins of which are still seen today.¹ To meet the requirements of the plantations, more slave labor was introduced onto the island during the 1670s.^{2,4} Slavery lasted until emancipation on August 1, 1834.^{2,4} The islands remained under English rule from 1667 until 1967, when Antigua attained statehood.^{1,3} On November 1, 1981, Antigua became independent.^{1,3}

The earliest reference to lymphatic filariasis in Antigua was by James Hendy, Physician to His Majesty’s Naval Hospital at Barbados and Physician General to the Militia of the Island - Barbados.^{4,7} He reported one case of elephantiasis seen in Antigua in his “Treatise on the glandular disease of Barbados, proving it to be seated in the lymphatic system,” which was published in 1784.^{4,7} In 1829, J.M. Titley described a case of chylous lymph scrotum in a patient in Antigua.^{4,8} Although there were no accounts of lymphatic filariasis in the writings of local physicians before 1838, two laymen, who resided in Antigua, J. Sturge and T. Harvey, mentioned the occurrence of

elephantiasis on the island.^{4,9}

The disease had become such a serious problem towards the end of the 19th century that it dominated discussions at the meeting of members of the Leeward Islands Branch of the British Medical Association in 1891 and 1892.^{4,7,10-13} In the 1891 meeting, A.P. Boon stated that he had seen a dozen cases of elephantiasis of the scrotum and many cases of hydrocele in 18 years of practice (1873-1891) on Antigua.^{4,10}

In 1892, F.J. Freeland reported that he had seen numerous cases of chyluria in his practice.^{4,11} He documented three cases of chyluria, where the patients were positive for microfilariae in both peripheral blood and in urine.¹¹ W.J. Branch stated that he had seen many cases of elephantiasis and was in agreement with Boon on possible clinical manifestations of lymphatic filariasis: pain, fever, and lymphantigitis.^{4,12,14} In 1892, C.R. Edwards noted the rarity of hydrocele in boys as opposed to adult males and stated that in all cases of elephantiasis of the scrotum, hydrocele was also observed.^{4,13}

Several cases of lymphatic filariasis that were acquired in Antigua were reported at the turn of the 20th century.¹⁵ For example, in 1914, in Montreal, Canada, George C. Armstrong saw a 14-year-old girl from Antigua, who for 2 years had painful swellings in the groin, from which enlarged glands were surgically removed.¹⁵ Shortly after, the child became seriously ill with nausea, vomiting, and delirium.¹⁵ She fell into a semi-conscious state, and died.¹⁵ The cause of death was said have been a virulent streptococcal infection of the lymph glands in the groin, which progressed to sepsis.¹⁵ Microfilariae were also found in the peripheral blood of the child.¹⁵ Eric C. Marshall in 1915, and D.M. Grisworld in 1918, referred to lymphatic filariasis as a prevalent disease in Antigua.^{4,16,17}

In 1937, F.W. O’Connor conducted a study to determine the microfilaremia prevalence in Antigua.⁴ The island was divided into three parallel regions based on its three distinct geological strata, which traverse the island in a northwest to southeast direction.⁴ The western region includes volcanic rocks, the central area consists mainly of clay, and the northern and eastern parts are composed limestone.⁴

O’Connor observed that 130/619 (21.0%)

persons residing in the western volcanic area, 429/1,631 (26.3%) persons from the central clay area and 54/276 (19.6%) persons from the northern and eastern limestone area had microfilaremia.⁴ A total of 613 (24.3%) persons ranging in age from 3 to 87 years were found to be microfilaria-positive.⁴ In addition, 24/75 (32.0%) inmates at the Feines Institute were

positive for microfilariae.⁴ Table 1 summarizes microfilaremia prevalence in the three distinct geological soil regions, while Tables 2-4 provide details of microfilaremia prevalence in towns located in the three areas.

Table 1. Microfilaremia prevalence, Antigua, by geologic region, 1937⁴

Region	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Volcanic area	619	130	21.0
Clay area	1,631	429	26.3
Limestone area	276	54	19.6
Total	2,526	613	24.3

Table 2. Microfilaremia prevalence in the western area of volcanic soil, Antigua, 1937, O'Connor⁴

Town/Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Liberta	192	53	27.6
Old Road	11	2	18.2
Crabb's Hall	2	1	50.0
Belvedere	2	0	0.0
Sawcolts	28	8	28.6
Jennings	77	22	28.6
English Habour	129	7	5.4
Falmouth Harbour	98	23	23.5
Five Islands	24	5	29.8
Bishops	6	0	0.0
John Hughes	28	7	25.0
Bolans	5	0	0.0
Bendals	10	2	20.0
Breakneck	1	0	0.0
Christian Hill	2	0	0.0
Blubber	1	0	0.0
Urlings	1	0	0.0
Hamiltons	1	0	0.0
Roses	1	0	0.0
Total	619	130	21.0

Table 3. Microfilaremia prevalence in the central area of clay soil, Antigua, 1937, O'Connor⁴

Town/Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Saint Johns	604	114	18.9
Green Bay	185	44	23.8
Grays Hill	8	3	37.5
Gray Farm	70	22	31.4
Freetown	8	0	0.0
Saint Johnstons Village	10	0	0.0
All Saints	146	44	0.1
Sweets	178	52	29.2
Potters	164	60	36.6
Freemans	51	22	43.1
Seaview Farm	124	48	38.7
Bethesda	74	19	25.7
Buckleys	5	0	0.0
Delaps	2	0	0.0
North Sound	1	1	100.0
Burkes Estate	1	0	0.0
Villa	1	0	0.0
Lyons Estate	1	0	0.0
Total	1,631	429	26.3

Table 4. Microfilaremia prevalence in the north and eastern area of limestone soil, Antigua, 1937, O'Connor⁴

Town/Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Newfields	3	1	33.3
Parham	104	26	25.0
Willkies	7	1	14.3
Piggots	15	5	33.3
Cedar Grove	108	13	12.0
Barned Hill	7	1	14.3
Winthrops	13	3	23.1
Seatons	4	2	50.0
Fitches Creek	3	0	0.0
Mercers Creek	1	0	0.0
Langfords	1	1	100.0
Blackmans	1	1	100.0
Judges Estate	2	0	0.0
Wetherells	1	0	0.0
Sion Hill	1	0	0.0
Cotton Estate	2	0	0.0
Bettys Hope	1	0	0.0
Donovans	1	0	0.0
Lepers Home	1	0	0.0
Total	276	54	19.6

According to published official national reports beginning in 1953, there were 85 cases of filariasis; in 1954 (123); 1955 (89); 1956 (115); 1957 (62); 1958 (35); and 1959 (12).¹⁸ In 1950, 1957, and 1959, two, one, and one deceased persons, respectively, were also reported to have had filariasis.¹⁸

In November 1958, to determine the prevalence of protozoan and helminthic infections among migratory agricultural workers in Clewiston, Florida, Henry Beye, Charles Brooks, and Elizabeth Guinn collected blood specimens from 470 workers

originally from the British West Indies.¹⁹ Of three migratory workers from Antigua, one was positive for microfilariae.¹⁹

K.H. Uttley compiled the rates of death considered to be due to filariasis from the annual reports of Antigua for the period 1918-1957.^{20,21} A total of 357 deaths had been attributed to filariasis.^{7,20} He observed that severe elephantiasis had diminished considerably in frequency during the previous 100 years (Table 5).^{20,22}

Table 5. Rates of death attributed to lymphatic filariasis per 1,000 population, Antigua (Uttley, 1959)²⁰

Year	Rate per 1,000	Year	Rate per 1,000
1918	0.17	1938	0.08
1919	0.17	1939	0.06
1920	0.28	1940	0.05
1921	0.17	1941	0.05
1922	0.38	1942	0.00
1923	0.17	1943	0.05
1924	0.20	1944	0.05
1925	0.03	1945	0.08
1926	0.10	1946	0.05
1927	0.32	1947	0.03
1928	0.06	1948	0.00
1929	0.06	1949	0.00
1930	0.06	1950	0.02
1931	0.06	1951	0.00
1932	0.09	1952	0.00
1933	0.12	1953	0.00
1934	0.09	1954	0.00
1935	0.09	1955	0.00
1936	0.11	1956	0.00
1937	0.03	1957	0.02

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THE ARAWAKS are believed to have been the initial inhabitants of the island,^{1,2} but when the Portuguese explorer Pedro a Campos charted Barbados in the 17th century, he found the island uninhabited.^{1,2} He named it "*Os Barbados*" which means "the bearded ones," after the bearded fig trees, which grew on the beaches.^{1,2} Before departure, he left some wild pigs on the island.^{1,3}

In 1625, after departure from Pernambuco in Brazil, the *Olive*, an English ship, accidentally arrived on the shores of Barbados,³ where the crew found fruits and wild hogs.^{1,3} They claimed the island for King James I.³ On February 17, 1627, another English ship, the *William and John*, arrived with 80 settlers and 10 slaves,^{1,3} who established a town called Jamestown (later called Holetown).²

Within a few years, there were about 40,000 settlers on the island, mostly small yeomen farmers.^{1,3} However, after the "sugarcane revolution" of the 1650s, most of these farmers left for other islands.^{1,3} From about 1640 to 1650, labor on the sugarcane plantation was replaced through the massive importation of slaves, which continued until emancipation in 1844.^{1,3,4}

In addition to the growing populations of settlers and slaves, Britain exported diverse groups to the island.³ These included unrepentant Royalists from the Civil War, prisoners taken by the Commonwealth forces in the Scottish and Irish campaigns, Nonconformists, and Quakers.³ Within 70 years after the first settlement was established, the island was extensively deforested and heavily populated.^{3,5}

A strong fortress system – 26 forts along 34 kilometers (21 miles) of coast – kept the island from invasion, and Barbados became the only island in the Caribbean to remain under uninterrupted British rule for more than 3 centuries.² In 1966, Barbados received its full sovereignty.²

The earliest record of elephantiasis in the Americas comes from Barbados, where Richard Towne, a local doctor, in 1726, published his treatise on the diseases of the West Indies, especially Barbados.^{3,6} He stated that "Elephantiasis as a disease..... has a great affinity to the best Accounts we have of the Leprae of the Arabians." In 1750, the Reverend Griffith Hughes, a rector of Saint Lucy's

Parish, recorded that "Elephantiasis is likewise a Disease very common..... and hitherto incurable."^{7,8}

William Hillary, a medical practitioner in Barbados from 1747 to 1758, included a section on elephantiasis in his published work "Observations on the changes of the air, and the concomitant epidemical diseases of the Island of Barbadoes."^{8,9} He stated that elephantiasis occurred among slaves and settlers alike.³

In 1784, James Hendy, Physician to His Majesty's Naval Hospital at Barbados and Physician General to the Militia of the island,^{8,10,11} wrote about elephantiasis in his "Treatise on the glandular disease of Barbadoes, proving it to be seated in the lymphatic system."¹⁰ The author stated "The Glandular disorder was unnoticed in the Island till eighty years ago." thereby dating the first clinical manifestations of this disease on the island to about 1704.^{10,12}

Hendy gave a detailed account of the clinical symptoms of filarial elephantiasis among settlers and slaves, relating 26 case histories.¹⁰ He recognized that the same condition was called "Saint Thomas's leg" in India.^{3,10,13} Hendy stated that the condition was also found in Ceylon (Sri Lanka) and Japan, as quoted from the writings of the doctoral thesis of Englebert Kaempfer.^{10,14} He recommended a lace stocking as beneficial in reducing the swelling.¹⁰

From all accounts, within 70 years of extensive deforestation and settlement, poor living conditions, overcrowding and immense importation of indentured labor and slaves, elephantiasis had become well established on the island.^{3,5}

Barbados is believed to have become a center from which lymphatic filariasis spread to South Carolina in the United States and islands in the Caribbean.^{3,11,12,15-19} Many Caribbean islands received immigrants – settlers and slaves – from Barbados during the 17th century.^{3,11,12,15-19} Elephantiasis was so common in Barbados during the 17th century that in the English-speaking New World, it was called "Barbados leg."^{3,5,11,12,17,18,20-22}

In 1854, P. Duchassaing wrote that the use of rainwater or stagnant water as a source of drinking water predisposed one to filarial infection in Barbados.^{3,23} On September 20, 1899, at the Boston City Hospital, a 22-year-old man from Barbados was

examined, and tested positive for microfilariae in both blood and hydrocele fluid.²⁴ Subsequent examination of his asymptomatic 21-year-old brother revealed that he also was microfilaremic.²⁴

In 1901, George C. Low visited Barbados, and examined patients from all over the island at the General Hospital and the Central Almshouse, as well as patients in private medical practices.²⁵⁻²⁹ Of 600 patients, 76 (12.7%) were positive for microfilariae.²⁵⁻²⁹ Of these, 27 (35.5%) had signs and symptoms suggestive of filarial disease and 49 (64.5%) were asymptomatic.^{25,27-29} *Culex fatigans* mosquitoes were collected from the wards and corridors of the General Hospital; 23/100 (23.0%) mosquitoes were infected with larvae at various stages of development, while one (1.0%) mosquito harbored mature L3 larvae.²⁵

In 1902, of 500 recent settlers in Trinidad originally from Barbados, two (0.4%) had microfilaremia.³⁰ In 1907, at Saint Elizabeth's Hospital in Boston, a Barbadian-born male presented with chyluria due to filariasis.³¹ In 1908, 5/29 (17.2%) Barbadians living in Guyana were examined and found to be positive for microfilariae.³² In 1916, J.F. Siler reported that although cases of elephantiasis

were still present in Barbados, both the number and the severity of cases were much reduced.³³

In official national reports for 1951 and 1952, two cases of filariasis were reported for each year.³⁴ In 1959, K.H. Uttley stated that M.A. Byer, a Barbadian pathologist, had not seen microfilariae in the blood of any resident examined since 1933.³⁵ In 1965, M.T. Ashcroft also stated that in personal communications with Byer and a "Dr. Adams," they informed him that the prevalence of microfilaremia was very low and that elephantiasis was rarely seen.³⁶

In November 1958, to determine the prevalence of protozoan and helminthic infestation among migratory agricultural workers in Clewiston, Florida, Henry Beye, Charles Brooks, and Elizabeth Guinn collected blood specimens from 470 workers originally from the British West Indies.³⁷ Two (1.8%) of 113 migratory workers from Barbados were positive for microfilariae.³⁷

Cases of lymphatic filariasis were reportedly seen in 1975,³⁸ and in 1979, Hawking wrote that annual medical reports indicated that lymphatic filariasis was present and assumed to occur throughout Barbados.³⁹

Table 1. Studies on lymphatic filariasis in Barbados, 1901-1958

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1901	General Hospital, Central Almshouse and private medical practice	600	76	12.7	G. C. Low, 1901 ²⁵ ; Low, 1902 ²⁶ ; Low, 1908 ²⁷ ; Low, 1908 ²⁸ ; Low, 1908 ²⁹
1902	Barbadians recently settled in Trinidad	500	2	0.4	G. A. Vincent, 1902 ³⁰
1908	Barbadians recently settled in Guyana	29	5	17.2	K. S. Wise, 1909 ³²
1958	Migratory agricultural workers in Clewiston, Florida	113	2	1.8	H. Beye, C. Brooks and E. Guinn, 1961 ³⁷

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THE ORIGINAL INHABITANTS of Colombia are believed to have included at least three groups: the Chibchas, the Caribes, and the Arawaks.¹ The Chibchas occupied the high plains, the cool central region, and the Sierra Nevada de Santa Marta; the Caribes lived along the Atlantic coast; and the Arawaks lived in the Amazon, in the Putumayo, and Caqueta river basins.¹

It is said that Alonso de Ojeda, a companion of Christopher Columbus, landed on the Guajira Peninsula, on the Atlantic coast in 1499.^{1,3} A year later, Rodrigo de Bastidas arrived in the same area.³ The first Spanish settlements were established in 1509 and 1510 by Alonso de Ojeda, at Santa María la Antigua de Darién (present-day Acandí), on the western side of the Golfo de Urabá.^{1,3} In 1525, Rodrigo de Bastidas established Santa Marta, currently the oldest existing city in Colombia.^{1,3} Expeditions continued inland and on August 6, 1538, an expedition led by Gonzalo Jiménez de Quesada established Santafé de Bogotá.^{1,3}

The original inhabitants tolerated the arrival of the Spaniards, but rebelled when attempts were made at colonization and land confiscation.⁴ Soon after, the Spaniards, who included conquistadors, administrators, and Roman Catholic clergy, established a number of towns, including Cartagena in 1533.² In 1544, Colombia was incorporated into the viceroyalty of Peru, where it remained until 1739, when it then became a part of New Granada.^{2,3} New Granada was a region comprising the territories of modern-day Colombia, Venezuela, Ecuador and Panama.^{2,3}

In the 1500s, the economy was based upon mining precious metals and jewels, but by the 1700s, sugarcane and tobacco became important export commodities, in part as a result of the exhaustion of mineral and metal resources.³ Labor was initially provided by the original inhabitants, who were replaced by slaves sometime after 1593.³ Slavery persisted until emancipation in 1848.³

After a 14-year struggle, in which Simón Bolívar Palacios and his troops won the battle of Boyacá in Colombia on August 7, 1819, independence was attained from Spain in 1824.⁴ Simón Bolívar united Colombia, Venezuela, Panama, and Ecuador

into the Republic of Greater Colombia.⁴ After he died, on December 17, 1830, Venezuela and Ecuador seceded.⁴

The earliest reference to lymphatic filariasis is found in the reports of Ricardo de la Parra, who in 1868, described cases of elephantiasis in detail.^{5,6} In addition, he mentioned the possibility of a filarial worm as the etiologic agent.^{5,6} In 1889, Manuel Uribe Angel observed cases of lymphangitis and elephantiasis, and recognized that these clinical manifestations were a consequence of *Wuchereria bancrofti* infection.^{6,7}

In 1891, Juan Bautista Londoño observed cases of elephantiasis in Antioquia, in the western region, and reported a series of cases of elephantiasis that possibly had etiologic agents other than *W. bancrofti*.^{6,8} In 1892, Andrés Posada Arango made splendid observations of elephantiasis and determined its epidemiologic pattern in Colombia.^{6,9} In 1894, Evaristo García reported cases of elephantiasis that he had treated with surgery.^{6,10}

In 1905, Pedro Luis Alvarez stated in his thesis for graduation in medicine that most cases of elephantiasis in Antioquia were not the result of filarial infection.^{6,11} In 1906, Pedro Quesada published a report on the frequency of hospital patients with hydrocele and elephantiasis in Cartagena, which is on the north Atlantic coastal region.^{6,12} In 1909, A.B. Herrick observed a case of chyluria in a Colombian man at the Ancón Hospital in Panama.¹³

Eramos Baños conducted the first extensive study on the parasitologic and clinical aspects of lymphatic filariasis in Colombia in 1911.^{6,14} Eight persons who lived on the Atlantic coast were found to be positive for *W. bancrofti* microfilariae.^{6,14} This study was published as a thesis for graduation in medicine at the Universidad Nacional.^{6,14} Other reports also indicate that *W. bancrofti* had been detected at least since 1917.^{15,16} In 1918, Julio Gómez and Manuel Obregón reported on the endemicity of filariasis in Cartagena and on its treatment with Salvarsan.^{6,17}

In November 1929, a foreign worker was seen at the Barranca Hospital with chronic lymphadenitis of the groin.¹⁸ He was microfilaria-positive.¹⁸ During the months of January and February 1930, 15

workmen from the Tropical Oil Concession in Barranca-Bermeja were admitted to the El Centro Hospital with symptoms suggestive of filariasis.¹⁸ Eight (53.3%) were positive for microfilariae.¹⁸ Between January and December 1930, 12 workmen from the same Concession were seen by the Medical Department of the Concession with clinical manifestations suggestive of filariasis.¹⁸ They were later found to be positive for microfilariae.¹⁸ The workmen were treated with intravenous injections of either tartar emetic or T.A.B. vaccine (mixed typhoid-paratyphoid AB) protein shock therapy.¹⁸ The treatment reportedly was associated with some reduction in microfilaremia, and apparently all workers recovered and resumed work at the Concession.¹⁸ Filariasis was considered indigenous to the provinces in the valley that border the Magdalena River, midway between the Caribbean and Pacific coasts.¹⁸

In 1935, Augusto Gast Galvis reported a case of filariasis from the central part of the country, in which microfilariae were detected in blood collected during the day.^{6,19} In 1942, E.L. Ferreira in Mompós, in northwestern Colombia, described an operation for treatment of an advanced case of elephantiasis, presumed to be of filarial origin.^{6,20} In 1951, Eduardo Acosta published the results of an experiment in which a patient from Cartagena was successfully infected with *W. bancrofti* larvae carried by laboratory-bred *Culex fatigans* mosquitoes.^{6,21}

In 1952, E.C. Faust, M. Agosín, A. García-Laverde, W.Y. Sayad, V.M. Johnson, and N.A. Murray reported two unusual cases of suspected filariasis.²² One patient, seen in mid-August 1950, was a 60-year-old woman, a native of Palestine, who had once lived in Port-au-Prince, Haiti for 9 years, and subsequently 11 years in Cartagena.²² She was examined by Moisés Agosín in Santiago, Chile.²² On examination, her upper and lower limbs showed symmetrical erythema, desquamation, and non-pitting edema of moderate degree.²² Her differential white blood count revealed 11% and 8% eosinophilia, on 2 successive days.²² Examination of a sample of her blood revealed worms morphologically similar to *W. bancrofti* microfilariae in most respects, but considerably larger.²²

The other patient, a 34-year-old man from Buena Ventura, in the southwestern region, was seen on March 31, 1948 at Hospital San Juan de Dios, Bogotá.²² He had a history of pain in the epigastrium and a painful enlargement in the right cervical

region.²² On examination, he was asthenic, febrile, had tenderness in the epigastric region, and a painful, enlarged, hard, nonmovable, right cervical lymph node.²² He had anemia, a total white cell count of 47,000 with relative neutropenia and marked lymphocytosis.²² He died the following morning.²² On autopsy, acute lymphoid leukemia was tentatively confirmed and a pulmonary nodule was found to contain an adult female *W. bancrofti* worm.²²

In 1952, filarial disease was reported in the northern coast near Cartagena, in the valley of the Magdalena River, and at Buena Ventura, on the Pacific coast.^{22,23} In 1958, Julio Blair examined six patients with elephantiasis, who were amicrofilaremic and were treated surgically.⁶ In 1965, a 70-year-old farmer, who lived in a rural area of River Lebrija, Santander del Sur, in northeastern Colombia, was examined by David R. Botero, Alberto M. Restrepo, and Hernán A. Vélez.⁶ The patient had clinical signs of lymphatic filariasis, was found to be amicrofilaremic, but harbored an adult female worm in a cervical lymph node.⁶

In 1974, C.J. Marinkelle indicated in a personal communication that two cases of *W. bancrofti* infection had been seen; one to the east and one to the west of Cartagena.²⁴

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THE EARLIEST INHABITANTS of the island of Cuba are said to have come from South America around 3500 B.C.¹ This group was later joined by the Arawaks (Taino), who were primarily farmers.¹ On October 27, 1492, Christopher Columbus (Cristobal Colon) came upon the island during his first voyage.¹ He called it "*Juana*," thinking that he had found the Asian mainland.³ Although Cuba was known to Spanish merchants and explorers, no settlements were established until 1512, when Diego Colon (Columbus' son) established Santiago.^{1,3} Within the same year, Diego Velázquez de Cuéllar led a 300-member party from Hispaniola to Cuba, to claim the island for the Spanish Crown.¹ He later became governor of Cuba,³ establishing seven settlements within 2 years.¹ Following this inauspicious start, Cuba remained under Spanish rule for the next 4 centuries.³ In 1513, landowner Amandor de Lares brought 4 slaves from Hispaniola, and this appears to be the first record of slavery in Cuba.³ In 1514, the city of Habana was established by Pánfilo de Narváez.³

Large estates were established under the "*encomienda*" system, which essentially enslaved the Arawaks under the pretext of providing religious instruction.¹ In 1520, the first large group of slaves (300) arrived to work in a gold mine called Jaugua, and were continually brought to Cuba until emancipation on October 7, 1886.³ By 1542, when the "*encomienda*" system was abolished, the Indian population had been decimated.^{1,3} The original inhabitants were soon replaced by more slaves imported to work on cattle ranches.¹ Bovine products – leather and dried beef – were the mainstay of the Cuban economy until the early 18th century, when tobacco became the principal cash crop of the colony.¹

On June 6, 1762, the British invaded Cuba, capturing and occupying Havana for 11 months.¹ During this period, more slaves were imported and trading links were expanded.¹ As a result, sugarcane became the major industry and by 1820, Cuba was the world's largest producer of sugar from sugarcane.¹

After the revolutionary activities in the region between 1810 and 1825, Cuba and Puerto Rico were the only remaining Spanish colonies in the Western Hemisphere.¹ Spanish loyalists fled former colonies, arriving en masse in Cuba.¹

On October 10, 1868, the island's first war of independence, called "*Grito de Yara*," began at Yara, and was led by a Creole planter, Carlos Manuel de Céspedes.^{1,3} After 10 years of fighting, in which Antonio Maceo y Grajales and Máximo Gómez, among many others, played primary roles,³ a pact was signed with Spain that granted the former, amnesty.^{1,3}

Among those granted amnesty in 1878 was José Julián Martí y Pérez, a famous poet and advocate for independence.^{2,3} He left Cuba shortly thereafter only to return in 1895 to participate in the second war of independence, "*Grito de Bara*," which began on February 24, 1895.³ José Julián Martí y Pérez died during one of the first skirmishes of the war which he had organized.^{2,3} Gómez and Maceo also participated in this war against Spain, which lasted until December, 1898.³

In January 1898, the U.S. warship *Maine*, set sail for Cuba,^{1,3} with the primary objective of protecting United States concerns and interests in Cuba.³ It was anchored just outside the Havana harbor, and on February 15, 1898, was destroyed in an explosion.¹ The United States blamed Spain, and so began the Spanish-Cuban-American War.³ The U.S. formally declared war on April 25, 1898 and hostilities continued until December of the same year,³ when the Spanish surrendered.^{1,3} On January 1, 1899, the United States installed a military government on the island³ and stipulations were made so that the United States would respect the right of Cuba to self-determination, while occupying Cuba militarily.^{1,3} On May 20, 1902, Cuba declared its independence and a governor was installed by the United States to oversee reconstruction.^{1,3}

By the 1920s, two-thirds of the farmland on the island was owned by U.S.-based companies.^{1,3} The collapse of commodity prices and the struggle for political stability in the 1920s and 1930s led to civil unrest in Cuba.^{1,3} Many of these episodes were quelled by Gerardo Machado y Morales, who had assumed power on May 20, 1925, only to resign on August 4, 1933.^{1,3} Carlos Manuel de Céspedes, the son of Carlos Manuel de Céspedes of the *Grito de Yara* took over, but was overthrown on September 4, 1933, in a coup led by army sergeant Fulgencio Batista.^{1,3} Batista ruled for more than 20 years and

was ousted in 1959, in a campaign led by Fidel Castro, the current president.^{1,3}

There are no available records of either elephantiasis or chyluria in Cuba from the time of the Arawaks to the early part of the 16th century, when many slaves were imported.⁵ The initial discovery of microfilariae of *Wuchereria bancrofti* occurred in Paris on August 28, 1863.⁶ The organisms were detected in the milky chylocele fluid collected from a young man who was originally from Havana.⁶

In 1881, Charles (Carlos) J. Finlay demonstrated microfilariae in blood obtained from a patient in Havana, Cuba⁷⁻⁹ and in 1884, he made similar findings in another patient.¹⁰ Finlay informed his colleague, John Guit  ras, who within the same year, also observed microfilariae in blood drawn from four Cubans who resided in Key West, Florida (one of whom was the patient seen in 1884 by Finlay).¹⁰

At the turn of the 20th century, lymphatic filariasis was an important disease in Cuba, as indicated by the numerous accounts of cases reported by several authors.¹¹⁻²¹ Nu  ez compiled data on patient records at Hospital Mercedes from March 1886 to January 1906.^{21,22} He observed that 356/62,548 (0.6%) patients at the hospital were diagnosed with filariasis, "exhibiting one form of clinical expression or another."^{21,22} He also compiled data from Hospital No. 1, from 1900 to July 1904.^{21,22} Of 19,586 patients, 70 (0.4%) were diagnosed with filariasis.^{21,22}

On April 20, 1903, a 37-year-old man was seen at Hospital de Nuestra Se  ora de las Mercedes, Havana.²³ He had a febrile illness for 18 days, and had a positive Widal test.²³ His illness did not abate and he subsequently died.²³ On autopsy, microfilariae of *W. bancrofti* were seen in the pulmonary arteries.²³ In 1904, Mario G. Lebreo performed experiments to confirm Manson's work – that larvae mature in mosquitoes (in this case, *Culex*), move to the proboscis, and fall onto the human skin during a blood meal.^{15,16,24} In 1910, M.G. Lebreo conducted a study in which 207 (17.8%) of 1,161 persons had microfilaremia.^{25,26} Most infected persons had no clinical manifestations, and were children between the ages 2 to 16 years old.^{25,27}

In 1915, Julio Ort  z Cano, a surgeon, is reported to have isolated one adult male and five adult female worms from the spermatic cord of a patient.²⁵ The worms were described and identified as *W. bancrofti* by Leonel Plasencia.²⁸ In 1919, E. Francis published the results of a filariasis survey conducted

in nine cities in the southern United States.²⁹ All nine persons with microfilaremia had "histories either of having been born and raised in Charleston or having spent a considerable period of time in Charleston or of having been born in Cuba which is a well-known filarial focus."²⁹

In 1925, Pedro Kour   reported seeing four patients who were microfilaria-positive.^{30,31} In 1927, he also identified adult worms, both males and females, extracted from tissue excised by a surgeon, Ricardo Mach  n, from enlarged inguinal lymph nodes and the spermatic cord.^{30,31}

On December 30, 1927, L.F. Rodr  guez Molina observed a case of filariasis with an unusual clinical manifestation, "vesical filariasis" in a 20-year-old man, which was mistaken for a tumor of the bladder.^{24,30,32} In 1939, Kour  , Sanuevo, and Sotolongo Guerra collected night blood smears from patients in Havana hospitals.²⁴ Only one patient was microfilaria-positive.²⁴ In 1945, employees at a Naval Base in Trinidad were examined, and only one Cuban employee was microfilaremic.^{33,34}

In 1946, a surgeon, Joaqu  n Corbera y Caridad extracted adult worms from tissue excised from the swollen left inguinal cord of one of his patients.³⁵ In 1949, Antonio P  rez Ara reported a case of epididymitis in a patient from Havana.³¹ Adult filarial worms were found in adjacent tissue and extracted.³¹

In 1979, M. Lastre examined a patient who, for 40 years, had had a hydrocele.²⁶ He was microfilaremic and had never traveled outside Cuba.²⁶ In 1980, 16 African students were found to be positive for microfilariae and treated with diethylcarbamazine and levamisole.^{36,37} In 1981, only one (4.6%) of 22 other African students was positive for microfilariae.^{36,37}

In 1982, an indigenous case of filariasis from the city of Pinar del R  o was reported, in which the patient had elephantiasis of the lower limbs.²⁶ Microfilariae were found in fluid drawn from a testicular hydrocele.²⁶ The patient had lived his entire life in Pinar del R  o.²⁶ As a consequence of this discovery, the Instituto de Medicina Tropical "Pedro Kour  " adopted the use of a filarial antibody assay to identify additional autochthonous cases in which the patient may not have had microfilaremia.²⁴

In 1983, 377 blood samples were tested for microfilariae and antifilarial antibody; 256 smears were from persons living in Pinar del R  o, in the neighborhood of a man with elephantiasis; 111 smears were from blood bank donors; and 10 smears were

from patients with clinical signs of filariasis, residing in formerly disease-endemic areas.²⁷ No microfilariae were detected.²⁷ However, an antifilarial antibody titer of 1:512 was observed in 41/256 (16.0%) neighbors, in 3/10 (30.0%) filarial patients, and no blood bank donors.²⁷ These results suggested that the two populations had at one time been in contact with *W. bancrofti*.²⁷

In 1985, 60 patients with symptoms of lymphangitis and lymphedema of the lower limb were examined at the Institute of Angiology.²⁷ It was unclear whether any of these cases were of filarial etiology. Investigations conducted to determine the prevalence of filarial infection are compiled in Table 1.

Table 1. Studies on lymphatic filariasis in Cuba, 1886-1990

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1886-1906	Patients from Hospital Mercedes	62,548	356	0.6	Nuñez, 1906 ²²
1900-1904	Patients from Hospital No. 1	19,586	70	0.4	Nuñez, 1906 ²²
1910	Mostly asymptomatic children 2-16 years old	1,161	207	17.8	M. G. Lebrede, 1910 ²⁵
1945	Naval base in Trinidad	—	1	—	R. F. Platzer et al., 1945 ³⁴
1981	African students	22	1	4.6	J. A. Pividal Grana, et al., 1981 ³⁷
1983	Neighbors of a man with elephantiasis living in Pinar del Rio	256	41 (‡)	16.0 (‡)	G. Hormilla et al., 1990 ²⁷
1983	Blood bank donors	111	0	0.0	G. Hormilla et al., 1990 ²⁷
1983	Patients with clinical signs of filariasis, residing in previously endemic areas	10	3 (‡)	30.0 (‡)	G. Hormilla et al., 1990 ²⁷

‡ antifilarial antibodies

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DOMINICA is believed to have been colonized initially by the Arawaks, followed by the Caribs, who called it "Wai'tukubuli," which means "tall is her body."¹⁻³ Christopher Columbus came upon the island during his second voyage on November 3, 1493, a Sunday, hence its name, from the word "Domingo."¹⁻³ Just as with Saint Kitts, Antigua, and Guadeloupe, the Spanish laid claim to the island, but settlements were not established until the 1600s.^{1,2}

In 1607, Captain John Smith and his followers stopped at the Dominican coastal settlement of Portsmouth for a couple of days before heading north to establish Jamestown, the first permanent English settlement in North America.³ In 1635, France laid claim to the island, and a few years later sent a contingent of missionaries, who were driven off by the Caribs.³

During the 17th and 18th centuries, control of the island was hotly contested by the British, the French, and the Caribs.^{1,2} In 1660, the two European powers agreed to leave Dominica to the Caribs, but the arrangement lasted only a few years.^{1,3} In 1686, the island was declared neutral, again with little success, as France and England renewed hostilities.¹ The Caribs, who were divided between the opposing forces, suffered heavy losses.¹ French settlers from the neighboring French West Indies began establishing coffee plantations on the island towards the end of the 17th century.³ In the 1720s, France sent a governor, who took formal possession of the island.³

For the remainder of the 18th century, Dominica changed hands between the French and British several times.³ In 1763, under the "Treaty of Paris," the

French reluctantly ceded Dominica to the British and in 1795 and 1805, unsuccessfully tried to recapture the island.³ After 1805, the island remained firmly in the possession of the British, who established sugarcane plantations on its more accessible slopes.³ Nevertheless, its position between the French colonies of Guadeloupe and Martinique and the strong French presence over the years ensured that, despite being under English rule, the French influence remained.³

By 1939, Dominica was still part of the Leeward Islands Federation, but in 1940 it became part of the Windward Islands Federation.¹⁻³ It gained full internal autonomy in 1967 as a West Indies Associated State and became an independent republic within the Commonwealth on November 3, 1978.¹⁻³

The earliest evidence of lymphatic filariasis on the island was reported in 1902, when George C. Low found 11 (7.6%) of 144 persons positive for microfilariae.⁴⁻⁷ Two of these persons had elephantiasis.⁵⁻⁷

In 1945, 33,970 employees at a Naval Base in Trinidad were examined.^{8,9} Of those who were microfilaria-positive, three were from Dominica.^{8,9} In 1959, R.H. Uttley wrote that the annual health reports for the country in 1937 and 1956 had indicated that filariasis was present, but specific data on these cases were unavailable.¹⁰⁻¹² J.S. Robinson had informed Uttley that *Wuchereria bancrofti* was not found on the island at that time, though occasional cases of elephantiasis were observed.¹²

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Dominica.¹²

Table 1. Studies on lymphatic filariasis in Dominica, 1902-1945

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1902	Unknown	144	11	7.6	G. C. Low, 1902 ⁴ ; Low, 1908 ⁵ ;
1945	Naval base in Trinidad	—	3	—	Low, 1908 ⁶ ; Low, 1908 ⁷ Platzer, 1945 ⁸ ; Beye, 1961 ⁹

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THE ORIGINAL INHABITANTS of French Guiana are believed to have been primarily the Arawaks and the Caribs.^{1,2} In 1498, during his third voyage, Christopher Columbus passed by the French Guianian coast without stopping.^{1,3,4} In 1500, Vicente Pinzon disembarked on the French Guianian coast.⁵ In circa 1503, a group of colonists established a settlement on a nearby island for some years, and called it Cayenne.³ From 1604-1624, under Henry IV, expeditions were conducted on the mainland, the northeastern coast, one of which was to become the present-day capital, Cayenne.^{3,5}

By the mid-17th century, the Dutch, British, Spanish, and French had established colonies in the region.¹⁻⁴ In 1817, although territorial and commercial arrangements shifted frequently, France consolidated its control over the region.^{2,4} The economic mainstays of the colony became sugarcane and rainforest timber.² Slaves were brought to work the plantations.² The output from plantations never matched that of other French Caribbean colonies, as their success was limited by tropical diseases and the hostility of the original inhabitants.² After emancipation in 1848, the local sugarcane industry virtually collapsed.^{1-3,5}

The colony enjoyed a brief period of prosperity in the 1850s when gold was discovered, but this soon dwindled.^{4,5} Between 1852 and 1939, some 70,000 prisoners were shipped to French Guiana, to reduce the cost of prisons in France and to contribute to the development of the colony.^{2,3,5} The last penal settlement was closed in 1953.²

On March 14, 1946, French Guiana became a French Overseas Department, under which the territory effectively became an integral part of France.²⁻⁵ In the 1960s, the European Space Centre and a satellite launching base were established in Kourou.^{2,5}

The earliest record of lymphatic filariasis is found in the reports of A. Clarac, who in 1902 saw and described cases of lymphangitis, lymphadenitis, elephantiasis, hydrocele, chyluria, and hematochyluria in French Guianese patients.^{6,7} In 1909, E. Brémont examined the blood of 17 patients from various hospitals and penal settlements in Saint-Laurent-du-Maroni, but none were microfilaremic.^{7,8}

In 1914 and 1915, together with Brémont, J. Thézé examined 133 patients from a hospital in Cayenne and found that 37 (27.9%) had microfilaremia, while 24 (18.0%) had lymphedema of the leg.⁹ In 1917, M. Léger and E. Brémont estimated the microfilaremia prevalence of the general population in Cayenne to be 27.4%.⁷ Within the same year, E. Brémont and M. Léger conducted a study of filariasis in 230 residents of Cayenne.⁷ They found that 58 (25.2%) were microfilaria-positive.⁷

In 1920, M. Léger tested 55 children who were younger than 15 years old; nine (16.4%) were positive for microfilariae.¹⁰ On January 23, 1934, J. Tisseuil examined 47 patients at the Institute of Hygiene in Cayenne and found 11 (23.4%) persons who were microfilaria-positive.¹¹ Within the same year, Tisseuil found that 45/241 (18.7%) persons from the general population of Cayenne were microfilaria-positive.¹¹ In 1935, he reported that 2/20 (10.0%) patients at the Institute of Hygiene in Cayenne had microfilaremia.¹¹

In 1944 and 1945, H. Floch conducted an investigation in Cayenne and observed that 59/430 (13.7%) participants were positive for microfilariae.¹²⁻¹⁴ Of these microfilaremics, 47 were originally from French Guiana, eight were originally from Martinique and four were originally from Guadeloupe.¹²⁻¹⁴ Therefore, of 327 persons from French Guiana, 47 (14.4%) were microfilaria-positive.¹²⁻¹⁴ The 430 participants involved in the study included 165 military personnel, 54 patients from the General Hospital, 137 patients from a hospital in Saint-Denis, and 66 children and eight adults from the Pasteur Institute.¹³ Of 2,143 *Culex fatigans* mosquitoes collected and dissected during this study, 236 (11.0%) were infected with filarial larvae.^{12,13}

In 1946, the authors conducted another study and found that 56/376 (14.9%) persons and 3/51 (5.9%) children in Cayenne had microfilaremia, as did 3/54 (5.6%) persons and 3/21 (14.3%) children in rural villages.^{12,13}

From 1945 to 1951, 53 persons with clinical manifestations suggestive of lymphatic filariasis were seen at the General Surgery Service at the Central Hospital in Cayenne and at the Pasteur Institute in Cayenne.¹⁵ Of these, 38 (71.7%) were microfilaria-

positive.¹⁵ Investigations on the prevalence of

microfilaremia in French Guiana are shown in Table 1.

Table 1. Studies on lymphatic filariasis in French Guiana, 1914-1951

Year	Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1914-1915	Patients from a hospital in Cayenne	133	37	27.9	J. Thézé, 1916 ⁹
1917	General population of Cayenne	179	49 [50*]	27.4	E. Brémont and M. Léger, 1917 ⁷
1917	Residents of Cayenne	230	58	25.2	E. Brémont and M. Léger, 1917 ⁷
1920	Children 15 years and younger	55	9	16.4	M. Léger, 1920 ¹⁰
1934	Patients at the Institute of Hygiene, Cayenne	47	11	23.4	J. Tisseuil, 1936 ¹¹
1934	Sample of general population of Cayenne	241	45	18.7	J. Tisseuil, 1936 ¹¹
1935	Patients at the Institute of Hygiene, Cayenne	20	2	10.0 [12.0*]	J. Tisseuil, 1936 ¹¹
1944- 1945	165 military personnel; 54 patients from the General Hospital; 137 patients from Saint-Denis; 66 children and 8 adults from Pasteur Institute, Cayenne	430	59 [includes 8 from Martinique and 4 from Guadeloupe]	13.7	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1944-1945	Same study as above, but only French Guianese	327	47	14.3	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1945	Residents of Cayenne	376	56	14.9	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1945	Children in Cayenne	51	3	5.9	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1945	Children in rural villages	54	3	5.6	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1945	Children in rural villages	21	3	14.3	H. Floch, 1946 ¹² ; H. Floch and P. de Lajudie, 1947 ¹³
1945-1951	Patients at the General Surgery Service, Central Hospital, Cayenne	53	38	71.7	M. Casile, 1952 ¹⁴

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THE INHABITANTS of Guadeloupe at the time of the arrival of Christopher Columbus were the Caribs, who called the island "*Karukera*," which means "island of beautiful waters."^{1,2} Christopher Columbus came upon the island during his second voyage in November 1493, and named it after a monastery in Spain, "*Santa Maria de Guadalupe de Estremadura*."

^{1,2} Similar to Saint Kitts and Antigua, the Spanish laid claim to the island, but settlements were never established until the arrival of the French.^{1,2}

In 1635, French settlers arrived with indentured labor.^{1,2} Slaves were imported in 1650, until emancipation in 1848.^{1,2} In the first half of the 17th century, Guadeloupe did not enjoy the same levels of prosperity as its neighbors.¹ In 1759, the English invaded the island, but it was later returned to France in 1763, under the provisions of the "Treaty of Paris."¹ In 1810, the English reinvaded the island, but handed it back to Louis XVIII during the Restoration, only to recapture it for a third time during the Napoleonic era.¹ The English finally handed over the island to France in 1815, when Napoleon was exiled to Saint Helena, and it has since remained a department of France.^{1,2}

Guadeloupe is an archipelago of nine inhabited islands, of which Basse-Terre, Grande-Terre, and Marie-Galante are the three largest.³ Basse-Terre is volcanic in origin with interior mountains, Grande-Terre is of low limestone formation and fairly flat, while most of the seven other islands are volcanic in origin.^{3,4}

The earliest reference to lymphatic filariasis was by P. Duchassaing in 1854, who stated that "the apparent use of rainwater or stagnant water predisposed to infection in Guadeloupe."^{5,6} On July 27, 1870, Jules Crévaux, a French naval surgeon, observed larval embryos in the urine (hematochyluria) of a young Creole in Guadeloupe.^{7,8} Isolated cases of lymphatic filariasis were soon documented by other authors: A. Corre in 1872, Venturini in 1880, and M. Lanceraux in 1888.⁹⁻¹³

The earliest filariasis survey in Guadeloupe was conducted by L. Stévenel in 1913.¹⁴ He reported that four (33.3%) of 12 state employees (civil servants) examined at the Military Hospital in Pointe-à-Pitre, Grande-Terre were microfilaria-positive.¹⁴ Within the

same year, Clarac, Leboeuf and Rigollet gave accounts that Guadeloupe was heavily affected by lymphatic filariasis.^{15,16} In 1914, M. Léger and R. Le Gallen conducted a study in Marseilles, France, in which 23/150 (15.3%) conscripts from Guadeloupe were positive for microfilariae.¹⁷

In 1935, M. Advier examined the blood smears of patients at the Laboratoire de Bactériologie at Pointe-à-Pitre.^{13,18} Of 110 blood smears, 20 (18.2%) were positive for microfilariae.^{13,18} In 1936, M. Advier and Jolly conducted a blood survey which revealed that 15/153 (9.8%) persons were microfilaria-positive.¹³ In 1938, Jolly conducted another survey in which 12/249 (4.8%) persons had microfilaremia.^{13,19}

In 1944 and 1945, H. Floch conducted an investigation in French Guiana and observed that 59/430 (13.7%) participants were microfilaria-positive, of whom four were from Guadeloupe.^{20,21} The infected conscripts were among 33 Guadeloupeans who participated in the study, so that 4/33 (12.1%) Guadeloupean conscripts were positive for microfilariae.^{20,21}

In 1949 and 1950, J. Sautet and J. Languillon examined 149 inmates at the leprosarium on the island of Désirade, where none were microfilaria-positive.^{13,22-24} In 1949, E. Monestruuc, R. Blache, and R. Laborde conducted a study to determine the microfilaremia prevalence of 110 young conscripts from all over the island.²⁵ Of these, 15 (13.6%) were microfilaria-positive.²⁵

Between February 1964 and December 1966, a sample of 3,553 conscripts selected from the Dugommier Camp near Pointe-à-Pitre was examined for the presence of microfilaremia at the Pasteur Institute of Guadeloupe.¹³ The study was conducted by E. Courmes, P. Fauran, and J-P. Lespinasse, with help from the medical staff of the Marine troops at the Camp.¹³ The investigators observed that 62/3,553 (1.7%) conscripts were microfilaria-positive.¹³ The conscripts came from all over the archipelago; those positive for microfilariae were mainly concentrated in Pointe-à-Pitre, Grande-Terre with 11/439 (2.5%); in Saint Rose, Basse-Terre with 11/107 (10.3%); and in Capesterre on the island of Marie-Galante with 5/48 (10.4%).¹³

In 1969, J.F.B. Edeson and F. Kuhlow visited Guadeloupe on behalf of the World Health Organization.⁴ They reported that the microfilaremia prevalence in Pointe-à-Pitre was 2.5%; the entire island was 1.8%.⁴

In summary, lymphatic filariasis was historically reported in the south of Grande-Terre, the north of Basse-Terre, and on the island of Marie

Galante.²⁶ The highest prevalence occurred in Saint Rose in Basse-Terre and Capesterre in Marie Galante.²⁶ Lymphatic filariasis appears to have been absent on the islands of Terre-de-Haut des Saints, Désirade, Saint Barthélemy, and the French part of Saint Martin/Sint Maarten.²⁶ Studies on lymphatic filariasis conducted in Guadeloupe are shown in Table 1.

Table 1. Studies on lymphatic filariasis, Guadeloupe, 1913-1966

Year	Location/population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1913	Civil servants at the Military Hospital, Point-à-Pitre	12	4	33.3	L. Stévenel, 1913 ¹⁴
1914	Guadeloupean conscripts in Marseilles, France	150	23	15.3	M. Léger and R. Le Gallen 1914 ¹⁷
1935	Laboratoire de Bactériologie at Pointe-à-Pitre	110	20	18.2	M. Advier, 1937 ¹⁸ ; E. Courmes et al, 1968 ¹³
1936	Laboratoire de Bactériologie at Pointe-à-Pitre	153	15	9.8	Jolly, 1938 ¹⁹ ; E. Courmes et al, 1968 ¹³
1938	Laboratoire de Bactériologie at Pointe-à-Pitre	249	12	4.8	Jolly, 1938 ¹⁹ ; E. Courmes et al, 1968 ¹³
1944-1945	Guadeloupean conscripts in French Guiana	33	4	12.1	H. Floch, 1946 ²⁰ ; H. Floch and P. de Lajudie, 1947 ²¹
1949	Inmates at the leprosarium on the island of Désirade	99	0	0.0	J. Sautet, 1949 ²² ; E. Courmes et al, 1968 ¹³
1950	Inmates at the leprosarium on the island of Désirade	50	0	0.0	J. Languillon, 1950 ²² ; E. Courmes et al, 1968 ¹³
1949	Conscripts from all nine islands	110	15	13.6	E. Montestruc, R. Blache and R. Laborde, 1950 ²⁵
1966	Conscripts selected from the Dugommier Camp near Pointe-à-Pitre	3,553	62	1.8	E. Courmes, P. Fauran and J-P Lespinasse, 1968 ¹³

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THE ORIGINAL INHABITANTS of the island were the Arawaks,^{1,2} followed by the Caribs, who called the island "*Madinina*," meaning "island of flowers."^{1,2} When Christopher Columbus came upon the island, he named it "*Martinica*" in honor of Saint Martin.^{1,2} The Caribs prevented Spanish settlement.^{1,2}

In 1635, the French settled on the island under the leadership of Pierre Belain d'Esnambuc.^{1,2} Soon after, cultivation of sugarcane and the importation of slaves commenced.¹ Slavery persisted until emancipation in 1848.¹ Fierce battles continued between the Caribs and the French until 1660, when a treaty was signed under which the Caribs agreed to occupy only the Atlantic side of the island.^{1,2} However, the peace was short-lived, and the Caribs were soon completely extirpated.^{1,2}

During the 17th and 18th centuries, the British and French fought over their colonial possessions.^{1,2} In 1762, the British occupied Martinique, only to return it to the French in exchange for other territories.^{1,2} France was content to retain Martinique and Guadeloupe because of the importance of the sugarcane trade at the time.^{1,2} More unrest followed in the French Caribbean colonies when, in 1789, slaves encouraged by the French Revolution fought for their emancipation.^{1,2}

Martinique was again occupied by the English from 1794 to 1802, at the request of the plantation owners of the island, who wanted to preserve the status quo and avoid slave revolts.^{1,2} In the latter half of the 19th century, tens of thousands of immigrant workers from India came to Martinique to replace the slave workforce on the plantations after emancipation in 1848.^{1,2} In 1946, Martinique became a French Department, and in 1974, a Region of France.^{1,2}

The earliest record of lymphatic filariasis was reported in 1907, when W. Dufougeré examined 53 patients with elephantiasis and found that 11 (20.8%) were microfilaria-positive.³⁻⁵

On January 10, 1909, P.L. Simond, F.E. Noc, and P. Aubert saw a patient who had chyluric ascites; he was microfilaria-positive in blood and ascitic fluid.⁶ The patient had lived in Martinique for many years before emigrating to France.⁶ A few weeks later, on February 18, 1909, Simond, Noc, and Aubert saw another patient with chyluria; microfilariae were

detected in a sample of urine.⁶ He too was originally from Martinique before becoming a naval carpenter.⁶

In 1913, F.E. Noc and L. Stévenel reviewed 4,000 records of patients who had attended the dispensary of the Institute of Hygiene and Microbiology in Fort-de-France from 1909 to 1913.⁷ They observed that 12 (0.3%) patients were positive for microfilariae; 11 had microfilariae in the urine, five had microfilaremia, and one had microfilariae in fluid aspirated from vesicles in the labia.⁷ In addition, 88 (2.2%) of these patients had clinical manifestations of either lymphangitis (73), elephantiasis (10) or both (5).⁷ The researchers also examined 73 apparently healthy persons, four (5.5%) of whom were found to be microfilaria-positive.⁷

In 1924, a patient with a diagnosis of left inguinal hernia was found to be microfilaria-positive during routine blood examination.⁸ In 1932, another patient was admitted to the Urological Service in Val-de-Grâce, for a suspected hydrocele.⁹ During surgery, this proved to be a chylocele and microfilariae were found in the fluid.⁹

In 1935, E. Montestruc and Ch. Bertrand collected samples of blood from 108 healthy military personnel and 10 persons with lymphangitis.¹⁰ Of these 118 persons, two (1.7%) were microfilaria-positive.¹⁰ In 1936, Montestruc and Lapeyre observed that four (1.8%) of 228 persons from Fort-de-France, Schoelcher, and Case-Pilote, all located on the east-central Caribbean coast, were microfilaria-positive.¹¹ In total, between 1935 and 1936, Montestruc, Bertrand, and Lapeyre had detected microfilariae in the blood of six (1.8%) of 336 apparently healthy persons from different parts of Martinique.¹¹

On October 31, 1941, a seaman originally from Fort-de-France was examined at the U.S. Marine Hospital in Staten Island, New York.¹² Histologic sections of lymphatic vessels of his left spermatic cord were found to contain sections of gravid female adult filarial worms.¹² In 1944 and 1945, H. Floch conducted an investigation in French Guiana which included 70 persons from Martinique.^{13,14} On average, the participants from Martinique had lived in French Guiana for 3 months; eight (11.4%) were found to be microfilaria-positive.^{13,14} In 1949, Montestruc, R. Blanche, and R. Laborde conducted another survey in

Martinique and observed that 10/105 (9.5%) persons had microfilaremia.¹⁵

An extensive survey was conducted from July 1960 to May 1961, which included participants from the military, from hospitals in various communities across the island, and from metropolitan areas.^{16,17} The survey included eight regions: the northeast; along the Atlantic coast; along the north Caribbean coast; the central zone; the south coast; Fort-de-France; Le François; and the south Atlantic coast.^{16,17} Of 2,516 persons from all eight regions, 243 (9.7%) were microfilaria-positive.^{16,17} The highest microfilaremia prevalence, 18.4%, was observed in Le François,

where many sugarcane plantations were located, followed by 14.6% in Fort-de-France on the Caribbean coast.^{16,17} The lowest prevalence, 3.6%, was found in towns of the northeast region.^{16,17}

In Le François, *Culex fatigans* mosquitoes were collected from two houses in which at least one occupant was microfilaremic.^{16,17} Nine (31.0%) of 29 mosquitoes were infected with filarial larvae.^{16,17}

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Martinique.¹⁸ Table 1 summarizes the studies on lymphatic filariasis in Martinique.

Table 1. Studies on lymphatic filariasis in Martinique, 1907-1961

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1907	Patients with elephantiasis	53	11	20.8	W. Dufougeré, 1907 ³ ; Dufougeré, 1908 ⁴ ; M. Léger and R. Le Gallen, 1914 ⁵
1909-1913	Patients at the Institute of Hygiene and Microbiology, Fort-de-France	4,000	12	0.3	F. E. Noc and L. Stévenel, 1913 ⁷
1913	Apparently healthy persons	73	4	5.5	F. E. Noc and L. Stévenel, 1913 ⁷
1935	Healthy persons and patients with lymphangitis	118	2	1.7	E. Montestruc and Ch. Bertrand, 1935 ¹⁰
1936	Residents from Fort-de-France, Schoelcher and Case-Pilote	228	4	1.8	M. E. Montestruc, 1948 ¹¹
1944-1945	Conscripts from Martinique in French Guiana	70	8	11.4	H. Floch, 1946 ¹³ ; H. Floch and P. de Lajudie, 1947 ¹⁴
1949	Soldiers from throughout the island	105	10	9.5	E. Montestruc, R. Blache and R. Laborde, 1950 ¹⁵
1960-1961	Military hospital patients, communities spread across the island	2,516	243	9.7	R. Mille, F. Papa and M. Delloue, 1961 ¹⁶ ; R. Mille, F. Papa and M. Delloue, 1962 ¹⁷

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THE CARIBS INHABITED Montserrat at the time of the arrival of Christopher Columbus and called the island “*Alliouagana*,” which means “land of the prickly bush.”¹ Christopher Columbus came upon the island during his second voyage, on November 11, 1493.^{1,2} He named it after a monastery in Spain, “*Santa Maria de Montserrate*,” located in an area of similar mountainous terrain.² In Spain, this was the abbey where the founder of the Jesuits, Ignacio de Loyola, experienced the vision that led to the forming of his famous order of monks.¹ Although the Spanish laid claim to the island, settlements were never established until the arrival of the English.^{1,2}

In 1632, Thomas Warner brought English settlers and Irish Catholics from Saint Kitts to establish their own colony and to escape religious persecution.^{1,2} The island became a refuge for Catholics fleeing persecution in Virginia and for Irish prisoners sent by Cromwell in 1649, following his victory at Drogheda.¹ To develop the island economy, plantations were established and slaves were introduced in 1651, but the rugged terrain limited the success of the plantations.^{1,2} Slaves quickly outnumbered the rest of the populace, and on Saint Patrick’s Day in 1768, they revolted.¹ The revolt was

quickly suppressed, but the slaves were finally emancipated on August 1, 1834.^{1,3}

The French invaded Montserrat several times during the 17th and 18th centuries with the help of the Caribs² and the resident Irish, but the island was returned to British rule in 1783, under the “Treaty of Versailles”¹ and has remained a self-governing colony.^{1,2}

The earliest available reference to lymphatic filariasis in Montserrat was 1897, when Sir Patrick Manson found microfilariae in six (21.4%) of 28 blood films obtained from Saint Kitts and Montserrat.⁴ In an early review of the distribution of tropical and other diseases in Montserrat, E.B. McKinley reported that cases of lymphatic filariasis had been observed.⁵

In 1945, an employee at a naval base in Trinidad, who was from Montserrat, was found to be microfilaria-positive.^{6,7} Annual reports of the Medical Department from 1951 to 1953 stated that lymphatic filariasis was present.⁸⁻¹³ In 1958, four cases of lymphatic filariasis were listed in official national reports.¹⁴

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Montserrat.¹⁵

Table 1. Studies on lymphatic filariasis in Montserrat, 1897-1945

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1897	Blood films from Saint Kitts and Montserrat	28	6	21.4	P. Manson, 1897 ⁴
1945	Employees at a naval base in Trinidad	—	1	—	R. F. Platzer and W. K. A. Lawlor, 1945 ⁶

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THE EARLIEST INHABITANTS of Panama were the Cuevas and the Coclé cultures.¹ These groups were decimated by disease and by conflicts with the Spanish, who arrived in the 16th century.¹ In 1510, after several forays along the Panamanian Caribbean shore, the Spanish established Nombre de Dios, a settlement at the mouth of the Río Chagres on the Caribbean coast.¹ The Panamanian Pacific coast later became the route into Peru, and the wealth generated by these incursions was carried overland from the Pacific port of Panama (City) to Nombre de Dios, on the Caribbean Sea.¹

Panama became a province of Colombia when the latter gained its independence in 1821.¹ In 1846, Colombia signed a treaty permitting the United States to construct a railway across the isthmus and to defend it with military force.¹ The idea of a canal across the isthmus had been broached even in the 16th century, but an attempt by the French to build one in 1880 resulted in the death of 22,000 workers from malaria or yellow fever.¹ A Frenchman, Ferdinand de Lesseps, who stood to gain handsomely from a United States buyout of the French rights to build a canal was named "envoy extraordinary" by Washington, D.C.¹ He negotiated and signed a canal treaty with the United States, despite the objections of the Colombian government.¹ The financial and strategic interests of the United States momentarily coincided with the sentiments of Panamanian revolutionaries, and a revolutionary group declared Panama independent on November 3, 1903, with the overt support of the United States.¹⁻³ The United States began the reconstruction of the canal in 1904, and 10 years later, the first ship negotiated the engineering marvel.¹

Blood samples from workers at various hospitals in the Canal Zone were collected during the daytime and examined for infectious agents, especially malaria.⁴ These daytime examinations yielded only sporadic cases of microfilaremia and may account for the apparently low microfilaremia prevalence of *Wuchereria bancrofti* in Panama at that time.⁴

Records obtained from 1905 to 1913 show that of 144,423 blood samples drawn at various hospitals in the Canal Zone, 111 (0.08%) were positive for microfilariae.⁴ These cases were distributed

throughout the Zone: some were recorded in Ancón, Colón, Miraflores, and Santo Tomas, that is, on the Pacific as well as the Atlantic coast.⁴ Table 1 lists the number of cases of *W. bancrofti* microfilaremia by year.

Table 1. *W. bancrofti* microfilaremia by year from hospital records, 1905-1913⁴

Year	No. of persons microfilaria-positive
1905	2
1906	1
1907	3
1908	3
1909	9
1910	29
1911	44
1912	13
1913	7

In 1909, A.B. Herrick stated that "there have been a few cases in which filaria was found accidentally and in which at that time there were no manifestations of pathological processes arising from filaria."³ He reported that at least 20 patients with varying manifestations of lymphatic filariasis had been seen at the Ancón Hospital on Taboga island.³ There were 14 cases of elephantiasis, some involving both legs of the patient, as well as the labia or scrotum; three cases of hydrocele containing milky fluid (with microfilariae present in the fluid of one); two cases of chyluria with microfilariae present in the blood of one; and one case of adenitis, which was treated surgically.³ So, 2/20 (10.0%) patients were microfilaria-positive.³ The patient with elephantiasis of the labia was an 18-year-old Panamanian, who had always lived on Taboga island (off the Panamanian coast).³ The other patient with a genital manifestation was a Jamaican farmer from Rio Grande, who had lived in the Isthmus (Panama) for 22 years and had scrotal elephantiasis.³ In the same study, Herrick also examined a Trinidadian, who had lived in Colón for 22 years, was microfilaria-positive, and had chyluria.³ Another patient from Colombia also had chyluria.³

In 1913, elephantiasis was reported as "relatively common" by Clarac, Leboeuf, and Rigollet.^{4,5} Monthly medical reports of the

Department of Sanitation of the Isthmian Canal Commission provided evidence of occasional cases of elephantiasis, lymph scrotum, and chyluria.⁴ A number of cases of lymphatic filariasis were also recorded in hospital charts.⁴ These cases occurred not in the fixed population of this country, but rather in the heterogeneous group of canal employees who congregated in the Canal Zone during the period of active canal construction (from 1881-1914),^{1,2} many of whom came from islands in the West Indies.⁴

In 1914, two patients with lymphatic filariasis were admitted to hospitals in the Zone, but there is no mention of any laboratory-confirmed cases.⁴ In 1918, the staff of the Board of Health Laboratory in the Zone examined several hundred thick blood smears

for malarial parasites, and took the opportunity to conduct a lymphatic filariasis survey.⁶ They observed that 12/504 (2.4%) brush cutters and 21/566 (3.8%) patients from Ancón and Santo Tomas hospitals were positive for microfilariae.⁶

In 1945, of 33,970 employees examined at a naval base in Trinidad, the blood smear of the only employee from Panama was found to be microfilaria-positive.^{7,8} In 1965, reports state that Ameridians from Costa Rica, who migrated south to Boca del Toro in northern Panama, were microfilaria-positive.⁹

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Panama.⁹ Table 2 lists the studies on lymphatic filariasis conducted in Panama.

Table 2. Studies on lymphatic filariasis in Panama, 1905-1945

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1905-1913	Day-time blood samples from various hospitals in the Canal Zone	144,423	111	0.1	A. J. Smith, 1917 ⁴
1918	Brush cutters from the constantly shifting population in the Panama Canal Zone	504	12	2.4	W. L. McFarland, 1922 ⁶
1918	Patients from Ancón and Saint Thomas hospitals	556	21	3.8	W. L. McFarland, 1922 ⁶
1945	Panamanian employees at a naval base in Trinidad	1	1	100.0	R. F. Platzer and W. K. A. Lawlor, 1945 ⁷

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KNOWN AS THE ISLAND “*Boriken*” or “*Borinquen*,” which means “the great land of the valiant and noble Lord,” Puerto Rico was initially inhabited by the Taino Indians, who met Columbus during his second voyage, on November 19, 1493.^{1,2} In 1505, the Spanish *conquistadores* established settlements.⁴ They called the island “*San Juan*” for “Saint John the Baptist” and the newly established town, “*Puerto Rico*.”² In 1508, Juan Ponce de León, a lieutenant to Columbus, arrived from Hispaniola with more settlers and switched the two names, so that the town became San Juan and the island, Puerto Rico.^{1,3}

In 1509, Juan Ponce de León became the first governor of the island and spent 3 years trying to subdue revolts by the Tainos.¹ On January 27, 1513 slaves were introduced onto the island.^{1,2,4} Slavery continued until emancipation in 1873.^{1,2,4} It is believed that lymphatic filariasis was introduced to Puerto Rico through the slave trade.^{4,8} In 1523, the first sugarcane processing plant was built and by 1530, sugar had become the most important agricultural product of the island.² In 1542, coconut trees were introduced to the island and by 1570, the gold mines in operation at the time of Columbus were declared depleted.²

The island was attacked by the British in 1595, 1598, 1702, and 1797 and by the Dutch in 1625, but still it remained under Spanish rule until 1898.² On August 8, 1898, the Spanish-American War officially ended, which resulted in the United States acquisition of Spanish territories in the Western Pacific and Latin America.² On September 29, 1898, the island was officially ceded to the United States.² In 1903, Puerto Rico became a territory of the United States and on July 25, 1952, it was proclaimed a Commonwealth of the United States.²

The earliest reports of elephantiasis are from 1866, when Calixto Romero y Togores first described it on the island.^{4,8-11} He elaborated on the frequency of acute attacks preceding enlargement of limbs and on their final sequela – elephantiasis.^{4,9-11}

In 1875, E. Dumont reported on the clinical manifestations of lymphatic filariasis in more detail.^{4,5,8,11-13} He described three kinds of “elephantoid tumors” of the scrotum and observed the clinical signs of the so-called filarial lymphangitis: focal pain, chills, fever, appearance of red streaks and

sweating of the affected body parts.^{4,12} Together with Betances and Andinot, he performed surgery in an effort to treat elephantiasis.^{4,12}

In 1893, in Caguas, east-central Puerto Rico, Jiménez Cruz was said to have been the first on the island to confirm a diagnosis of lymphatic filariasis, by finding circulating microfilariae in blood.¹⁴ However, in 1890, a Manuel Figueroa reportedly observed the parasite both in the blood and urine of a patient with hematochyluria at Arecibo, in northwest Puerto Rico.⁴

From 1902 to 1903, while stationed at Ponce, in the south-central part of the island near the southern seaboard, Bailey K. Ashford found many cases of filariasis in the wards of Tricoche Hospital and in consultation with other physicians.¹⁴ He believed that about 10% of people in Ponce were infected with *Wuchereria bancrofti*.¹⁴

In 1903, when Ashford was transferred to Cayey, northeast of Ponce, he observed that 30/250 (12.0%) soldiers of the Puerto Rico Provisional Infantry Regiment of the United States Army had microfilaremia.¹⁴ In addition, Yabucoa, a coastal town on the southeastern seaboard, was noted to have persons with elephantiasis; this was also the case in Humacao, another coastal town on the southeastern seaboard, north of Yabucoa.¹⁴

The soldiers in Ashford’s study had a variety of signs and symptoms, including painful inguinal adenopathy and adenitis, fever, chills, sweats, lymphangitis and chyluria.¹⁴ The 30 infected soldiers came from different towns spread across the island, but 25 (83.3%) had lived in their respective communities for more than 10 years.¹⁴ Most patients had lived in Ponce (western south-central region), Aibonito (eastern central region), Humacao (southeast region) and San Juan (eastern north-central region).¹⁴

Several Puerto Ricans were diagnosed with lymphatic filariasis in the United States at the turn of the 20th century. In 1911, two students at the University of Pennsylvania in Philadelphia were found to be microfilaria-positive.¹⁵ In 1912, a third student working as a hospital intern in Philadelphia was found to be microfilaria-positive.¹⁵ In March 1913, another medical student at the Baltimore Medical College had microfilaremia.¹⁶ In November 1913, a 28-year-old man examined in Philadelphia for

chills, fever, bloody and milky urine, and an enlarged scrotum was found to be positive for microfilariae.¹⁷ On January 28, 1914, at the Quarantine Hospital in San Juan, a 10-year-old boy was reported to have died as a result of septicemia and acute filarial lymphangitis; initially he was suspected of having bubonic plague.¹⁸

In an investigation of the Institute of Tropical Medicine and Hygiene of Puerto Rico, 400 persons in Utuado, a mountain town, were examined from 1913 to 1914.^{14,19} Five (0.05%) had clinical manifestations of lymphatic filariasis and none were positive for microfilariae.^{14,19} In 1916, J. Bird examined four families in Carolina, and found that at least one person in each family had microfilaremia.²⁰ From 1914 to 1915, in Puerta de Tierra, a municipality of San Juan, out of approximately 6,250 persons examined by A. Martinez Alvarez, 50 (0.8%) had clinical signs of lymphatic filariasis.^{21,22}

In 1927, on arrival in Puerto Rico, F.W. O'Connor was informed by W.A. Hoffman of the high prevalence of microfilaremia in boys at a specific school.²³ Together, both men examined 45 *Culex fatigans* mosquitoes collected from the dormitory at the boys' school; four (8.9%) were infected with larvae of *W. bancrofti*.²³ In the same, F.W. O'Connor and colleagues reported an additional 60 cases of lymphatic filariasis.²³

Alice M.B. Burke conducted a survey to determine the prevalence of filarial infection and disease in families of filariasis patients.²⁴ The survey

took place in Aguadilla (84 persons); Puerta de Tierra, a barrio of San Juan (115 persons); and other locations on the island (49 persons).²⁴ In 18 (54.6%) of 33 families, one member had lymphedema of the leg and a second member showed some other evidence of lymphatic filariasis.²⁴ Of 248 family members, 20 (8.1%) had microfilaremia, while 59 (23.8%) had lymphedema of the leg.²⁴

Beginning in May 1927, Hoffman and colleagues surveyed 32 localities (26 towns / municipalities and six institutions).²⁵ They found 197/3,857 (5.1%) persons to be microfilaria-positive (Tables 1 and 2).²⁵ The institutions included the Boys' Charity School, the Girls' Charity School, the George O. Robinson Orphanage for Girls, and the Blanche Kellogg Institute for Girls, all in Santurce; the Boys' Reform School near Mayagüez; and the Polytechnic Institute at San Germán.^{13,25} Santurce, Mayagüez, and San Germán are located in the northeastern, western, and southwestern parts of Puerto Rico, respectively.²⁵

The communities with the highest microfilaremia prevalence were: Puerta de Tierra (San Juan) 13.1%; Boys' Charity School, Santurce 10.0%; Hato Tejo (Bayamón) 9.8%; Boys' Reform School, Mayagüez 9.2%, and the town of Mayagüez 9.2% (Table 2).^{13,25} Communities with no reported microfilaremia were Lares, Guánica, Yauco, Utuado, and Trujillo Alto, while the rest had prevalences ranging from 0.9% in Ensenada to 8.3% in San Sebastián.^{13,25}

Table 1. Microfilaremia prevalence in six institutions for children, 1927, Hoffman et al.^{13, 25}

Place	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Boys' Charity School, Santurce	279	28	10.0
Girls' Charity School, Santurce	313	12	3.8
George O. Robinson Orphanage for Girls in the Condado District of San Juan	50	2	4.0
Blanche Kellogg Institute for Girls, Santurce	63	5	7.9
Polytechnic Institute, San Germán	114	2	1.8
Boys' Reform School, Mayagüez	120	11	9.2
Total	939	60	6.4

Table 2. Microfilaremia prevalence in 26 municipalities by Hoffman et al., 1927²⁵ and Hoffman et al., 1932¹³

Region	Town/ Place	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Towns located in the North from west to east	Presbyterian Hospital, Condado District of San Juan	285	8	2.8
	Aguadilla	400	30	7.5
	San Sebastián	120	10	8.3
	Manatí	57	2	3.5
	Hatos Tejas (Bayamón)	112	11	9.8
	Puerta de Tierra (San Juan)	107	14	13.1
	Trujillo Alto	108	0	0.0
	Loíza Nueva (Canóvanas)	49	3	6.1
	Mayagüez	153	14	9.2
East	Fajardo	119	7	5.9
Towns in the Interior (central region) from west to east	Lares	120	0	0.0
	Utua	102	0	0.0
	Aibonito	101	3	3.0
	Comerio	150	2	1.3
	Cidra	148	6	4.1
	Agua Buenas	75	4	5.3
	Cayey	82	3	3.7
	Caguas	249	13	5.2
	San Lorenzo	98	6	6.1
Towns in the South, from west to east	Ensenada	108	1	0.9
	Guánica	38	0	0.0
	Yauco	43	0	0.0
	Ponce	34	2	5.9
	Central Mercedita	18	1	5.6
	Coamo	162	4	2.5
	Central Aguirre	108	4	3.7
	Total	3,146	148	4.8

In the latter half of 1928, in collaboration with officials from the School of Tropical Medicine, the Presbyterian Hospital in San Juan, and various municipal hospitals, O'Connor, G.R. Burke and Enrique Koppisch examined 57 persons who had lymphangitis and/or elephantiasis.²⁶ Of these 57 persons, two (3.5%) had microfilaremia, while 33 (57.9%) had both elephantiasis and a history of lymphangitis.²⁶ Of the participants involved in the study, 50 were residents of Aguadilla, two were from Arecibo, and one person each was from Santurce, Fajardo, Pueblo Nuevo, Callegón de Fuerte, and Río Piedras, located in north-central Puerto Rico, southeast of San Juan.²⁶

On April 6, 1929, an autopsy was performed on a middle-aged Puerto Rican man.²⁷ Histological

sections of his spermatic cord revealed adult male *W. bancrofti*, and adult female worms with uteri filled with embryos.²⁷

In September 1929, a 3-year report of the Pathology Department of the School of Tropical Medicine indicated that microfilariae were detected in three (1.3%) of 225 autopsies performed at 20 different institutions in nine towns.²⁸ That same month, at the Presbyterian Hospital in San Juan, 32 persons with recent attacks of lymphangitis were treated with sulpharsphenamine dissolved in novocaine, which was subcutaneously administered by Jenaro Suárez, G.R. Burke, and F.W. O'Connor.²⁹ This treatment reportedly provided temporary relief.²⁹

In 1930, 60 persons with acute lymphangitis were seen and examined by Suárez at the Presbyterian

Hospital in San Juan, where he was investigating the possible bacteriological etiology of this syndrome.³⁰ He examined 23 males and 37 females; all but one, a 2-year-old boy, had lymphedema or elephantiasis, and none were positive for microfilariae.³⁰

In 1931, William R. Torgerson, together with colleagues from the University Hospital of the School of Tropical Medicine at the University of Puerto Rico in San Juan, performed the Auchincloss operation for treatment of elephantiasis on five patients.³¹ The surgery reportedly provided temporary improvement.³¹

In 1931, another survey was conducted in Santurce with 518 participants from the Boys' Charity School and the Blind School; volunteers from the outpatient municipal dispensary; and patients from the general wards of the Presbyterian Hospital and the University Hospital of the School of Tropical Medicine.⁵ Of these participants, 40 (7.7%) had microfilaremia and 26 (5.0%) had clinical manifestations of lymphatic filariasis.⁵ Mosquitoes were collected from the grounds of the Boys' Charity School and the School for the Blind, and from houses around both schools.⁵ Of 543 female *Culex quinquefasciatus* mosquitoes collected, 31 (5.7%) were infected with larvae of *W. bancrofti*.⁵

In 1932, a complete report of the earlier study conducted by Hoffman et al., from May 1927 until

October 1928, was published.¹³ In addition to the 32 communities previously mentioned, seven more had been surveyed: Vieques, Yabucoa, Humacao, Naguabo, Ceiba, Maricao, and Añasco (Table 3).¹³ Among the newly surveyed towns, the highest microfilaremia prevalences were found in Naguabo 14.0%, Ceiba 12.5%, and Humacao 8.8%.¹³

Of 4,968 persons from 39 communities, 273 (5.5%) had microfilaremia^{13,25}; 208 (4.2%) persons had clinical manifestations of lymphatic filariasis.¹³ The highest prevalences of clinical manifestations were observed in Puerta de Tierra (21.5%), Ponce (14.7%), Aguadilla (10.8%), and Caguas (10.4%).¹³

In February 1932, 18/480 (3.8%) soldiers of the 65th Infantry Regiment stationed at San Juan were found to be microfilaria-positive during a study to determine the effectiveness of gentian violet as a treatment for *W. bancrofti* infection.³²

In 1935, O'Connor, C.R. Hulse and coworkers reported the results of three surveys conducted between 1929 and 1931.⁴ Seventy-seven communities from across the island were studied.⁴ Of 2,098 persons, 165 (7.9%) had microfilaremia; 434 (20.7%) persons were amicrofilaremic, but had clinical manifestations of lymphatic filariasis.⁴ Seven (9.1%) of the 77 communities had microfilaremia prevalences above 9.0% (Table 4).⁴

Table 3. Microfilaremia prevalences in seven communities surveyed in 1927, Hoffman et al., 1932¹³

Region	Town	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Vieques, an island off the eastern coast of Puerto Rico	The whole island	254	16	6.3
West	Maricao	111	1	0.9
	Añasco	151	6	4.0
	Ceiba	24	3	12.5
	Naguabo	179	25	14.0
East	Humacao	148	13	8.8
	Yabucoa	16	1	6.3
	Total	883	65	7.4

Table 4. Seven of 77 communities with microfilaremia prevalences of more than 9.0%, Puerto Rico, 1929-1931⁴

Location	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Aguada	199	27	14.1
Aguadilla	209	29	13.9
Puerta de Tierra (San Juan)	158	18	11.4
San Juan	117	13	11.1
Santurce	83	8	9.6
Bayamón	129	12	9.3
Vega Alta	110	10	9.1

In the summer of 1937, 544 soldiers stationed at San Juan and 250 at Cayey, both of the 65th Infantry Battalion, were examined for microfilaremia by staff of the School of Tropical Medicine at the Station Hospital in San Juan.³³ Of 794 soldiers, 15 (1.9%) were microfilaria-positive, all but one from the Cayey Station.³³

From 1943 to 1944, in collaboration with the medical staff of the Military and the 161st General Hospital Laboratory, Z.T. Bercovitz and Harry Shwachman examined for selective service 16,439 male inductees between the ages of 18 and 38 years.³⁴ They came from 77 communities (including the island of Vieques) spread across the island; 563 (3.4%) were microfilaria-positive.³⁴ In addition, Shwachman examined 217 randomly selected young Puerto Ricans for testing.³⁴ Of these, two (0.9%) had microfilaremia,

using a concentration method, while none were detected using the thick blood smears.³⁴

In September 1946, F. Hernández Morales and colleagues conducted a survey to determine the microfilaremia prevalence in patients with attacks of lymphangitis and their relatives at the University Hospital in San Juan.³⁵ They found that 8/58 (13.8%) patients and 6/138 (4.4%) relatives had microfilaremia.³⁵

These investigators conducted another survey to determine the prevalence of lymphatic filariasis in inmates at the Insular Penitentiary for Men at Río Piedras.³⁶ Of 1,256 men examined, 57 (4.5%) were microfilaria-positive.³⁶ The inmates originally came from more 30 communities, scattered across the island (Table 5).³⁶

Table 5. Towns of origin of microfilaria-positive inmates, Hernández Morales, 1946³⁶

Town	No. of persons microfilaria-positive	Town	No. of persons microfilaria-positive
Aguas Buenas	1	Gurabo	3
Aibonito	1	Juncos	2
Arecibo	1	Lares	1
Barceloneta	1	Manatí	2
Barranquitas	1	Maricao	1
Bayamón	1	Mayagüez	3
Caguas	4	Naguabo	1
Canóvanas	1	Ponce	3
Carolina	1	Puerta de Tierra	1
Cataño	1	Río Piedras	1
Cayey	5	San Juan	2
Ceiba	1	San Lorenzo	1
Cidra	1	San Sebastián	1
Fajardo	1	Santurce	7
Guayama	1	Villalba	1
Unknown	5		

In 1947, in San Juan, 26 persons with *W. bancrofti* infection were treated with diethylcarbamazine (DEC).^{37,38} After 15 months, 10 (38.5%) remained microfilaria-positive, although the density of microfilariae in the peripheral blood was much lower than before treatment.³⁹

From August 1947 to February 1948, J.F.

Maldonado, Hernández Morales, I. Fox, and C.J. Thillet conducted surveys of microfilaremia at the Insular Home for Girls, the Home for Blind Children, and the Insular Home for Boys, in Santurce (Table 6).^{40,41}

Table 6. Microfilaremia prevalence in Santurce, Puerto Rico 1947-1948, Maldonado et al.^{40, 41}

Institution	No. of persons examined (children and employees)	No. of persons microfilaria-positive (children and employees)	Microfilaremia prevalence (%)
Insular Home for Boys	297	31	10.4
Insular Home for Girls	210	8	3.8
Home for Blind Children	74	1	1.4
Total	581	40	6.9

Of 529 resident children, 38 (7.2%) were microfilaria-positive, compared with two (3.8%) of 52 employees.^{40,41} In addition, 102/693 (14.7%) mosquitoes collected from the Insular Home for Boys, 4/200 (2.0%) mosquitoes from the Insular Home for the Girls and 2/200 (1.0%) mosquitoes from the Home for Blind Children were found to be infected with *W.*

bancrofti (Table 7).^{40,41} Two (0.3%) of these 1,093 *C. quinquefasciatus* were found to harbor mature larvae.^{40,41}

Table 7. *Wuchereria bancrofti* infection prevalence of *Culex quinquefasciatus* collected in 1947-1948 in Santurce, Puerto Rico by Maldonado et al., 1948^{40, 41}

Institution	No. of mosquitoes collected and dissected	No. of mosquitoes infected with <i>Wuchereria bancrofti</i> larvae	Infection rate (%)
Insular Home for Boys	693	102	14.7
Insular Home for Girls	200	4	2.0
Home for Blind Children	200	2	1.0
Total	1,093	108	9.9

From December 1947 to February 1948, a study was conducted to determine the effects of DEC on human microfilariae carriers, as well as on the mosquito vector.⁴² The participants were children and resident employees at the Insular Home for Boys, who were identified as infected in the 1947-1948 study.⁴² At the beginning of the study, 29/269 (10.8%) boys and 2/28 (7.1%) employees were microfilaria-positive, while 102/693 (14.7%) *C. quinquefasciatus* mosquitoes were infected with filarial larvae.⁴² DEC was administered twice a day for 21 days to 24 (77.4%) of 31 microfilaremic persons.⁴²

Blood examinations were conducted monthly for 12 months.⁴² Most patients became microfilariae-negative.⁴² Remarkable drops in the prevalence of mosquito infection in the area were also observed during these monthly surveys.⁴² This study was an important contribution to the control of lymphatic

filariasis during the early years of DEC.⁸ It not only showed the long-term effects of the drug on microfilaremia, but also illustrated, for the first time, DEC's capacity to reduce infection in vector mosquitoes.⁸

According to published official national reports, beginning in 1949, there were 27 cases of filariasis; in 1950 (5); 1951 (1); 1952 (1); 1953 (1); 1954 (1); and 1955 (1).⁴³ In similar documents from 1956 to 1959, filariasis was reported, but recorded as plus signs (+) rather than as the number of cases.⁴³

In 1957, F. Lichtenberg and R. Medina reviewed data on autopsy and surgical specimens handled by the Department of Pathology of the School of Medicine at the University of San Juan.⁴⁴ These specimens had been obtained from 38 institutions in Puerto Rico and the Virgin Islands during the period of January 1940 to December 1952.⁴⁴ Adult filarial

worms were found in the intrascrotal organs of 19/499 (3.8%) autopsy and 66/463 (14.3%) surgical specimens from males more than 10 years of age.⁴⁴

To determine the prevalence of lymphatic filariasis and the etiology of tropical hydrocele in Puerto Rico, investigators examined 42 hydrocele patients at the San Juan General Hospital between August 1957 and September 1958.⁴⁵ Of these, 27 (64.3%) had histopathological findings suggestive of lymphatic filariasis.⁴⁵ Complement fixation tests for filariasis in both serum and hydrocele fluid from 41 men showed that 21 (51.2%) and 24 (58.5%), respectively, had test results consistent with lymphatic filariasis.⁴⁵ Therefore, through clinical examination, pathological and serological investigations, the authors estimated that 27/42 (64.3%) hydrocele

patients had evidence suggestive of filarial disease.⁴⁵

In addition, autopsies were conducted during the period 1959-1961 on 330 males aged 10 years or older at the time of death at city hospitals and the Medicolegal Service of San Juan.⁶ These autopsies focused primarily on the male genitalia: spermatic cord, testis, and epididymis.⁶ In 79 (23.9%) of these autopsies, adult filarial worms were present.⁶

While these studies indicate that lymphatic filariasis infection and disease still occurred up until 1948, the microfilaremia prevalence and mosquito infection rate appear to have greatly diminished since the turn of the 20th century (Tables 8 and 9).^{6,45,46} By 1962, cases of elephantiasis and severe urogenital lesions were reportedly rare.^{6,45,46}

Table 8. Studies on lymphatic filariasis, Puerto Rico, 1902-1948

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1902-1903	Patients from Tricoche Hospital in Ponce and city residents	—	—	10.0	B. K. Ashford, 1903 ¹⁵
1903	Soldiers in Cayey	250	30	12.0	B. K. Ashford, 1903 ¹⁵
1914-1915	Puerta de Tierra, a municipality of San Juan	6,250	50	0.8	A. Martinez Alvarez, 1916 ²² ; A. Martinez Alvarez, 1920 ²³
1927	Persons from Aguadilla, Puerta de Tierra and various parts of the island	248	20	8.1	A. M. B. Burke, 1928 ²⁵
1927-1928	Persons from 32 communities (Preliminary data)	4,085	208	5.1	W. A. Hoffman et al., 1928 ²⁶
1927-1928	Persons from 39 communities (Complete data)	4,968	273	5.5	W. A. Hoffman et al., 1932 ¹⁴
1928	Patients from the Presbyterian Hospital of San Juan and various municipal hospitals, including Aguadilla	57	2	3.5	F. W. O'Connor, 1929 ²⁷
1929-1931	Persons from 77 communities across the island	2,098	165	7.9	F. W. O'Connor and C. R. Hulse, 1935 ⁴
1931	The Boys' Charity School, the Blind School, and a non-random sample of volunteers from the general population, Santurce	518	40	7.7	M. K. Tampi, 1931 ⁵
1932	Soldiers in San Juan	480	18	3.8	B. K. Ashford and H. McC. Synder, 1933 ³²
1937	Soldiers from San Juan and Cayey	794	15	1.9	A. G. Oliver and J. Oliver, 1938 ³³
1943-1944	Male inductees for military service from 77 communities across the island	16,439	563	3.4	Z. T. Bercovitz and H. Shwachman, 1946 ³⁴

Table 8. (Cont.) Studies on lymphatic filariasis, Puerto Rico, 1902-1948

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1946	Patients with recurrent attacks of lymphangitis in San Juan	58	8	13.8	F. Hernández Morales and J. Oliver González, 1946 ³⁵
1946	Relatives of patients with recurrent attacks of lymphangitis in San Juan	138	6	4.4	F. Hernández Morales and J. Oliver González, 1946 ³⁵
1946	Inmates at the Insular Penitentiary for Men, Río Piedras	1,256	57	4.5	F. Hernández Morales and G. González Barrientos, 1946 ³⁶
1947-1948	Children and employees of the Insular Home for Girls, the Insular Home for Boys and the Home for Blind Children	581	40	6.9	J. F. Maldonado et al., 1948 ^{40,41}

Table 9. Prevalence of *Wuchereria bancrofti* infection in *Culex fatigans* mosquitoes, Puerto Rico, 1927-1948

Year	Collection site	No. of mosquitoes collected and dissected	No. of mosquitoes infected with <i>Wuchereria bancrofti</i> larvae	Infection rate (%)	Author and year of publication
1927	Boys' dormitory at the Boys' Charity School, Santurce	45	4	8.9	F. W. O'Connor, 1927 ²⁴
1931	Boys' Charity School, the Blind School, and houses near both schools, Santurce	543	31	5.7	M. K. Tampi, 1931 ⁵
1947-1948	Insular Home for Girls, the Insular Home for Boys, and the Home for Blind Children, Santurce	1,093	108	9.9	J. F. Maldonado, F. Hernández Morales, I. Fox, and C. J. Thillet, 1948 ⁴⁰

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THE ORIGINAL INHABITANTS of these islands were the pre-ceramic Sibonay, who are believed to have arrived about 10,000 B.C. from Central America.^{1,2} The next group of people to colonize the islands were the Arawaks, who came from the Orinoco River area in modern-day Venezuela.^{1,2} The third group were the Caribs from South America, who called Saint Kitts "*Liamuiga*" which means "fertile land" and Nevis "*Oualie*."^{1,2}

Christopher Columbus came upon both islands during his second voyage, on November 12, 1493, naming Saint Kitts "*San Jorge*" and Nevis "*San Martin*."^{1,2} Due to inaccuracies in the early maps and charts of the Caribbean, successive explorers unknowingly "renamed" San Jorge "*San Christobel*" and San Martin "*Santa Maria de las Nieves*."^{1,2} Although the Spanish laid claim to the islands, settlements were never established.^{1,2}

In 1623, Sir Thomas Warner arrived in San Cristobel claiming it as the first British territory in the West Indies.^{1,2} The following year, he returned with groups of settlers from England to both islands and anglicized their names to Saint Christopher and Nevis.^{1,2} In 1625, the French arrived and settled in Saint Christopher, where together with the English, they massacred the Caribs, after they learned of a plot to rid the island of Europeans.^{1,2} The few Caribs that survived left the island.^{1,2} This event is remembered by a place called "Bloody Point" on the island.^{1,2}

In 1629, the Spanish returned and "evicted" the English and French, who returned soon after,

when Spain was unable to occupy the islands.^{1,2} Indentured labor from the British Isles was initially used to cultivate the fields on both islands, but was soon replaced by slaves.^{1,2} The first records of slaves on the islands appear in the will of James Hewitt, dated August 9, 1649.^{1,2} Slaves were continually introduced until their emancipation on August 1, 1834.^{1,2} The French and the British engaged in intermittent wars over the islands from 1664 until 1783, interchanging ownership.^{1,2} The British eventually prevailed and were awarded ownership of both islands under the provisions of the "Treaty of Versailles."^{1,2} Sometime during the 18th century, the name Saint Christopher was shortened to Saint Kitts and the British continued their rule of both islands until September 19, 1983, when independence was achieved.^{1,2}

There are no records of either elephantiasis or chyluria until the late 1800s. In 1897, Sir Patrick Manson found microfilariae in 6/28 (21.4%) blood films obtained from both Saint Kitts and Montserrat.^{3,4} In 1902, G.C. Low undertook an extensive study of the Leeward Islands, where he observed that 47/143 (32.9%) persons were microfilaria-positive in Saint Kitts.⁵⁻⁸

K.H. Uttley compiled the deaths that were attributed to filariasis in the official annual reports of Saint Kitts for the period 1918-1957 (Table 1).⁹ He observed from such reports that deaths caused by elephantiasis had diminished considerably during the previous 100 years.⁹

Table 1. Reported death rate per 10,000 population attributed to filariasis in Saint Kitts, Uttley, 1959⁹

Year	Death rate	Year	Death rate	Year	Death rate	Year	Death rate
1918	2.8	1928	1.7	1938	0.3	1948	—
1919	2.4	1929	—	1939	0.5	1949	—
1920	—	1930	—	1940	0.3	1950	—
1921	2.1	1931	—	1941	—	1951	0.4
1922	—	1932	—	1942	—	1952	0.0
1923	—	1933	—	1943	1.8	1953	0.0
1924	1.9	1934	—	1944	—	1954	0.0
1925	2.2	1935	—	1945	—	1955	0.0
1926	—	1936	0.8	1946	—	1956	0.0
1927	—	1937	1.0	1947	—	1957	0.0

In September 1929, an outbreak of abscesses and cellulitis occurred, and from February to May 1930, A.W. Grace and F.B. Grace studied the affected patients at the Pogson Hospital, Sandy Point (10 miles from Basseterre).¹⁰ Of 17 patients with abscesses, three (17.7%) were microfilaria-positive, compared with 120 (19.3%) "routine" patients, who were microfilaria-positive.^{10,11} The authors concluded that lymphatic filariasis was highly endemic in Saint Kitts.^{10,11}

According to published official national reports, beginning in 1950, there were 103 cases of filariasis; in 1951(179); 1952 (81); 1953 (76); 1954 (28); 1955 (21); 1956 (99); 1957 (103); and 1958 (16).¹²

To determine the prevalence of protozoan and helminthic infestation among migratory agricultural workers, in November 1958 in Clewiston, Florida, Henry Beye, Charles Brooks, and Elizabeth Guinn collected blood specimens from 470 workers originally from the British West Indies.¹³ Of 10 migratory workers from Saint Kitts, one had microfilaremia.¹³ In 1965, M.T. Ashcroft wrote that according to Lake, microfilaremia prevalence was much lower in Saint Kitts than in previous years.¹⁴

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Saint Kitts.¹⁵

Table 2. Studies on lymphatic filariasis in Saint Kitts and Nevis, 1897-1958

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1897	Blood films from Saint Kitts and Montserrat	28	6	21.4	P. Manson, 1897 ³ ; E. L. Opie, 1901 ⁴
1902	A sample of hospital patients and healthy persons	143	47	32.9	G.C. Low, 1902 ⁵ ; Low, 1908 ⁶ ; Low, 1908 ⁷ ; Low, 1908 ⁸
1930	Patients with abscesses at the Pogson Hospital, Sandy Point	17	3	17.7	A. W. Grace and F. B. Grace, 1931 ¹⁰
1930	'Routine' patients at the Pogson Hospital, Sandy Point	621	120	19.3	A. W. Grace and F. B. Grace, 1931 ¹⁰
1958	Migratory agricultural workers in Clewiston, Florida	10	1	10.0	H. Beye, C. Brooks and E. Guinn, 1961 ¹³

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THE INITIAL INHABITANTS of the island are believed to have been the Arawaks.¹⁻³ They were followed by the Caribs, who called the island “*Iouanalao*,” which may have meant “where the iguana is found.”^{1,2} The name was later changed to “*Hiwanarau*”¹ and then evolved to “*Hewanorra*.”^{1,2}

Christopher Columbus is said to have come upon the island on December 13, 1502 (Saint Lucy’s Day), but neither the true date of “discovery” nor the exact explorer is in fact known, for according to evidence from Columbus’ log, he appeared to have missed the island and was not even in the area on Saint Lucy’s Day.^{1,2} Juan de la Cosa, a lesser-known explorer who had served at one time as a navigator for Columbus, may have reached this island in 1499, although there is also evidence suggesting that de la Cosa may have arrived in 1504.^{2,3} On a map drawn by de la Cosa in 1500, an island called *El Falcon* may have been Saint Lucia.³

Another school of thought states that a group of French seamen who were shipwrecked on the island on December 13, 1502 named the island after the Virgin-Martyr of Syracuse, “*Sainte Alousie*.”¹ Although the French chroniclers du Terte and Pare Labat, who were in the region during the 17th and 18th centuries, respectively, referred to the island as *Sainte Alousie*, a slight variation “*Sainte Alouzie*,” is found in French nautical records of 1624 and 1628.¹ However, a Vatican globe of 1502 marks the island as *Santa Lucía*, suggesting that it was at least claimed by Spain.¹ The Spanish Cedula of 1511 names “*Sancta Lucia*” as a possession of the Spanish Crown, while a Spanish map of 1529 uses “*Saint Luzia*.”¹ Despite these conflicting records about its origins, Saint Lucians have decided to celebrate December 13 as their National Day.¹

In the 1550s, a notorious buccaneer, Francois le Clerc, also known as “*Jambe de Bois*,” “*Wooden Leg*,” or “*Peg-Leg le Clerc*,” is said to have set up a base on Pigeon Island from where he preyed on treasure-laden Spanish galleons.² Circa 1600, a Dutch expedition arrived and established a base at Vieux Fort.²

In 1605, 67 Englishmen on the ship *Olive Branche* en route to Guiana were shipwrecked at Saint

Lucia.²⁻⁴ After a month, the crew, who were reduced to 19, were forced to flee the Caribs by canoe.^{2,3} From 1638 to 1639, the first recorded settlement led by Sir John Warner was established by the English who came from Bermuda and Saint Kitts, but the colonists were killed by the Caribs about 3 years later.^{2,4} In 1642, the King of France, claiming sovereignty over the island, ceded it to the French West Indian Company,²⁻⁴ who in 1650 sold it to M.M. Houel and Du Parquet.⁴

There were repeated attempts by the Caribs to expel the French, and several governors were murdered.⁴ From 1660, the British began to renew their claim to the island and fought for its possession.⁴ Most of the settlers were French, who had already developed a plantation economy based on slave labor.⁴ Slavery continued until emancipation in 1848.^{3,4} The first permanent settlement, Soufriere, was established by the French in 1746.^{2,3} In 1778, the British launched their first major invasion at the “*Battle of Cul de Sac*.”² By 1780, 12 settlements and a large number of sugarcane plantations had been established.²

In all, Saint Lucia changed hands 14 times^{3,4} before it became a British Crown Colony in 1814 through the “*Treaty of Paris*.”²⁻⁴ In 1967, Saint Lucia gained full internal self-government, becoming a State, in voluntary association with Britain.⁴ On February 22, 1979 it gained full independence.⁴

The earliest reference to lymphatic filariasis was made in 1899, by Ortho Galgey, who mentioned in a document that he had examined persons who were microfilaria-positive.⁵ In 1902, George C. Low reported that 27/356 (7.6%) persons, whose blood he examined, were microfilaria-positive.⁶⁻⁹ Of these, five (18.5%) had lymphedema of the leg.⁶⁻⁹

Annual reports from 1955 and 1956 state that filariasis was present, but details are unavailable.¹⁰⁻¹² In 1955, 17 cases of filariasis were published in official reports¹³ and in 1958 and 1959, the presence of filariasis was indicated with a plus sign (+) in official records.¹³

The latest available report, published in 1975, suggests that lymphatic filariasis was present in Saint Lucia.¹⁴

Table 1. Studies on lymphatic filariasis in Saint Lucia, 1902⁶⁻⁹

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1902	General population	356	27	7.6	G.C. Low, 1902 ⁶ ; Low, 1908 ⁷ ; Low, 1908 ⁸ ; Low, 1908 ⁹

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THE ARAWAKS are believed to have been the initial inhabitants of Saint Vincent.¹ The next group to colonize the island were the Caribs, who called it "*Hairoun*."¹ Christopher Columbus came upon the island during his third voyage in 1498, but no settlements were established.^{1,2} In 1675, a passing Dutch ship laden with settlers and their slaves was shipwrecked between Saint Vincent and Bequia.¹ Only the slaves survived; they settled in Saint Vincent and later intermarried with the Caribs.¹ Their descendants still live in Sandy Bay and in a few places in the northwest.¹

The Caribs aggressively prevented European settlement until the 18th century.¹ In 1722, the British attempted to colonize Saint Vincent, but French settlers, who were already living peaceably with the Caribs and were growing tobacco, indigo, cotton, and sugarcane, foiled the attempt.¹ Possession of Saint Vincent and the Grenadines was hotly disputed until 1763, when they were ceded to Britain.^{1,2} They were lost to the French again in 1778, but in 1783, they were regained under the "Treaty of Versailles."^{1,2}

In 1795, the indigenous population, with the aid of Frenchman Victor Hugo, engaged in what has come

to be known as the "Carib Wars" against the British.^{1,2} Within a year the British gained the upper hand and, in 1796, deported more than 5,000 indigenous people to Roatán, off the coast of present day Belize, where their descendants still live today.^{1,2}

In 1969, the island became a British Associated State with complete internal self-government¹ and in 1979, Saint Vincent and the Grenadines gained full independence.¹

The earliest records of lymphatic filariasis appear in 1897, when Sir Patrick Manson found microfilariae in 6 (4.0%) of 152 blood films sent to him from Saint Vincent.³ In 1908, George C. Low visited Saint Vincent, and examined 100 patients, six (6.0%) of who had microfilaremia.⁴⁻⁶ Two of these patients had signs or symptoms of lymphatic filariasis, while four were asymptomatic.⁴⁻⁶ In 1945, 33,970 employees at a naval base in Trinidad were examined.^{7,8} Of those found to be microfilaria-positive, 26 were from Saint Vincent.^{7,8}

In 1958, Low is said to have observed no cases of lymphatic filariasis.⁹ The latest available reports, published in 1975, indicate that lymphatic filariasis was present in Saint Vincent and the Grenadines.¹⁰

Table 1. Studies on lymphatic filariasis in Saint Vincent and the Grenadines, 1897-1945

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1897	Blood films sent from Saint Vincent	152	6	4.0	P. Manson, 1897 ³
1908	General population	100	6	6.0	G.C. Low, 1908 ⁴ ; Low, 1908 ⁵ ; Low, 1908 ⁶
1945	Naval base in Trinidad	—	26	—	R. F. Platzer et al, 1945 ⁷

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THE POWHATANS, who once lived along the present-day Virginia coast, are believed to have been the first inhabitants seen by the English.¹ Around 1586, John White, an English explorer, is said to have led an unsuccessful attempt to establish settlements in this area.¹ On December 20, 1606, three English ships, the *Susan Constant*, the *Godspeed* and the *Discovery*, set sail from England, carrying 104 men and boys.¹ On May 14, 1607, the ships arrived on the present-day Virginia coast, where the Jamestown settlement was established, along the banks of the James River.¹ In late August 1619, the first slaves arrived from the West Indies on a Dutch vessel and were sold to the governor and a merchant in Jamestown.² Slavery officially ended on December 18, 1865 with general emancipation.³

From the early days, Charleston, South Carolina was a major port of entry for slaves in the United States, including slaves from well-established foci of lymphatic filariasis in the Caribbean, particularly Barbados.⁴⁻¹²

The earliest record of elephantiasis appears to have been in 1775, when Bernard Romans published "A Concise Natural History of East and West Florida."⁸ He lived for 20 years in Georgia and Florida and traveled throughout the Carolinas.⁸ In his book, Romans chronicles elephantiasis as "...a loathsome disease [that] appears some times...after severe acute disorders, especially if the patient has been obliged to keep [in] his bed long, likewise after a violent exercise has brought on a surfeit: this is called the Elephantiasis from the swelling of the feet and legs...."^{8,13}

Subsequent reports of elephantiasis originated from other areas along the east coast.⁸ In the 19th and early 20th centuries, cases of elephantiasis were reported as far north as Northampton near Boston, and in Philadelphia, New York, and New Orleans further south.^{14,15}

J.H. Salisbury of New Orleans was thought by some to have been the first to observe microfilariae.¹⁶ In 1868, he identified and described "this little entozoon in three cases only [that were] met in urine."¹⁶ In one patient, a 65-year-old woman from Cleveland, he observed that "her urine was passed milky, with granular cystine, and was

dense and scanty."¹⁶ On examination, her urine contained 10 to 15 ova in a single drop of urine.¹⁶ He stated that the eggs (embryo) contained the animal (microfilariae) in various stages of development.¹⁶ He classified the organism as a *Trichina*, designating it *Trichina cystica*.¹⁶

In June 1875, at New York Hospital, Robert Abbé examined a patient born in Saint Croix, who had "milky urine."^{17,18} Subsequently, he observed "tough mucus gelatinous blood-streaked masses [and] the smaller worm" that were passed on micturition.^{17,18}

In 1884, John Guitéras described microfilariae in detail, when he examined blood samples from four Cubans residing in Key West, Florida.⁴ They appear to have acquired lymphatic filariasis in Cuba, for earlier that year, one of these patients had been seen by Charles (Carlos) Finlay in Havana.⁴

After many futile attempts with J.J. Edwards in 1885 to find an autochthonous case of lymphatic filariasis, Guitéras, on February 13, 1886, made what has become known as the earliest record of an indigenous case of lymphatic filariasis in the United States.^{4,19} In Charleston, South Carolina, Guitéras found three microfilariae in the peripheral blood of a 40-year-old woman who had lived only in Charleston, South Carolina except for 5 years in Augusta, Georgia.⁴

Reports of other cases of lymphatic filariasis soon followed: 13 cases by W.M. Mastin in 1888; 22 by P.G. de Saussure in 1890; two cases by R.M. Slaughter in 1891; two by J.M. Northington in 1914; and one case each reported by R. Matas in 1891, by F.P. Henry in 1896, by T.A. Dunn in 1898, by J.M. Bertolet in 1901, by J.H. Cunningham Jr in 1904, and by E. Andrade in 1905.^{5,8,10,20-31}

Most of these patients had resided in Charleston, South Carolina, although at the time of diagnosis they were living in towns spread across eastern and southeastern United States: New Orleans, Louisiana; Mobile, Alabama; Eastman, Georgia; Richmond and Alexandria, Virginia; Philadelphia, Pennsylvania; Boston, Massachusetts; and Florida.^{8,20-23,25,26,28,31-34} Therefore, at least from 1889, lymphatic filariasis was known to be endemic in Charleston, South Carolina.^{5,6,8,10,29-31,34-46} Transmission of the parasite likely occurred much earlier.³⁰

Cases indigenous to the United States were found along the Atlantic seaboard as far north as Philadelphia, but most cases north of Charleston were considered to have been imported.³⁰ From 1907 to 1919, the annual reports of the Florida State Board of Health record that only two cases of microfilaremia were seen.⁵ Both cases, one in 1911 and the other in 1912, were reported by G.W. Simons, who was then in charge of the State Bacteriological Laboratory at Tampa.⁵ During the period 1911 to 1919, V.H. Bassett, a bacteriologist at the City Department of Health in Savannah, Georgia reported one person with microfilaremia, who was originally from the West Indies.⁵

Imported cases were also seen in the early part of the 20th century. In 1911, two Puerto Rican students at the University of Pennsylvania in Philadelphia and in 1912, a third student working as a hospital intern in Philadelphia, were found to be microfilaria-positive.^{47,48} In March 1913, another Puerto Rican medical student at the Baltimore Medical College was found to be microfilaria-positive.⁴⁹ In November 1913, a 28-year-old man, who was originally from Puerto Rico, was examined in Philadelphia for chills, fever, bloody and milky urine, and an enlarged scrotum.⁵⁰ He was microfilaria-positive.⁵⁰

In 1909, Francis B. Johnson, a pathologist at the Medical College of South Carolina in Charleston,

examined 131 blood slides; 13 (9.9%) contained microfilariae.²⁹ In 1914, Johnson conducted the first large-scale survey (400 persons) in the United States to determine the extent of lymphatic filariasis in Charleston.²⁹ Blood specimens were collected from the wards of Roper Hospital, Charleston, "without regard to the disease for which the patients were admitted."²⁹ The author observed that 77 (19.3%) persons had microfilaremia and one fourth of those infected had signs or symptoms of lymphatic filariasis.²⁹ Johnson concluded that "...filariasis was extremely prevalent in Charleston, S.C....there was no reason for assuming this to be the only place in the South showing such a high percentage of infection."²⁹

In 1915, Edward Francis, of the United States Public Health Service, examined 37 inmates at the Old Folks Home in Charleston and found that 13 (35.1%) were microfilaremic.⁵ From 1915 to July 1918, Francis conducted filarial surveys in nine cities (excluding Charleston) in five southern states: South Carolina, Georgia, Florida, Alabama, and Louisiana.⁵ Of 1,470 persons, nine (0.6%) were found to be microfilaria-positive (Table 1).⁵ Francis observed that the nine persons had "...histories either of having been born and raised in Charleston or having spent a considerable period of time in Charleston, or of having been born in Cuba, which is a well-known filarial focus."⁵

Table 1. Studies on lymphatic filariasis in southern United States, 1915-1918⁵

Dates	Location	Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
1915	Milledgeville, Georgia	Inmates from the State Insane Asylum	100	0	0.0
1915	Savannah, Georgia	Patients at the Georgia Infirmary, Charity Hospital, St. Joseph's Hospital, the United States Marine Hospital, and Abram's Home for the Aged	287	0	0.0
1916	Beaufort, South Carolina	General population, Old Folks Home, City Jail, and children from the Penn School on Saint Helena Island	67	1	1.5
1916	Tampa, Florida	Residents of the Hillsborough County Farm and Hospital, the County Jail, and the general population	115	3	2.6
1916	Jacksonville, Florida	Patients at the Duval County Hospital, Saint Luke's Hospital, and Brewster Hospital, and inmates of the City Prison Farm	200	2	1.0
1918	New Orleans, Louisiana	Residents of the Lafon Homes (on North Tonti and South Robertson streets), the Faith Home, Freedman's Home, the German Protestant Home, the Touro Shakespeare Home, and patients at Charity Hospital	304	0	0.0

Table 1. (Cont.) Studies on lymphatic filariasis in southern United States, 1915-1918⁵

Dates	Location	Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
1918	Mobile, Alabama	Patients at the United States Marine Hospital, the City Hospital, and residents of the County Poorhouse	131	0	0.0
1918	Columbia, South Carolina	Patients at the State Hospital for the Insane, and residents of the County Poor House	221	3	1.4
1915-1918	Georgetown, South Carolina	Inmates on the chain gang, jail, and the general population	45	0	0.0
Total			1,470	9	0.6

Some have attributed the disappearance of filariasis in Charleston to an intensive mosquito control campaign mounted during the early 1920s.^{31,38} However, official publications of that era mention no such campaign except for a one-man oil-spraying operation in 1926 and remarks made by L. Banov about anti-mosquito measures.^{51,52} Others attribute the disappearance of the disease to the construction of the municipal sewerage-water system, which began in the 1890s, originally intended to abate typhoid and other water-borne diseases.¹⁰ Polluted water is the preferred breeding site for the urban vector of lymphatic filariasis, *Culex quinquefasciatus* (*fatigans*).¹⁰ Presumably, other social and economic developments, such as improved housing and reduced immigration from disease-endemic areas, also contributed to the reduction in filariasis.¹⁰ Therefore, Charleston, once regarded as the bedrock of filarial endemicity in the United States, apparently became filaria-free circa 1930.^{8,31,38,39,41,53}

In 1930 and 1933, two cases of lymphatic filariasis, one in Ottawa, Kansas, the other in Washington, D.C., were diagnosed by the presence of microfilaremia.^{54,55} Both patients had never left their places of residence and possible routes of transmission remain unclear.^{54,55}

Although there were no further reports of endemic transmission of lymphatic filariasis in the United States, during World War II, many American servicemen stationed in the South Pacific Islands, especially Samoa, became infected.^{35,38-40,43,56-83} A lower percentage of those stationed at Guadalcanal and Bougainville in the Solomon Islands became infected.^{56,64,66,74,80,81} During World War II, about 38,300 Naval personnel were exposed to *Wuchereria bancrofti*, and a total of 10,421 cases of lymphatic filariasis were diagnosed in servicemen in the Marine Corps and Navy by December 1, 1945.^{79,84,85} The possibility of the establishment of endemic foci on return to the United States was a concern.^{38,56,}

^{66,71,73,75,79,82,86-89} Therefore, many of the returning servicemen with lymphatic filariasis were treated, and symptoms were closely monitored.^{62,64-66,70,72,77,79}

From May 1942 to May 1943, a group of 553 soldiers was stationed in Tongatabu in the South Pacific.⁹⁰ In June 1943, they were replaced by a smaller group of 156 troops.⁹⁰ From February to July 1944, both units were sent to a temperate area of Australia for reconditioning and examination.⁹⁰ A team of medical personnel examined both units to determine the prevalence of filarial infection.⁹⁰ Of 709 troops, one (0.1%) was found to be microfilaremic in blood collected during the day.⁹⁰

From April 1943 to February 1944, a team of Naval medical personnel conducted a study in 35 village campsites in Samoa, where both U.S. troops and local inhabitants resided.⁹¹ Although there was no mention of the microfilaremia prevalence among Samoans or the frequency of occurrence of early signs of lymphatic filariasis among the troops, the team found that 655 (9.9%) of 6,634 *Aedes scutellaris* var. *pseudoscutellaris* mosquitoes were infected with larvae of *W. bancrofti*.⁹¹

A group of 564 soldiers who had been stationed on a *W. bancrofti*-endemic island for 364 days (from June 1943 to June 1944) were all amicrofilaremic but 62 (11.0%) soldiers had clinical manifestations, including hydrocele and lymphangitis of the extremities.⁹²

Nearly 10,000 patients passed through the Marine Barracks at Klamath Falls in Oregon, which was established in October 1943.⁶² The Barracks was opened to servicemen with lymphatic filariasis and malaria in May 1944.⁶⁵ During a period of 17 months, 2,595 Navy personnel were reported to have acquired filarial infection.^{84,85} Other servicemen were sent to various hospitals within the country and to mobile medical units abroad for treatment: 140 patients at the United States Naval Hospital in Oakland, California⁶³; 145 patients at Halloran General Hospital in Staten

Island, New York, and Harmon General Hospital in Longview, Texas^{67,68}; 737 patients at a mobile Naval Hospital in New Caledonia⁷⁴; 35 patients at the South Pacific Forward Area⁷¹; and "several" patients at a base hospital in American Samoa.⁷²

Many servicemen developed signs and symptoms within 3 to 5 months of arrival on the islands.^{35,39,40,57-65,67-72,74-77,79,83,93-96} Several hundred servicemen had recurrent acute signs and symptoms. These included muscle aches; fatigue; fever; lymphadenitis; retrograde (i.e., centrifugal) lymphangitis in the epitrochlear, cervical, axillary, femoral, and inguinal areas; and transient but painful swellings of the spermatic cord and scrotum.^{35,39,57-59,61,62,64,65,67-73,75,76,79,94,96-99}

Microfilariae were rarely observed in the peripheral blood,^{34,35,40,56-58,62,64-68,71,72,79,80,81,98} but adult parasites were identified in biopsy specimens of involved lymph nodes (especially epitrochlear lymph nodes) in a number of patients.^{35,39,40,56-59,63,64,67,68,72,75,76,78,79,80,81,89} No cases of elephantiasis were reported.^{39,56,64,66,74,75,79} The clinical course of the disease was monitored, and new drugs and diagnostic procedures (e.g., a skin test) were tried and evaluated.^{35,39,56,62,65,70,79} Various drug treatments failed to bring lasting or uniform relief, although palliative measures seemed to be temporarily effective in some cases.^{35,39,62,70,72,75}

At the Marine Barracks in Klamath Falls, it was observed that following the removal of servicemen with early signs of filariasis from the disease-endemic areas in the South Pacific, the disease tended to run a self-limited course after one or more clinical reactivations or acute attacks.^{35,56,65,66,71,77,79} The rehabilitation and restoration of the patients at the Marine Barracks was a combined program of military, vocational, educational, and medical training and supervision, especially for those weakened by previously prescribed inactivity.^{56,62,64-66,70,71,79} Here, the servicemen were on active duty and they increased their physical exertion in combination with outdoor recreation.^{56,64,65,77,79} Almost 80 percent were former patients from various Naval hospitals and convalescent centers, and the rest were those evacuated directly from the field.^{56,64,65,77,79,100}

Many of the infected servicemen were concerned that the disease would impair sexual performance, for the soldiers had seen the genital deformities suffered by adult men in the Pacific Islands.^{56,60,64,65,79,101} However, no impairment of sexual function was observed in military

personnel.^{35,39,56,62,64-66,70,72,77,79,101,102}

The threat of spread of transmission in the United States soon appeared improbable because many of the servicemen returned to temperate climates similar to that of Klamath Falls, where meteorologic conditions and climate did not favor transmission.^{38,56,61,62,66,69,72,79} However, some scientists were concerned about large numbers of infected servicemen returning to the South, where *W. bancrofti* transmission had once occurred.⁷⁵

The occurrence of lymphatic filariasis in the American Armed Forces stationed in the South Pacific Area provided a unique opportunity to study the early clinical manifestations of filariasis in otherwise healthy young men.^{79,88,103,104} Accurate knowledge of the incubation period was obtained.⁷⁹ In addition, evidence suggested that *W. bancrofti*, and not bacteria, was the sole cause of early lymphatic filariasis.^{79,105} These findings supported the hypothesis that the signs and symptoms of early filariasis were due in part to the development of immunologic reactivity against *W. bancrofti*, at least in immune-naïve hosts.^{79,105}

Since World War II, there have been reports of at least one questionable autochthonous case and several imported cases of filariasis, in which microfilariae were shown. In September 1960, a woman, who had been born in New York and whose recent travels had not extended outside a 100-mile radius of New York, developed a painless mass in her right groin.^{106,107} Three weeks after onset, enlarged non-tender nodes were excised and on histologic examination, three dead worms were found.^{106,107} They were so degenerated that the investigators were unable to make a positive identification.¹⁰⁶ Although *W. bancrofti* was not identified, it could not be excluded.¹⁰⁶

On September 5, 1963, Samoan and non-Samoan children who resided in Laie, Oahu, Hawaii, were examined in an effort to determine whether the Pacific bancroftian filariasis had been secondarily transmitted in Laie, Oahu.¹⁰⁸ All of the children attended the same school and lived in the same area in Laie, Oahu.¹⁰⁸ The study showed that 3/55 (5.5%) of the Samoan-born children had microfilaremia; none of the Samoan children born in Hawaii and none of the non-Samoan children had microfilaremia.^{108,109}

In 1967, a man from Bombay, India, who lived in New York, had a cutaneous nodule on his right arm examined.¹¹⁰ Excisional biopsy revealed sections of a female filarial nematode.¹¹⁰ Subsequent night blood smears tested positive for microfilariae of

W. bancrofti.¹¹⁰ The patient was subsequently successfully treated with diethylcarbamazine (DEC) at a dose of 100 mg given three times per day for three weeks.¹¹⁰

In 1969, a study was conducted to assess the prevalence of filariasis in Samoan immigrants in Los Angeles, California, and Laie, Hawaii.¹⁰⁹ The authors examined 112 participants at a clinic in Laie, Hawaii, and 62 participants at two clinics in Los Angeles.¹⁰⁹ They also reviewed the cases of four Samoans who were hospitalized in Los Angeles.¹⁰⁹ The investigators observed that 25/178 (14.1%) Samoans had microfilaremia.¹⁰⁹

C. quinquefasciatus mosquitoes from coastal South Carolina; San Diego, California; and Hawaii have been shown in the laboratory to be potential vectors for the sub-periodic Pacific form of bancroftian filariasis.^{73,75,80,81,111} However, this mosquito seems to be an inefficient vector of the Polynesian strain of non-periodic *W. bancrofti*.^{108,109} Thus, the risk of transmission of non-periodic *W. bancrofti* into the continental United States appears to be low.^{108,109} In contrast, *C. quinquefasciatus* and *Culex pipiens* from the northern United States and Oahu, Hawaii have been found under laboratory conditions to be efficient vectors of the Okinawa, Asian, and Caribbean strains of nocturnally periodic *W. bancrofti*.^{108,112} However, there has been no documented transmission of nocturnally periodic *W. bancrofti* in the United States since before World War II.^{108,112}

In 1977, a young girl from India who immigrated to Harrisburg, Pennsylvania, developed right inguinal adenopathy of 2 months' duration.⁴⁴ The lymph nodes were excised and histologic examination revealed several sections of degenerating microfilariae within an adult female *W. bancrofti* worm.⁴⁴ The patient was successfully treated with DEC.⁴⁴

Between January 1981 and March 1982, a survey on lymphatic filariasis was conducted to determine the prevalence of filariasis among immigrants and the risk of transmission in Central and

South Florida.⁴⁵ The authors observed that 45/668 (6.7%) recent Haitian immigrants had microfilaremia.⁴⁵ Some showed varying degrees of inguinal lymphadenopathy, but no definitive signs of filarial disease were seen during the course of the survey.⁴⁵ Most of the participants came from five coastal urban areas in Haiti: Port-au-Prince, Duvalierville, Gonaïves, Lèogane, and Cap-Haïtien.⁴⁵ During this survey, a Haitian with an enlarging scrotal mass was referred to the investigators from a hospital in Naples, Florida.⁴⁵ Degenerating female adult worms were observed in histologic sections of the epididymis.⁴⁵

In 1985, another Haitian immigrant examined in Charleston, South Carolina was diagnosed with an incarcerated right inguinal hernia.⁴⁶ A gelatinous semi-translucent portion of tissue and an attached portion of the spermatic cord were surgically excised.⁴⁶ Histologic examination revealed microfilariae, and the patient was treated with DEC.⁴⁶ In 1986, a patient from India, who lived in Rochester, Minnesota, was seen with a painless left peritesticular mass.⁸³ On histologic examination, adult worms were found.⁸³ The patient was treated with DEC.⁸³

In 1967, during the Vietnam war, a study on lymphatic filariasis was conducted to determine whether American soldiers in South Vietnam were at risk of infection.^{43,113} The authors observed that one (0.9%) of 112 servicemen who were deployed for field duty in Hon Quan district was microfilaria-positive.^{43,113} The soldier was treated with DEC.¹¹³ In 1971, a recently returned Vietnam veteran presented with nausea and right-sided mid-abdominal pain at the Charleston Veterans Administration Hospital.⁴³ No microfilariae were found in blood smears, but the patient did have hypereosinophilia, which responded to DEC treatment.⁴³

Currently, transmission of *W. bancrofti* in the continental United States is considered unlikely, but it cannot be excluded as a possibility. It is probable that thousands of microfilaremic immigrants come to this country every year.

Table 2. Studies on lymphatic filariasis in the United States of America 1909-1967

Year	Setting/Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1909	Blood slides	131	13	9.9	F. B. Johnson, 1915 ²⁹
1914	Wards and the emergency department at Roper Hospital, Charleston	400	77	19.3	F. B. Johnson, 1915 ²⁹
1915	Residents of the Old Folks Home, Charleston	37	13	35.1	E. Francis, 1919 ⁵
1915-1918	Residents of Columbia, Beaufort, and Georgetown, SC; Savannah and Milledgeville, GA; Jacksonville and Tampa, FL; Mobile, AL and New Orleans, LA.	1,470	9	0.6	E. Francis, 1919 ⁵
1942-1944	American troops stationed at the South Pacific island of Tongatabu (daytime blood)	709	1	0.1	T. McP. Brown et al., 1946 ⁹⁰
1963	Children at Laie Elementary School, Laie, Oahu:				R. M. Worth, 1964 ¹⁰⁸
	Samoan children born in Samoa	55	3	5.5	
	Samoan children born in Hawaii	20	0	0.0	
	Non-Samoan children	186	0	0.0	
1967	American troops in Hon Quan district, Vietnam	112	1	0.9	E. J. Colwell et al., 1970 ¹¹³

Table 3. Studies on mosquitoes in the South Pacific Islands where American troops were infected in 1943-1944⁹¹

Year	Population	No. of mosquitoes dissected	No. of mosquitoes infected with microfilariae	Infection rate (%)	Author and year of publication
1943-1944	<i>Aedes scutellaris</i> var. <i>Pseudoscutellaris</i> mosquitoes collected from 35 village campsites	6,634	655	9.9	E. E. Byrd, L. S. St. Amant, and L. Bromberg, 1945 ⁹¹

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THE U.S. VIRGIN ISLANDS consists of Saint Croix, which is the largest, Saint Thomas, and Saint John, the smallest.¹ Saint Croix, previously known as “Ayay” by the Caribs and the Arawaks, was renamed “*Santa Cruz*,” after the arrival of Columbus on its northwestern coast, at the Salt River, during his second voyage, on November 14, 1493.^{1,3,4}

Columbus named the islands “*Las Virgenes*” in honor of Saint Ursula, a 4th century martyr, who supposedly rounded up 11,000 virgins for a pilgrimage to Rome and, as the legend goes, was killed by the Huns on her return to Cologne, Germany.^{3,5,6}

Saint Croix

A constant state of war existed between the Caribs and the Spaniards for nearly a century, with the result that by 1596, Saint Croix was described as “wholly uninhabited.”¹ In the early 1600s, the English and the Dutch settled on the western and eastern parts of the island, respectively.^{1,3} After much conflict, the Dutch left the island, and the English ruled until 1650, when the Spanish took over, dominating the island until the English finally left, 15 years later.^{1,3,4}

In 1665, the French captured the island, named it ‘*Saint Croix*’ and 10 years later, sold it to the Governor of Saint Kitts, de Poincy, who later deeded the island to a religious group called the Order of Saint John of Jerusalem (Knights of Malta).^{1,3,4} In 1675, the French West Indian Company bought the island.¹ For 20 years, it flourished under its new governor, DuBois.¹ After his death in 1695, weak administration, drought, and sickness eroded these advances to such an extent that the island was soon considered abandoned.¹

In 1733, the Danish West Indian and Guinea Company bought the island from the French,^{1,3,4} and in 1734 began importing slaves.⁴ Slavery persisted until emancipation in 1848.^{2,3} The Company successfully governed Saint Croix, cultivating primarily sugarcane for nearly 2 centuries, until January 1917, when it sold Saint Croix, Saint Thomas, and Saint John to the United States.^{1,4,7-9}

Saint Thomas and Saint John

In the 1600s, Saint John and Saint Thomas had become the property of the Danish West Indian and Guinea Company.² Previously, only a handful of British squatters and well-to-do Danes occupied these islands.² By 1717, permanent settlements were established at the port of Coral Bay (Saint John) as well as at Charlotte Amalie (Saint Thomas).² The islands became prosperous from agriculture, chiefly sugarcane, cotton, and tobacco.²

After a successful slave revolt, Saint John was ruled by slaves from November 23, 1733 to the spring of the following year.² The British invaded the islands in 1801 and again in 1807, and ruled until 1814.² The Danes recaptured the islands in 1814 and until 1917 governed both islands together with Saint Croix.^{2,4,7}

Lymphatic filariasis in the U.S. Virgin Islands

There are no records of either elephantiasis or chyluria in these three islands until the mid-19th century.^{1,2} Lymphatic filariasis was first mentioned in 1844, in the Death Registrations of the Moravian Mission Records for Saint Croix, which stated that some deceased persons had suffered from “rose” (lymphangitis).^{4,10,11} Thereafter, this condition and elephantiasis appeared frequently in such reports.^{4,10,11} In 1881, L. Neumann, a Danish physician, wrote a thesis on filariasis in Saint Croix, in which he stated that about 6.0% of the population had elephantiasis.^{4,11,12}

In June 1875, at the New York Hospital, Robert Abbé examined a patient born in Saint Croix, who had “milky urine.”^{13,14} Subsequently, Abbé examined tough mucus gelatinous blood-streaked masses that were passed on micturition and observed “the smaller worm.”^{13,14} This report was soon followed by others from Saint Croix, including Solis-Cohen in 1889, Pontopidan and Blanchard in 1890, Lambert in 1892, and Solis-Cohen in 1892.¹⁵⁻¹⁹

In 1901, at the Johns Hopkins Hospital, E.L. Opie examined a patient originally from Saint Thomas, who had “spasms” and a right inguinal hernia of 8 years’ duration.²⁰ Unfortunately, the patient died the next day.²⁰ An autopsy showed an

enlarged thoracic duct and the presence of male and female adult *Wuchereria bancrofti* worms within a dilated lymphatic vessel and microfilariae within a branch of the hepatic vein.²⁰

In 1917, following the acquisition of the Virgin Islands by the United States, C.S. Butler and E.G. Hakansson found that 3,725/14,901 (25.0%) residents of Saint Croix were microfilaria-positive.³ In 1924, H.M. Stenhouse found microfilariae in 10.0% of blood smears examined at the municipal laboratory in Saint Croix.¹² Subsequently, he reported on the prevalence of renal disease and its relation to lymphatic filariasis.²¹ In his review of medical records of 100 patients with renal disease, 31 (31.0%) had microfilariae.²¹ In a second study, 8/30 (26.7%) persons with renal disease were microfilaremic.²¹ Stenhouse identified 611 cases of lymphatic filariasis through a hospital record review during a 5-year period.²¹

In 1919, during the period of organization of the Health Department under the U.S. Naval Medical Service, an order was given to include examination of blood for *W. bancrofti* microfilariae in routine patient evaluation.^{7,21} This policy provided data for H.V. Hughens, who, in 1927, reviewed admissions records from the Municipal Hospital in Christiansted (northeastern coast) and the Municipal Hospital in Frederiksted (southwestern coast).⁷ These two towns are 15 miles apart.⁷ The review period was from April 1919 to February 1920.⁷ Of 505 persons admitted to the Municipal Hospital in Frederiksted, 161 (31.8%) had microfilariae identified in blood smears, and 59 (11.7%) had clinical manifestations of lymphatic filariasis.⁷ Of 1,237 persons admitted to the Municipal Hospital in Christiansted, 143 (11.6%) had clinical manifestations of lymphatic filariasis.⁷

In 1936, James Knott conducted extensive investigations on filariasis in Saint Croix.⁴ In one study, he examined 2,000 schoolchildren and observed that 320 (16.0%) had microfilaremia.⁴ In another study at the Old People's Farm in King's Hill, located mid-way between Frederiksted and Christiansted, Knott observed that 57/124 (46.0%) persons were microfilaria-positive.⁴ In a third study, males from all over the island between the ages of 16 and 25 were examined at the Civilian Conservation Corps camp; 43.0% were microfilaremic.⁴ In a subsequent study, F.W. O'Connor observed that 39.0% persons had microfilaremia in 2 ml of blood, compared with 53.0% in 10 ml of blood.⁴ These studies provided evidence that filariasis was highly

prevalent in Saint Croix, with foci in Frederiksted, Christiansted, and King's Hill.⁴

From October 1935 to December 1936, O'Connor and H.A. Beatty collected *Culex fatigans* mosquitoes to determine if these insects were infected with *W. bancrofti*.⁴ The mosquitoes were collected from the Christainsted Municipal Hospital, from Christiansted town and its environs, from King's Hill, and from Castle Coakley, located in central Saint Croix, east of King's Hill.⁴ Of 5,000 mosquitoes, 1,254 (25.1%) were infected, while 115 (2.3%) were found to harbor mature (infective) larvae.⁴

In a study to determine the microfilaremia prevalence in the populations of Saint Thomas and Saint Croix, 195 persons were selected at random from Saint Thomas in April and May 1939.²² In addition, 200 consecutive patients were tested on admission to the Municipal Hospital at Christensted, Saint Croix between March and July 1939.²² Only one (0.5%) person from Saint Thomas was microfilaria-positive, while 50 (25.0%) persons from Saint Croix were microfilaria-positive.²²

In New York during the summer of 1943, H.W. Brown studied lithium antimony thiomalate as a treatment for lymphatic filariasis; 13 of his patients, all microfilaria-positive, were originally from the U.S. Virgin Islands.²³ This treatment temporarily reduced the microfilaremia count in these patients for an average of 4-5 months.²³ In 1946, nine persons from Saint Croix, who were serving in the U.S. Navy, were found to be asymptotically infected with *W. bancrofti*.²⁴

To ascertain the effects of DDT residual house spraying on lymphatic filariasis in Saint Croix, Brown and R.W. Williams collected pre- and post-spraying blood samples for smears from children under 15 years of age.^{25,26} They also collected and dissected mosquitoes.^{25,26}

This work was done in Christiansted, Frederiksted, and on various sugarcane estates.^{25,26} The pre-spraying mosquito survey was conducted during June and July 1946, while the pre-spraying blood survey was conducted in October 1946.^{25,26} Of 1,311 children examined, 164 (12.5%) had microfilaremia; 113/1,682 (6.7%) *Culex quinquefasciatus* mosquitoes were infected with filarial larvae, while 7/1,682 (0.4%) mosquitoes harbored mature filarial larvae.²⁶

DDT residual spraying began in October 1946 and continued for 21 months.^{25,26} The post-spraying mosquito survey and the post-spraying blood survey

were done in June 1948, 21 months later.²⁷ The post-spraying survey showed that 96/906 (10.6%) children were microfilaria-positive.^{26,27} Of 846 *C. quinquefasciatus* mosquitoes, 31 (3.7%) were infected; none harbored mature filarial larvae.^{26,27} Thus, after 21 months of DDT residual house spraying, the proportion of mosquitoes containing *W. bancrofti* decreased significantly, but there was only a

slight reduction (not statistically significant) in the prevalence of microfilaremia in children.^{26,27}

Before mass treatment with diethylcarbamazine (DEC) was attempted for the control of lymphatic filariasis in Saint Croix, surveys were conducted in five counties during April 1948.²⁷⁻²⁹ Of 724 persons of various age groups, 123 (17.0%) were microfilaria-positive (Table 1).^{27,28}

Table 1. Lymphatic filariasis surveys in Saint Croix, April, 1948 by Hewitt and White^{27,28}

County	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Bethlehem Village	139	26	18.7
Grove Place	207	27	13.0
Bethlehem Sugar Mill	155	36	23.2
King's Hill	134	25	18.7
La Vallee	89	9	10.1
Total	724	123	17.0

From June 14 to June 24, 1948, DEC tablets were distributed.²⁷ Each adult received 21 100-mg tablets and each child 21 50-mg tablets.^{27,28} One tablet was taken three times a day for 7 days.^{27,28} In June 1949, 14 months after mass treatment, a follow-up survey was conducted (Table 2). Of 434 persons of

various ages selected from the five counties, 31 (7.1%) were microfilaria-positive.^{27,28} Thus, there was a pronounced reduction in the microfilaremia prevalence in all five listed counties 1 year after treatment with DEC.²⁷

Table 2. Post-treatment microfilaremia prevalence, Saint Croix, June 1949 by Hewitt and White^{27,28}

County	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)
Bethlehem Village	84	4	4.8
Grove Place	79	4	5.1
Bethlehem Sugar Mill	91	10	11.0
King's Hill	130	10	7.7
La Vallee	50	3	6.0
Total	434	31	7.1

In 1947, during a study to determine whether phenyl arsenoxide was suitable for the treatment of lymphatic filariasis, six microfilaremic patients, all originally from Saint Croix, were treated with the drug.^{30,31} Later that year, an additional 12 patients from Saint Croix were treated with arsenamide.³¹ In both instances, the patients responded well to

treatment, as they became amicrofilaremic within 11 to 12 months.^{30,31}

The latest available reports, published in 1975, suggests that lymphatic filariasis was present in the U.S. Virgin Islands, particularly Saint Croix.³² Table 3 and 4 summarize the studies on lymphatic filariasis in the U.S. Virgin Islands.

Table 3. Studies on lymphatic filariasis in the US Virgin Islands, 1917-1949

Year	Setting / Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1917	General population, Saint Croix	14,901	3,725	25.0	C. S. Butler and E. G. Hakansson, 1917 ³
1919-1920	Hospital admission records from Frederiksted Municipal Hospital, Saint Croix	505	161	31.9	H. V. Hughens, 1927 ⁷
1924	Blood smears, Municipal Laboratory, Saint Croix	—	—	10.0	H. M. Stenhouse, 1924 ¹²
1925	Case histories of persons with renal disease, Saint Croix	100	31	31.0	H. M. Stenhouse, 1925 ²¹
1925	Persons with renal disease, Saint Croix	30	8	26.7	H. M. Stenhouse, 1925 ²¹
1925	District of Christiansted, Saint Croix	—	—	10.0	H. M. Stenhouse, 1925 ²¹
1925	District of Frederiksted, Saint Croix	—	—	20.0	H. M. Stenhouse, 1925 ²¹
1936	Schoolchildren, Saint Croix	2,000	320	16.0	Survey conducted by J. Knott, but cited by F. W. O'Connor and H. A. Beatty, 1938 ⁴
1936	Old People's Farm, King's Hill	124	57	46.0	Survey conducted by J. Knott, but cited by F. W. O'Connor and H. A. Beatty, 1938 ⁴
1936	Males 16-25 years old, Civilian Conservation Corps camp	—	—	43.0	Survey conducted by J. Knott, but cited by F. W. O'Connor and H. A. Beatty, 1938 ⁴
1939	Randomly selected persons, Saint Thomas	195	1	0.5	G. M. Saunders, 1941 ²²
1939	Admissions, Christiansted Municipal Hospital, Saint Croix	200	50	25.0	G. M. Saunders, 1941 ²²
1946	Children < 15 years old in Christiansted, Frederiksted, and various sugarcane estates, Saint Croix	1,311	164	12.5	H. W. Brown and R. W. Williams, 1949 ²⁶
1948	Children < 15 years old, Christiansted, Frederiksted, and various sugarcane estates, Saint Croix (after 21 months of DDT spraying)	906	96	10.6	H. W. Brown and R. W. Williams, 1949 ²⁶
1948	Five counties in Saint Croix	724	123	17.0	H. W. Hewitt, 1949; H. W. Hewitt and E. White, 1950 ²⁷
1949	Five counties in Saint Croix (post-treatment with DEC)	434	31	7.1	H. W. Hewitt, 1949; H. W. Hewitt and E. White, 1950 ²⁷

Table 4. Prevalence of *W. bancrofti* infection in *Culex quinquefasciatus* mosquitoes, 1935-1948, US Virgin Islands

Year	Setting	No. of mosquitoes collected and dissected	No. of mosquitoes positive for microfilariae	Infection rate (%)	Author and year of publication
1935-1936	Christiansted, King's Hill, and Castle Coakley, Saint Croix	5,000	1,254	25.1	F. W. O'Connor and H. A. Beatty, 1938 ⁴
1946	Christiansted, Frederiksted, and various sugarcane estates, Saint Croix	1,682	113	6.7	H. W. Brown and R. W. Williams, 1949 ²⁶
1948	Christiansted, Frederiksted, and various sugarcane estates, Saint Croix (after 21 months of DDT spraying)	846	31	3.7	H. W. Brown and R. W. Williams, 1949 ²⁶

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THE ORIGINAL INHABITANTS of Venezuela are believed to include three groups: the Chibchas, the Caribes, and the Arawaks.^{1,2} Christopher Columbus first sighted Venezuela during his third voyage, when he saw the Península de Paria from his ship, which was anchored off the coast of Trinidad.³ Three days later, on August 1, 1498, Columbus likely became the first European to set foot on the South American mainland.³ He christened the territory "*Isla de García*," and spent the next 2 weeks exploring the Río Orinoco Delta.³ Columbus was fascinated with the vast source of fresh water and the pearl ornaments of the indigenous population.³

A year later, Alfonso de Ojeda and the Florentine, Amerigo Vespucci, led a second expedition to Venezuela.³ The expedition sailed westward along the coast of "*Tierra Firme*" (as South America was then known) as far as Lago de Maracaibo.³ Vespucci saw huts built on piles above the lake that reminded him of Venice, leading him to name the area "Little Venice," or "*Venezuela*."¹⁻³ In 1521, the first Spanish settlement on the mainland was established at Cumaná.^{1,2}

Subsequent expeditions along the north coast of South America were driven largely by a desire for pearls and rumors of precious metals.^{1,2} By the 1520s, the oyster beds between Cumaná and the Isla de Margarita, located at the western end of the Península de Paria, had been depleted.^{1,2} Spanish explorers, in the meantime, pushed eastward to establish El Tocuyo and then Valencia in 1555.^{1,2} After more than a decade of fierce fighting with the indigenous population, forces under Diego de Losada established the settlement of Santiago de León de Caracas in 1567.³ The value of Caracas lay not only in the fertile agricultural lands in its vicinity, but also in its accessibility to the seaport that would later become "*La Guaira*."³

Soon after, the original inhabitants were captured as slaves to provide labor needs in Panama and the Caribbean islands, where gold and silver bullions from Mexico and Peru were shipped back to Spain.³ In 1528, more Spaniards and Germans arrived in search of precious metals and jewels, but they were unsuccessful.³ By the 1620s, cocoa became the principal export, which it remained for the next 2

centuries.³ Cocoa plantations created a great demand for slaves, who were imported during the 17th and 18th centuries.³ Slavery persisted until emancipation in 1854.³

The Spanish were eventually ousted by Simón Bolívar Palacios, when Venezuela was taken from Spain, during a decisive victory in 1821 at Campo Carabobo, near Valencia.¹ Bolívar had already brought independence to Colombia, and went on with his lieutenant Antonio José de Sucre, to liberate Ecuador, Peru, and Bolivia.¹ A united state of "*Gran Colombia*," which would unify Colombia, Venezuela, and Ecuador, did not survive his death.¹ In 1830, Venezuela declared full independence under a new constitution.¹

The earliest reference to lymphatic filariasis appears in a text by Moncorvo, who in 1904 listed Venezuela as "an infested country."^{4,5} During 1911 and 1912, a study on bloodsucking *Diptera* in Venezuela was conducted by J. Surcouf and R. Gonzales Rincones.^{5,6} They described clinical manifestations of chyluria and elephantiasis in Venezuela, and identified Puerto Cabello, on the northwestern coast, as an important focus for endemic lymphatic filariasis transmission.^{5,6} Other texts at the time also listed Venezuela as "an infested country" for lymphatic filariasis.⁷⁻⁹

In 1921, P.D. Rodriguez Rivero documented several cases of elephantiasis and chyluria in Venezuela, and reported on operative treatment for elephantiasis.¹⁰ On October 17, 1922, P.A. Guzmán made the first observation of microfilaremia in Venezuela.¹¹ Microfilariae were also found in the urine of the same patient.¹¹ He examined 12 patients with various symptoms of lymphatic filariasis and 38 family members of these patients; all 50 of them had microfilaremia.¹¹ Furthermore, Guzmán reported that eight persons from Puerto Cabello presented with clinical manifestations of lymphatic filariasis; acute lymphangitis, elephantiasis of the legs, or chyluria.¹² All were microfilaria-positive in blood, lymph fluid, or urine.^{12,13}

In 1923, a patient with pain in the right flank and pain on micturition was found to be microfilaria-positive during blood examination.¹⁴ In 1927, a focus of endemic *Wuchereria bancrofti* was identified in

Trujillo, located in the western region of the country.¹⁵⁻¹⁸ Within the same year, P. Mühlens reported an endemic focus in Puerto Cabello, where 14 (18.9%) of 74 laborers had microfilaremia.¹⁹ In addition, Mühlens stated that although he was unable to search for microfilariae in other nearby areas, it was highly possibly that the disease was endemic in other coastal towns, especially in and around Maracaibo.¹⁹

Other researchers have also documented lymphatic filariasis in Puerto Cabello.^{20,21} In January-December 1948, Reginald Hewitt, from Lederle Laboratories of the American Cyanamid Company, A.L. Briceño Rossi from the Institute of Hygiene in Caracas, and Argimiro Bracamonte, Soriano Rutman, Torres Suels and Páez Maya, all from Puerto Cabello, investigated the effectiveness of diethylcarbamazine (DEC) for treatment of lymphatic filariasis in Puerto Cabello.²²⁻²⁵ Sixteen microfilaremic persons from Puerto Cabello were given DEC three times a day for an average of 18 days and were examined at monthly intervals for one year.^{22,23} After 12 months, the microfilarial density in all patients was zero.^{22,23}

According to published official national reports, beginning in 1949, there were 64 cases of filariasis; in 1950 (80); 1951 (135); 1952 (65); 1953 (21); 1954 (15); 1955 (17); 1956 (47); 1957 (12); 1958 (52); and 1959 (31).²¹ In 1952 and 1961, Briceño Rossi and

Pifano, respectively, reported that lymphatic filariasis was not found among the Amerindians living in the central-interior of the country at the basin of the Orinoco River.¹⁷

In 1976, the records of patients from 1954 to 1974 at the Hospital Vargas, Caracas were reviewed by Gómez and colleagues.²⁶ The records revealed that 78 patients had clinical histories suggestive of filarial lymphedema or elephantiasis.²⁶ All of these patients came from states in the northern coastal region; 17 (21.8%) from the Federal District and 12 (15.4%) from Miranda.²⁶ Other reports suggested a focus of filariasis in the north-central coastal areas of Venezuela.²⁷ Cases were said to have declined between 1971 and 1991.²⁷

In 1999, Eusebio Placencias, Jorge Arias, and Jacinto Convit conducted a survey in Puerto Cabello and Patanemo.²⁸ Of 563 persons, whose ages ranged from 48 to 96 years, only one person tested positive for circulating filarial antigen using ICT filariasis card test.²⁸ Two additional ICT card tests on this patient proved negative.²⁸ This study suggests an absence of *W. bancrofti* in these areas.²⁸ Sanitary engineering projects that accompanied the development of both cities during the past 50 years may have interrupted transmission.²⁸

Table 1. Studies on lymphatic filariasis conducted in Venezuela

Year	Setting / Population	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1927	Laborers from Puerto Cabello	74	14	18.9	P. Mühlens, 1932 ¹⁹
1999	General population in Puerto Cabello and Patanemo	563	0	0 (antigen)	E. Placencias, J. Arias and J. Convit, 1999 ²⁸ (unpublished)

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PART 5

Countries with Possible but Not Well-Documented Transmission

ARUBA AND BONAIRE, together with Curaçao, are part of the Netherlands Antilles and are called the Netherlands Leeward islands.¹ This distinction causes much confusion, as these islands are located in an area also known geographically as the “British Windward Islands.”^{1,2}

The original inhabitants of Aruba and Bonaire were the Caiquetios, a tribe of peaceful Arawak Indians, who lived in small communities under a chieftain or a priest.¹ They survived principally on fish and shellfish and collected salt from the Charoma salt pan to barter with their mainland neighbors (in present-day Venezuela) for supplements to their diet.¹

In 1499, the Spanish navigator Alfonso de Ojeda, one of Columbus’ lieutenants, accompanied by the Florentine, Amerigo Vespucci, and the Spanish cartographer, Juan de la Cosa, came upon these islands (Curaçao included).¹ In 1527, the first Spanish settlers arrived, and by 1621, the Dutch had become frequent visitors.¹ In 1634, the Dutch came with the Netherlands West Indian Company and took over Curaçao, apparently deporting the Spaniards and a few remaining Arawak Indians to Venezuela.¹ On Aruba and Bonaire, the Arawak Indians maintained their identity until about the end of the 19th century, when they were absorbed into the rest of the population.¹

Conflicts between Britain and the Netherlands in the second half of the 17th century led to skirmishes and conquests in the Caribbean.¹ In 1678, the “Peace treaty of Nijmegen” gave the Dutch the following: the islands of Aruba, Bonaire, and Curaçao, plus three smaller islands in the “British Leeward” group of islands, Saint Eustatius (Statia), Saba, and the southern half of Saint Martin/Sint Maarten.¹

From 1828 to 1845, all Dutch West Indian colonies were governed from Suriname.¹ In 1845, the Netherlands Leeward islands were joined to Willemstad, the capital of Curaçao, to form one colonial unit called Curaçao and Dependencies.¹

In the 20th century, the economy prospered with the discovery of oil in Venezuela and the subsequent decision by the Dutch-British Shell Oil Company to set up a refinery on Curaçao because of its political stability, its good port facilities, and its climate which was better than that found around Lake Maracaibo in

Venezuela.¹ Likewise, in 1924 another refinery was built on Aruba, which brought unprecedented wealth to the island, as well as growth in population.^{1,3} World War II was another turning point in the economic development of these islands, as demand for oil soared and British, French, and later U.S. forces were stationed on the islands.¹

Lymphatic filariasis in Aruba and Bonaire

Occasional records of lymphatic filariasis and elephantiasis in Aruba can be found in government reports from 1875 until 1946.² No cases were reported from Bonaire.

From 1942 to 1947, Edward Van der Kuyp made four and six visits to Aruba and Bonaire, respectively.³ On these trips, he collected mosquitoes and found that *Aedes aegypti* and *Culex quinquefasciatus* were the most common.³ Although he saw no cases of lymphatic filariasis, he believed that the preponderance of *C. quinquefasciatus* on both islands constituted a potential danger, if infected immigrants were to migrate to Aruba and Bonaire.³

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THE ORIGINAL INHABITANTS of Belize are believed to have been the Maya, who probably lived in this area from 1000 B.C. to 1500 A.D.¹ In 1525, Hernan Cortés arrived in southern Belize and the Spanish made attempts at settlement.^{1,2} In the 1530s, Montejo attempted to conquer Belize for Spain, but “*Nachankan*” and the Belize Maya defeated the Spanish.¹

Although regarded as a Spanish territory, the first settlers in Belize were English Puritans, who set up trading posts along the coast of Belize.¹ In the 1650s, various bands of shipwrecked sailors, buccaneers, and pirates established permanent bases in Belize, targeting the Spanish galleons carrying gold, silver, and hardwoods from Central America to Europe.¹ During this period, logging became the dominant occupation.¹ These pirates, who called themselves “Baymen,” prompted the Spanish to sign the “Treaty of Paris” in 1763 and the “Treaty of Versailles” in 1783 with the British, giving the British rights to harvest timber in exchange for protection against pirates preying on the Spanish galleons.¹

In 1720s, slaves were brought to Belize, until emancipation between 1834 and 1838.¹ In 1832, large numbers of the Garifuna people from the Bay Islands of Honduras migrated and settled along the coast of Belize.¹ In 1862, Belize become known as the Colony of “British Honduras” and by 1871, it was declared a Crown Colony after its assembly dissolved itself a year earlier.¹ In 1973, its name was changed from British Honduras to Belize and on September 21, 1981, Belize declared its independence.¹

In 1935, the presence of filariasis was mentioned in a report by E.B. McKinley, but without further details as to location and prevalence.³ Statistical reports from the Central Laboratory in Belize from circa 1955 to 1975 do not mention indigenous cases of filariasis.⁴

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CURAÇAO, TOGETHER WITH ARUBA and Bonaire, are part of the Netherlands Antilles and are called the Netherlands Leeward islands.¹ The original inhabitants of Curaçao were the Caiquetios, a tribe of peaceful Arawak Indians, who lived in small communities under a chieftain or a priest.¹ They survived principally on fish and shellfish and collected salt from the Charoma salt pan to barter with their mainland neighbors (in present-day Venezuela) for supplements to their diet.¹

In 1499, the Spanish navigator Alfonso de Ojeda, one of Columbus' lieutenants, accompanied by the Florentine, Amerigo Vespucci, and the Spanish cartographer, Juan de la Cosa, came upon the island.¹ In 1527, the first Spanish settlers arrived, and by 1621, the Dutch had become frequent visitors to Curaçao.¹ In 1634, the Dutch came with the Netherlands West Indian Company, took over Curaçao, and apparently deported the Spaniards and a few remaining Arawak Indians to Venezuela.¹ From 1639 to 1778, thousands of slaves were brought to Willemstad (capital of Curaçao) and sold to the mainland and other colonies.¹ The Dutch brought nearly half a million slaves to the Caribbean, most of whom went through Curaçao.¹

In 1642, Peter Stuyvesant ruled as governor of Aruba, Bonaire, and Curaçao and set up a slave depot, where slaves were held after the middle passage before being resold and sent to other islands or countries in the Americas.² Much of Curaçao's wealth came from the slave trade.¹ In 1645, Stuyvesant left for New Amsterdam, which later became modern-day New York.² In 1654, Dutch refugees from Brazil brought their sugarcane expertise, but by 1688, the crop was abandoned because of the very dry climate.¹ At about this time citrus fruits were introduced; however, salt remained a valuable commodity.¹

From 1666 into the early 19th century, the English and French alternately tried to capture Curaçao, but the massive fortresses built to defend the island prevented any successful invasions.¹ In 1678, the Dutch were given Aruba, Bonaire, Curaçao, and three smaller islands in the "British Leeward" group of islands, Saint Eustatius (Statia), Saba, and the southern half of Saint Martin/Sint Maarten, under the provisions of the "Peace treaty of Nijmegen."¹

However, from 1800 to 1803, and again from 1807 to 1815, Curaçao fell under British rule.¹ In 1815, it was reclaimed by the Dutch.¹

From 1828 to 1845, all Dutch West Indian colonies were governed from Suriname.¹ In 1845, the Netherlands Leeward islands were joined to Willemstad, to form one colonial unit called Curaçao and Dependencies.¹ The economy largely depended on trade with Venezuela, including a shipbuilding industry and some phosphate and salt pan mining.¹ The latter declined after emancipation in 1863.¹

In the 20th century, the economy prospered with the discovery of oil in Venezuela and the subsequent decision by the Dutch-British Shell Oil Company to set up a refinery on Curaçao because of its political stability, its good port facilities, and its climate compared with that found around Lake Maracaibo in Venezuela.¹ In 1954, Curaçao became an autonomous part of the Kingdom of the Netherlands.³

The earliest records of lymphatic filariasis and elephantiasis in Curaçao are from government reports, which begin in 1875 and continued until 1946.⁴⁻⁶ No details of the cases or description of signs or symptoms are given, and there appears to be have been no evidence of autochthonous cases.⁴⁻⁷

From 1941 to 1943, Edward Van der Kuyp conducted a survey of 39 persons in Curaçao (34 adults and five children).⁷ Of the four persons (10.3%) who had microfilariae (Table 1),⁷ one adult had resided in Curaçao for a substantial period of time, in addition to having lived in Saba and Saint Martin/Sint Martin.⁷ Of the other two microfilaremic adults, one had arrived from Saint Martin/Sint Martin 20 days before the survey, and the other had lived in Guadeloupe.⁷ The remaining microfilaria-positive person was a child who had arrived from Saint Martin/Sint Martin 10 days before the survey.⁷ Thus, only one of the four persons infected with *Wuchereria bancrofti* may have acquired the infection from Curaçao.⁷

In June 1942, A. Van der Sar and Philip H. Hartz saw a 46-year-old woman who had a cough and palpable lymph nodes in her left axilla.^{8,9,11} No microfilariae were seen in a sample of her blood, but histologic examination of a biopsy of her lymph nodes showed eosinophilia, and "infiltrates of small worms

were observed, which showed the characteristics of microfilaria.”^{8,9}

In 1944, Hartz documented three cases of lymphatic filariasis.¹⁰ He examined two young girls, both of whom had small masses between their breast and the axilla.¹⁰ The third patient was an adult man with a gradual enlargement of the left inguinal lymph nodes and a hydrocele.¹⁰ No microfilariae were obtained in blood samples of any of the patients, but fragments of *W. bancrofti* adult worms were identified in biopsy specimens from all three patients.¹⁰

In 1947, Hartz and Van der Sar examined a 30-year-old woman, who had a swelling between her left breast and axilla.¹¹ Histologic findings of a biopsy of her lymph nodes revealed disintegrated microfilariae surrounded by numerous eosinophils.¹¹

In 1950, Hartz examined a 24-year-old man, who had a right testicular swelling.¹² A diagnosis of testicular tumor was made, and semi-castration surgery was performed.¹² Histologic examination of the removed tissue revealed several disintegrated microfilariae surrounded by macrophages.¹²

Table 1. Studies on lymphatic filariasis in Curaçao, 1941-1943⁷

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1941-1943	Adults	34	3	8.8	E. Vander Kuyp, 1948 ⁷
1941-1943	Children	5	1	20.0	E. Vander Kuyp, 1948 ⁷

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THE ARAWAKS ARE BELIEVED to have been the initial inhabitants of Grenada.¹ The next group to colonize the island were the Caribs, who called it "*Camerhogue*."¹ Christopher Columbus named it "*Concepción*," when he came upon the island on his third voyage in 1498, but no settlements were established.¹ The island was later referred to as "*Mayo*" on maps, and was then changed by the Spaniards to "*Granada*," after the Spanish city.¹ The French then called it "*La Grenade*" and by the 18th century it came to be known as "*Grenada*."¹

The Caribs prevented European settlement until the 17th century.¹ In 1609, the English unsuccessfully tried to establish settlements, as did the French in 1638.¹ It was not until 1650 that a French expedition from Martinique made friendly contacts with the Caribs.¹ When relations soured, the French brought reinforcements and greatly reduced the number of inhabitants of the island.¹ "*Sauteurs*" or "*Morne des Sauteurs*," a site on the north coast, is named after this carnage, when Caribs apparently jumped to their death in the sea rather than surrender to the French.^{1,2}

Possession of the island and its dependencies was hotly disputed between the French and the British until 1783, when it was ceded to Britain under the "Treaty of Versailles."^{1,2} Planters imported slaves as laborers until emancipation in 1838.¹ British rule continued until its independence from Britain on February 7, 1974.¹

The earliest survey on lymphatic filariasis in Grenada was conducted by George C. Low, who in 1908 examined 174 patients and observed that none were positive for microfilariiae.³⁻⁵ On Carriacou island, a dependency of Grenada, Low found that one (3.6%) of 28 patients had microfilaremia.³⁻⁵ On further investigation, the patient was found to have resided in Saint Kitts since birth and had moved to Carriacou just 1 year previously.³⁻⁵

In 1930, no cases of lymphatic filariasis were reported,⁶⁻⁸ but in 1945, 33,970 employees at a naval base in Trinidad were examined, and two Grenadines were found to be microfilaria-positive.^{9,10}

Table 1. Studies on lymphatic filariasis in Grenada, 1908-1945

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1908	Unknown	174	0	0.0	Low, 1908 ⁴ ; Low, 1908 ⁵ ; Low, 1908 ⁶
1908	Carriacou Island	28	1	3.6	Low, 1908 ⁴ ; Low, 1908 ⁵ ; Low, 1908 ⁶
1945	Naval base in Trinidad	—	2	—	R. F. Platzer and W. K. A. Lawlor, 1945 ⁹ ; H. Beye et al., 1961 ¹⁰

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EVIDENCE exist of Maya settlement at Copán in western Honduras since at least 1000 B.C., but like other Maya city-states, it was abandoned mysteriously around 900 A.D.¹ In 1502, Christopher Columbus set foot on the American mainland for the first time at Trujillo in northern Honduras and named the country after the deep water off the Caribbean coast.¹ “Honduras” means “depths.”¹ The Spanish settled in Trujillo in 1525, but soon became interested in colonizing the cooler highlands.¹ In 1537, they established a capital at Comayagua in central Honduras, and this remained the political and religious center of the country for 350 years, until Tegucigalpa became the capital in 1880.¹

The Indians resisted Spanish colonialism, and, by some accounts, almost managed to drive the colonists from the mainland.^{1,2} The chief of the Lenca tribe, Lempira, led 30,000 Indians against the Spanish, but was treacherously murdered at peace talks in 1538,^{1,2} and by the following year, resistance was crushed.¹ In 1570, gold and silver were discovered near Tegucigalpa, attracting British and Dutch pirates to the Trujillo area.¹ Around 1600, the Spanish estimated that Roatán was home to 5,000 British buccaneers.¹ In 1643, Trujillo was raided by Dutch pirates and was not resettled by the Spanish until 1787.¹ While Spain concentrated its energies on the interior, the British were attracted to the Caribbean coast by stands of mahogany, and brought black settlers from Jamaica and other West Indian islands to harvest the timber.¹ After an appeal by chiefs of the Miskito Indians, a British protectorate was declared over the entire coastal region extending from Honduras into Nicaragua.¹ This lasted until 1859, when the area was relinquished to Honduras.¹

Independence from Spain was granted in 1821.^{1,2} Honduras briefly became part of independent Mexico, but seceded in 1823.^{1,2} Honduras then joined the Central American Federation, but conflicts between conservatives and liberals led to a break from the union, and in 1838, Honduras declared independence as a separate nation.^{1,2}

There are no available records of either elephantiasis or chyluria in Honduras until 1930, when a patient was admitted to the Truxillo Railroad Company Hospital in Puerto Castilla for work-related

injuries.³ During a routine blood examination he was found to be microfilaria-positive.³ The patient had no symptoms of lymphatic filariasis and had previously lived in Nicaragua and Costa Rica and had also visited Panama.³

In 1949, D.C.A. Butts mentioned in a report a possible focus of filariasis in Comayagua, in the southwest-central part of the country, northwest of Tegucigalpa.^{4,5}

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THE ORIGINAL INHABITANTS of Jamaica are believed to have been the Arawaks, who arrived on the island around 700 A.D.¹ In May, 1494 Christopher Columbus came upon the island.¹⁻³ Around 1509, the English arrived on the island with imported slaves² and in 1510, the Spanish arrived and established settlements at Sevilla La Nueva, near present-day Saint Anne's Bay.^{1,2}

In 1654, an English contingent sailed to the Caribbean, and after failing to capture Hispaniola (present-day Haïti and the Dominican Republic), turned on poorly defended Jamaica.¹ In 1655, despite the resistance of Spanish loyalists and freed slaves, the British took control of the island.¹⁻³ Labor for the production of cocoa, coffee, and sugarcane, which were major industries of the island, was provided by slaves until emancipation on August 1, 1834.¹ The island remained under British rule until 1962, when Jamaica became an independent member of the British Commonwealth.^{1,2}

Evidence of lymphatic filariasis in Jamaica is largely inferential. The earliest records of the possible occurrence of the disease in Jamaica are from the Covey Estate of John Tharp, dated April 24, 1805, in which two female slaves are recorded as suffering from elephantiasis.⁴ Around 1871, many Jamaicans reportedly migrated to Costa Rica to provide labor in the construction of the Northern Atlantic Railroad from Puerto Limón to San José and in the banana plantations in the late 19th century.⁵⁻⁹ Many of them settled in Puerto Limón, in the subdivisions of Cienguita (now Cristóbal Colón) and Jamaica Town (now Roosevelt).⁵⁻⁸

Studies conducted as early as 1946 showed that 56/706 (7.9%) residents of Cristóbal Colón and 45/300 (15.0%) residents of Roosevelt were microfilaria-positive.⁵⁻⁷ The author, D.C.A. Butts, speculated that lymphatic filariasis may have spread to Costa Rica from Jamaica, during the mass migration of the 1870s.⁵⁻⁹ These immigrants were confined by law to Puerto Limón.^{8,9} Until 1949, Puerto Limón appeared to be the only community in Costa Rica in which filariasis occurred.⁵⁻⁸ From these statements, it would seem that lymphatic filariasis did occur in Jamaica, at least in the 1870s.

On September 8, 1896, C.W. Young, at the

Charing Cross Hospital in London, examined a young man from Jamaica, who had a swollen scrotum and bilateral enlargement of the inguinal area.¹⁰ On examination, the author observed that in addition to thickened spermatic cords and a "doughy" feel on palpation of the scrotum, the patient was positive for microfilariae.¹⁰

In 1906, C.F. Wanhill found a single case of filariasis "among several troops of native soldiers" within Jamaica.¹¹ The patient, a gunner in the Jamaica Company Royal Garrison Artillery, was a native of Saint Elizabeth, Jamaica.¹¹ Wanhill stated that "after inquiring among local medical officers of experience ... while occasionally encountered in Jamaica, filariasis is almost unknown in that island."¹¹

Within the same year, in 1906, a "Dr. Mott" and Michael Grabham failed to find any microfilariae in a series of 400 blood slides, which were collected from the general population in Jamaica.¹¹ Several hundred blood smears were sent to England for examination by pathologists, and these were also negative.¹¹

In 1909, a Jamaican farmer, who 22 years before had emigrated to Rio Grande, Panama (Isthmus), presented to the Ancón Hospital in Panama with a swelling of his scrotum of 5 years' duration.¹² He may have acquired the disease after his departure from Jamaica.

One possible indigenous case was seen and reported by a "Dr. Myers" in Saint Anne's Bay, northwest of Kingston.¹³ The patient had elephantiasis of the scrotum.¹³ J.F. Siler also reported that imported cases of filariasis were occasionally observed.¹³

Data were compiled from November 1929 to April 1931 at the Public Hospital in Kingston, to determine the possible filarial and streptococcal etiologies of lymphangitis and elephantiasis.¹⁴ Of 250 persons in this study, none were positive for microfilariae.¹⁴ The authors concluded that while lymphatic filariasis occasionally appeared in Jamaica, no evidence of its spread was apparent, despite the presence of the mosquito vector.¹⁴

In 1955, surveys conducted at the Hansen Home Leprosarium in Jamaica suggested that lymphatic filariasis was absent from Jamaica and its dependencies, the Turks and Caicos.¹⁴

Table 1. Studies on lymphatic filariasis in Jamaica, 1906-1931

Year	Setting	No. of persons examined	No. of persons microfilaria-positive	Microfilaremia prevalence (%)	Author and year of publication
1906	General population	400	0	0.0	C. F. Wanhill, 1906 ¹¹
1929-1931	Public Hospital, Kingston	250	0	0.0	A. W. Grace, F. B. Grace and S. Warren, 1932 ¹⁴

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THE ORIGINAL INHABITANTS of Mexico are the Olmec, whose civilization began around 1500 B.C., and reached its height about 1200 B.C.¹ "Olmec," which means "people from the rubber country" were an advanced Mayan culture in religion, architecture, and mathematical systems.¹ By 500 A.D. two great cities had emerged, Teotihuacan (with a population of approximately 200,000) and Cholula, a religious center near Puebla which survived until the Spanish Conquest in 1521.¹ The height of Mayan civilization was reached between 600 and 900 A.D.¹

The Toltecs, whose capital was Tula, were the predominant civilization of this time.¹ Known for their fine architecture, elegant speech, and intellectual pursuits, they were the ancestors of the famous Aztecs, who were thriving at the time when Columbus arrived in 1492.¹

In 1519, a Spaniard named Hernan Cortés arrived from Cuba with a crew of 550 sailors and explorers and settled just north of present-day Veracruz.¹ By this time, the Aztec empire controlled vast territories from the Yucatán peninsula to the Pacific, with more than 370 individual nations under their authority.¹ Ruling from their capital city, Tenochtitlan, the Aztecs demanded heavy tribute from their subjects, which may have caused some to side with Cortés in his attack on the Aztecs.¹

The other factor on Cortés' side was the lucky coincidence that 1519 was the exact year when legend had it that the Aztec god, Quetzalcoatl, had promised his followers he would return – from the east – and so Cortés was mistaken for a god.¹ After 2 years of fighting and great loss of life on both sides, the Aztecs were defeated under their final ruler, Cuauhtemoc.¹ Under Spanish rule, local culture was suppressed and native traditions were discouraged.¹ Mexico achieved independence after the wars of 1810 and 1821.¹ In 1824, a constitution was adopted.¹

There are no definitive records of lymphatic filariasis in Mexico, but at the beginning of the 20th century, a number of scientists gave anecdotal accounts of observations of elephantiasis in Mexico, especially along its coast, becoming less common towards the north. These authors include Clarac, Leboeuf, and Rigollet in 1913; Newman and Mayer in 1914, and A.J. Smith in 1917.²⁻⁴ At present, there is no

evidence of the transmission of *Wuchereria bancrofti* in Mexico.

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THE EARLIEST TRACES of human habitation in Nicaragua are the 10,000-year-old "Footprints of the Acahualinca," preserved under layers of volcanic ash of people and animals near Lago de Managua.¹ Around the 10th century A.D., indigenous people from Mexico migrated to the Pacific lowlands of Nicaragua.¹ During the 15th century, the Aztecs moved south from the Pacific lowlands to establish a trading colony, where the Aztec culture was adopted by many Indians.¹ Nicaragua takes its name from "*Nicarao*," chief of the indigenous tribe, who was living around present-day Lake Nicaragua.² In 1502, Christopher Columbus came upon this area, when he sailed down the Caribbean coast.¹ In 1522, a Spanish exploratory mission reached the southern shores of Lago de Nicaragua.¹ Within a few years (1524), the Spanish colonized the region and founded the cities of Granada on Lake Nicaragua and León, east of Lake Managua.^{1,2} Granada became a comparatively rich colonial city, while León became a hotbed of liberalism.¹ The inhabitants of the heavily populated area around Managua put up a fierce resistance against the Spanish invaders, but their city still was destroyed.¹ For the next 3 centuries, Managua remained underdeveloped.¹

On September 15, 1821, along with the rest of Central America, Nicaragua gained its independence from Spain.^{1,2} It was part of Mexico for a brief time, then part of the Central American Federation (or United Provinces of Central America).^{1,2} Finally, in 1838, it achieved complete independence.^{1,2}

Soon after, Britain and the United States both became extremely interested in Nicaragua and the strategically important Río San Juan navigable passage from Lago de Nicaragua to the Caribbean.¹ In 1848, the British seized the port at the mouth of the Río San Juan on the Caribbean coast and renamed it Greytown.¹ This became a major transit point for hopefuls looking for the quickest route to Californian gold.^{1,3}

The earliest report of lymphatic filariasis comes from Bluefields on the Caribbean coast, in the Región Autonomista Atlántico Sur, east of the capital, Managua.^{5,6} In 1908, William F. Thornton examined a worker from a nearby banana plantation in Bluefields.⁴ He had lived along the coastal region of

Costa Rica for more than 1 year before moving to Nicaragua.⁴ The patient was found to be microfilaria-positive.⁴

In 1912, 1913, and 1914, several articles referred to the occurrence of lymphatic filariasis in Nicaragua.⁷⁻¹¹

In 1944, a survey party for the Inter-American Highway reported that two persons residing near Jinotepe and Diriamba in the Department of Carrazo had elephantiasis and were hospitalized for the condition.³ These two towns are on the west coast of Nicaragua along the Pacific Ocean, just south of Managua, which is in the Department of Managua.⁶

On July 19, 1945, 15 persons from the Jinotepe and Diriamba area, who had elephantiasis of the legs, were screened for microfilaremia.³ Of these, only one (6.7%) was microfilaria-positive.³ During the survey, police working in this area reported that they had recently seen two other persons with elephantiasis of the scrotum.³

In 1954, while conducting a study on the Miskito coast of northeastern Nicaragua along the Caribbean Sea, W. Müller stated that "*W. bancrofti* is apparently absent" in Nicaragua.¹²

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SABA AND SAINT EUSTATIUS, together with Saint Martin/Sint Maarten are part of the Netherlands Antilles and are called the Netherlands Windward Islands.¹

Saba

Although Christopher Columbus first came upon this island during his second voyage in 1493, no settlements were established.² In 1595, Sir Francis Drake sighted Saba, as did the Dutchmen Pieter Schouten in 1624, and Piet Heyn in 1626.² In 1632, some shipwrecked Englishmen landed here and found it inhabited.² In 1635, the French claimed it, but in the 1640s, the Dutch settled on Saba, building settlements at Tent Bay and The Bottom.² However, it was not until 1816 that the island became definitively Dutch, the interval being marked by 12 changes in sovereignty, with the English, Dutch, French, and Spanish all claiming possession.²

Saint Eustatius (Statia)

The original inhabitants of Saint Eustatius were the Caribs, as shown by evidence of artifacts of their occupation, which date back to 300 A.D.³ Although Christopher Columbus first came upon this island during his second voyage in 1493, no settlements were established.³ He named the island "*Statia*" which comes from "Saint Anastasia," but the Dutch later changed it to Sint Eustatius.³

In 1636, the Dutch first colonized Saint Eustatius and built Fort Oranje, but the island changed sovereignty 22 times before finally remaining Dutch, in 1816.³ The island reached a peak of prosperity in the 18th century, when the development of commerce brought about 8,000 people to the tiny island, over half of whom were slaves.³ Around 3,500 ships visited the port each year; trading in sugar, tobacco, and cotton proved more profitable than trying to grow them.³ The slave trade was particularly lucrative, and thus, the island gained the nickname of "The Golden Rock."³

During the U.S. Revolutionary War, Statia was a major trans-shipment point for arms and supplies from France for George Washington's troops,⁴ which

were stored in the yellow ballast brick warehouses built all along the Bay, and then taken by blockade runners to Boston, New York, and Charleston.³ In 1781, the English retaliated by taking the port without a shot being fired by troops under Admiral George Brydges Rodney, who captured 150 merchant ships and £5 million of booty before being expelled by the French the following year.³

With continuing transfers of power, the economy never recovered, many merchants were banished, and the population began a steady decline.³ The emancipation of slaves in 1863 brought an end to any surviving plantation agriculture and the remaining inhabitants were reduced to subsistence farming and dependency upon remittances from relatives abroad.³ Prosperity has returned only recently with the advent of tourism and the island is still relatively underdeveloped.³

Lymphatic filariasis in Saba and Saint Eustatius

The earliest records of cases of lymphatic filariasis and in Saba and Saint Eustatius are from government reports which begin in 1875 and continued until 1946.⁴ No details of the cases or descriptions of signs or symptoms are given, and there appears to be have been no convincing evidence of autochthonous cases.⁴

From 1923 to 1927, J.B. Emanuels collected *Aedes aegypti* and possibly *Culex fatigans* from Saba and Saint Martin/Sint Maarten.⁴ In October 1941, H. Spitzer collected mosquitoes that Edward Van der Kuyp identified as *A. aegypti* Linnaeus and *C. fatigans* Wiedemann.⁴ In April 1947, Van der Kuyp collected mosquitoes from houses and yards in Saba, Saint Eustatius, and Saint Martin/Sint Maarten.⁴ *A. aegypti* Linnaeus and *Culex quinquefasciatus* Say were the most common mosquitoes collected.⁴

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SAINT MARTIN/SINT MAARTEN, together with Saba and Saint Eustatius, are part of the Netherlands Antilles and are called the Netherlands Windward Islands.¹ The Caribs were the original inhabitants of Saint Martin/Sint Maarten and named the island "*Sualouiga*" meaning "land of salt."² There is some controversy as to whether Columbus ever came upon Saint Martin/Sint Maarten, during his second voyage in 1493.² Some claim it was *Nevis* he named "*Saint Martin of Tours*," and that later Spanish explorers misinterpreted his maps.² Nevertheless, the Spanish established no settlements here.²

It was not until 1629 that some French colonists arrived in the north, and then in 1631, the Dutch came to its southern part because of the salt ponds.² By this time, there were no Caribs left on the island, and the two nations amicably occupied the island until Spain reconsidered and took over Saint Martin/Sint Maarten from 1633 to 1648, fending off an attack by Peter Stuyvesant in 1644, which cost him his leg.^{2,3}

When the Spanish left, Dutch and French settlers returned and after a few territorial skirmishes, they divided the island among themselves, with the signing of the March 23, 1648 "Treaty of Mount Concordia."² Popular legend has it that the division was settled with a race starting from Oyster Pond.² The Frenchman went north and the Dutchman went south, but the Frenchman walked faster because he drank only wine while the Dutchman's penchant for Genever (a drink similar to gin) slowed him down.² Since 1648, however, Saint Martin/Sint Maarten has changed hands 16 times, including brief occupations by the British, but the Dutch-French accord has been peaceably honored, at least since it was last revised in 1839.²

At the height of its colonial period, sugarcane and livestock were the main agricultural activities, although the poor soil and lack of rain meant they were not very profitable.² Emancipation in 1863 broke up the plantation system and the population began to decline as ex-slaves left to look for work elsewhere.² Most of the salt produced from the Great Salt Pond behind Philipsburg was exported to the United States and neighboring islands, but by 1949, this industry had also ended and a further exodus to other islands took place.⁴ The remaining population survived on

subsistence farming, fishing and remittances from relatives abroad.⁴

From 1923 to 1927, J.B. Emanuels collected *Aedes aegypti* and possibly *Culex fatigans* from Saba and Saint Martin/Sint Maarten.⁴ In October 1941, H. Spitzer collected mosquitoes that Edward Van der Kuyp identified as *A. aegypti* Linnaeus and *C. fatigans* Wiedemann.⁴ In April 1947, Van der Kuyp collected mosquitoes from houses and yards in Saba, Saint Eustatius, and Saint Martin/Sint Maarten.⁴ *A. aegypti* Linnaeus and *Culex quinquefasciatus* Say were the most common mosquitoes collected.⁴

From 1941 to 1943, Edward Van der Kuyp conducted a survey of 39 persons in Curaçao (34 adults and five children).⁴ Of the four persons (10.3%) who had microfilariæ, one adult had resided in Curaçao for a substantial period of time, in addition to living in Saba and Saint Martin/Sint Martin.⁴ Of the other two microfilaremic adults, one had arrived from Saint Martin/Sint Martin 20 days before the survey, and the other had lived in Guadeloupe.⁴ The microfilaria-positive child had arrived from Saint Martin/Sint Martin 10 days before the survey.⁴ Thus, two of the four persons infected with *Wuchereria bancrofti* may have acquired the infection in Saint Martin/Sint Martin.⁴

Of the three islands that make up the Netherlands Antilles, Saint Martin/Sint Martin appears to be the only one that had some possible evidence of transmission.

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PART 6

Countries with Imported Cases but No Apparent Transmission

THE EARLIEST KNOWN human artifacts in Canada are found in the Bluefish Caves in northern Yukon, which date to some 20,000 years ago (circa 18,000 B.C.).¹ The Vikings (Norse) arrived in 1100 A.D. and established settlements in Anse aux Meadows in Newfoundland, on the east coast of Canada.¹ There is archeologic evidence that from 1300 to 1500, the Saint Lawrence Iroquoians once resided in the area of current Southwestern Québec and Ontario.¹

In 1534, Jacques Cartier, the first French explorer in Canada, referred to this area as Canada, from the Iroquois word "*kanata*" meaning "town or cluster of dwellings."^{1,2} Other reports have it that early Spanish or Portuguese explorers, disappointed in not finding gold or other riches, derided the country as "*aca nada*" or "*cà nada*" meaning "here nothing."²

Yet others have ascribed the word to Latin or Sanskrit.² The weight of opinion to date, however, favors the aboriginal origin noted in Cartier's report.² Whatever the source, the Constitution Act of 1867 declared that the name of the new dominion would be Canada.²

Between 1900 and 1971, 11 cases of lymphatic filariasis were reported from Canada (Table 1).³⁻¹² All of these cases occurred in immigrants, visitors, or travelers returning from filariasis-endemic areas.^{3,5-8,10-14} There is no evidence of *Wuchereria bancrofti* transmission to Canadian nationals from these imported cases.¹² This lack of transmission has been attributed to the absence of appropriate mosquito vectors and the small number of infected persons who could serve as sources of transmission within Canada.¹²

Table 1. Cases of suspected lymphatic filariasis reported in Canada, 1900-1971

Date	Location where patient was evaluated	Country of origin/ patient characteristics	Clinical features	Results of laboratory examinations	Treatment	Author and year of publication
1900	Toronto General Hospital, Toronto, Ontario	Barbados; 47-year-old male, lived in Barbados since birth except for 6 to 8 trips to Canada and Europe	Swelling of the scrotum; hydrocele for 19 years	Microfilariae found in night blood smear; an adult worm found in tissue excised from scrotum	Removal of the affected scrotal tissue through surgery	A. Primrose, 1905 ³
1912	Royal Victoria Hospital, McGill University, Montreal, Québec	Antigua; 14-year-old female lived in Antigua since birth, moved to Montreal 5 months previously	Swelling in groin for 2 years	Microfilariae found in night blood smear; streptococci obtained from blood culture	Removal of the enlarged lymphglands on each side; died a year later from bacterial complications of surgery	G.E. Armstrong and E. J. Mullally, 1914 ⁴
1954	Shaughnessy (D. V. A.) Hospital, Vancouver, British Columbia	Canada; 22-year-old male military veteran lived in Canada except for a 14-month service in Korea, 1952-1953	Sore throat for several days' duration; enlarged axillary lymph nodes	Eosinophilia 72%; presence of microfilariae on histological examination of excised lymph nodes		J.C. Colbeck and J. P. Gofton, 1955 ⁵
1954	Out-Patient Clinic, Toronto General Hospital, Ontario	Guyana; 34-year-old male, lived in Guyana since birth except for this trip to Canada	Asymptomatic, had no complaints; microfilariae were detected when patient underwent routine examination for emigration to Canada	Microfilariae found on routine blood examination 2 months before emigration, and found again in Canada on 2nd exam by immigration authorities	Had been treated with DEC for 3 weeks in Guyana; refused further treatment in Toronto	M.M. Lenczner and T. Owen, 1960 ⁶
1959	Humber Memorial Hospital, Toronto, Ontario	Ireland; 26-year-old male, civil engineer, came to Canada in 1954, where he resided ever since, except for a 7-month trip to Nigeria in 1957	Swelling of left arm involving fingers and shoulders	Eosinophilia 65%; microfilariae found in night blood smears	Elastic band-aging for 1 week; DEC for 3 weeks	M.M. Lenczner and T. Owen, 1960 ⁶

Table 1. (Cont.) Cases of suspected lymphatic filariasis reported in Canada, 1900-1971

Date	Location where patient was evaluated	Country of origin/ patient characteristics	Clinical features	Results of laboratory examinations	Treatment	Author and year of publication
1964	Montreal General Hospital, Montreal, Québec	Guyana; 55-year-old female immigrated to Canada 5 years previously	Painless small lump in left breast	Biopsy specimen showed fragments of adult worms on histologic examination; marked eosinophilia; microfilariae found in night blood smears	None reported	M. J. Miller and S. Moore, 1965 ⁸
1967	Division of Laboratories, Provincial Health Branch, TPDRS, Vancouver, British Columbia	Ghana; 29-year-old male	Fever and itchy thighs; had been treated with DEC before leaving Ghana when microfilariae were found in night blood smears	Microfilariae found in night blood smears in Ghana; eosinophilia 39% in Vancouver	A full course of DEC was given in Canada	E. J. Bowmer, 1973 ¹²
1967	Toronto General Hospital, Ontario	Madras, India; 30-year-old male	Discomfort and appearance of a small mass on right side of scrotum	On histology, marked eosinophilia and fragments resembling adult worms; microfilariae found in night blood smears	Treated with one course of DEC	M.M. Lenczner, et al, 1968 ¹³
1970	Division of Laboratories, Provincial Health Branch, TPDRS, Vancouver, British Columbia	China; 32-year-old female, immigrated to Canada in 1956, returned to Hong Kong for a 10-week vacation in 1969	Small lump in neck	Excised lymph node revealed structures resembling filaria; similar findings in lump from left side of neck in August 1972; diagnosis of suspected filarial cervical adenitis made in October 1972	None reported	E.J. Bowmer, 1973 ¹²
1971	Saint Joseph's Hospital, Hamilton, Ontario	Bombay, India; 24-year-old female, lived in Bombay except for visit to North Africa; moved to Hamilton, Ontario 3 years previously	Painful micturition with the appearance of milky urine	Microfilariae found in night blood smears	Treated with two 14-day courses of DEC	Kinsey, E., E. K. M. Smith, et al, 1971 ⁹
1971	Centre Hospitalier de l'Université Laval, Québec	Guyana; 61-year-old male naval marine served in the Pacific	Left inguinal hernia	Eosinophilia 21%; biopsy of lymph node revealed microfilariae	Given a prescription for "appropriate treatment"	M. Tremblay, 1971 ¹⁰

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THE ORIGINAL INHABITANTS of Chile included Aymara farmers in the desert north, who cultivated maize and tended flocks of llamas and alpacas; fisherfolk in the coastal areas; Diaguita Indians in the mountainous interior; Araucanian Indians in the center and south, whose fishing and agricultural settlements were barely touched by Incan incursions; and numerous groups of archipelagic hunters and fishers in the remote south.¹

In 1494, all territory west of Brazil was granted to Spain by the Spanish-Portuguese treaty.¹ The Spanish assigned the task of conquering Chile to Pedro de Valdivia, whose expedition reached Chile's fertile Mapocho Valley in 1541.¹ Santiago was founded in the same year, with the cities of La Serena, Valparaíso, Concepción, Valdivia, and Villarrica following soon after.¹ The Río Biobío marked the southern extent of Spanish incursions, where they were barred by the resistance of the fierce Mapuche tribes.¹ Valdivia rewarded his followers with enormous land grants, which resembled the great feudal estates of his Spanish homeland.¹

Although mining and business outstripped agriculture as Chile's merchant megaliths, it was the social structure of the estates that shaped colonial Chile.¹ The native population was devastated by the unwitting introduction of infectious diseases, and the mestizo population, the descendants of Spanish and Indian unions, were used as tenant laborers on these huge estates, many of which were still intact in the 1960s.¹

By the 1820s, the cumbersome methods by which taxation was extracted by a stagnant and complacent Spain allowed a flowering pan-American identity to blossom into a push for full independence.¹ Simón Bolívar and José de San Martín led armies of freedom fighters from Venezuela to Peru, and from Argentina into Chile.¹ Bernardo O'Higgins, son of an Irish immigrant and erstwhile viceroy of Peru, became supreme director of the new Chilean republic.¹

The newly independent Chile was a fraction of its eventual size, consisting of Santiago and Concepción, and with fuzzy borders with Bolivia and Argentina.¹ The coming of the railways and military triumphs over Peru and Bolivia in the War of the Pacific (1879-83) incorporated the mineral-rich

Atacama desert to the north and the southern temperate territories.¹ Chile quickly achieved a degree of political stability and relative democracy, enabling rapid agricultural development and the advancement of mining, industry, and commerce.¹ The now empowered working class and the nouveau riche both challenged the political power of the landowning oligarchy in a brief but bloody civil war in the 1890s.¹

There are no definitive records of cases of lymphatic filariasis in Chile, but in mid-August 1950, a 60-year-old Palestinian woman was examined by Moisés Agosín in Santiago.² The patient, who was visiting relatives in Chile, had lived in Port-au-Prince, Haïti for 9 years and was subsequently living in Cartagena, Colombia, where she had already lived for 11 years.² She had symmetrical erythema, desquamation and non-pitting edema of a moderate degree that involved both forearms and elbows.² Both legs and feet demonstrated similar elephantoid type of edema.² A blood examination revealed microfilariae, for which she was given 50-mg diethylcarbamazine (DEC) for 7 days.² Post-treatment examination was not conducted, since the patient returned shortly to Colombia.²

References

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2. Faust EC, Agosín M, García-Laverde A, Sayad WY, Johnson VM, Murray NA. Unusual findings of filarial infections in man. *Am J Trop Med Hyg* 1952; 1(2):239-49.

PART 7

Countries with No Documented Cases or Transmission

We have no records, reports or evidence of either transmission or cases of lymphatic filariasis, whether imported or autochthonous, in the following countries.

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| • Anguilla | • Paraguay |
| • Argentina | • Peru |
| • Bermuda | • Saint Barthélemy |
| • Bolivia | • Saint Pierre de Miquelon |
| • British Virgin Islands | • The Cayman Islands |
| • Ecuador | • The Islands of the Bahamas |
| • El Salvador | • Turks and Caicos Islands |
| • Greenland | • Uruguay |
| • Guatemala | |
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