Leprosy control and contagionism in Suriname

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Abstract

Leprosy is nowadays a disappearing but not yet defeated disease in Suriname. In colonial times it was a burden for colonial government and people, the majority of patients (in pre-abolition times) being slaves. In the 18th century a control system was established, with detection and isolation, anchored in legislation, as major methods. Dutch physicians working in Suriname in the 18th and first half of the 19th century proposed contingent contagionistic models, according to which leprosy was caused by a mixture of factors, infection being one of them. But in the first half of the 19th century European researchers generally denied infection as the cause of leprosy and the paradigm of anti-contagionism prevailed, considering heredity and environmental factors as its cause. At the same time in Suriname - because leprosy appeared uncontrollable - the fight against the disease was reinforced by promulgating more relentless laws to hunt and identify lepers. In line with this, the Suriname born Charles Louis Drognat-Landré defended the view (thesis Utrecht) that infection is the one and only cause of leprosy. His extreme contagionism was sharply rejected in The Netherlands, but then he published his ideas in French and so could reach the international scene and influence the Norwegian Hansen. The latter discovered the culpable micro-organism a few years later. We claim a correlation between the development of a typical Surinamese form of contagionism, the brutal leprosy control system and the autocratic, non-liberal (towards the slaves) political structure of the Dutch colony Suriname.

Key words: leprosy, contagionism, anticontagionism, legislation, slavery, Drognat-Landré

Introduction

The annual number of newly registered leprosy patients in Suriname shows a gradual decline over the past decades (Lai A Fat and Sabajo, 2003). The incidence (annual number of new patients per 10,000 inhabitants) dropped from about 5 cases in the 1970s to less than one case after 2008. This is caused mostly by a decrease in the number of paucibacillary cases. But the incidence of multibacillary cases remained, with about 0.7 cases, more or less stable over the past decades. So leprosy as a public health problem has not yet been defeated. Moreover, there are indications that with recent Brazilian immigration new leprosy cases are brought into Suriname. There is a historical irony in the fact that it was through migration as well that leprosy became endemic in Suriname. Mass migration during the African slave trade changed the disease ecology in Suriname dramatically from the 17th century onwards. A reservoir of leprosy got a free ride into Suriname and the disease became endemic. Ever since, leprosy has chastened the Surinamese society, becoming a heavy burden for people and government. Migration from the Asian continent in the late 19th and early 20th century introduced additional cases of leprosy. Probably all groups of people who came to Suriname from leprosy-endemic countries contributed to the leprosy reservoir (Menke and Niemel 2003, p. 23). This multiple origin of Surinamese leprosy is consistent with the findings of Monot et al. (2005), who studied the origin and worldwide spread (including the Caribbean region) of the disease, using comparative genomics. Suriname earned itself a name in the 20th century as a country with one of the highest leprosy rates in the world. This is illustrated with the information that in the 1920s an estimated 1% of the population of Suriname and 2-3% of the population in the capital Paramaribo suffered from leprosy (Flu, 1928, p. 65).
From the 18th century onwards, the Dutch colonial government considered leprosy a serious public health problem and a threat to the plantation economy. Therefore, a rigorous leprosy control system was established using detection and isolation (anchored in legislation) as major methods. This control system culminated in the 19th century, with relentless legislation to hunt and isolate lepers, and with the opening of three new leprosaria (Menke and Niemel, 2003, pp. 25-26).

The idea that leprosy is infectious is very old, going back to medieval and even Biblical times. However, in the 19th century, this concept was heavily criticized. The epidemiological question of how diseases that scourged Europe in those days, such as tuberculosis and cholera, were spread into and within populations was extensively debated. At the extremes of the discussion stood so-called contagionists and anti-contagionists, opposing each other (Ackerknecht, 1948; Worboys, 2000, pp. 1-43). The former group maintained that the prevention and control of epidemic diseases should be based on quarantine and isolation. The anti-contagionists were defined by their opposition to a singular focus on contagion to prevent and control epidemic diseases. However, most physicians did not fall neatly into either camp and they could be referred to as contingent-contagionists. The same debate can be identified in international medical writings about leprosy control, with contagionists, anti-contagionists and contingent contagionists defending their points of view. The latter adhered to different ideas, which encompassed earlier 18th-century conceptions about leprosy, in which the cause of the disease was seen as contagion with a ‘poison’ (transmitted by humans), but in which a weak(ened) constitution was necessary for the outbreak of the disease.

In the 1850s and 1860s the main hypotheses about leprosy came from three anti-contagionist schools of thought: hereditarian, sanitarian, and dietarian, the international scientific debate being dominated by Norwegian and British researchers (Pandya, 1998, p. 374; Robertson, 2003). Knowledge about leprosy and other diseases, gathered by Dutch physicians working overseas (in the West Indies as well as the East Indies) was considered important in scientific circles in the Netherlands, giving rise to PhD dissertations and other publications.7 In the 1860s, a concept (defended by a physician named Charles Louis Drognet-Landré) that originated in the by then declining colony Suriname (compared to the Dutch East Indies), influenced the course of the European leprosy debate and research (Drognet-Landré, 1867; 1869). The work of Drognet-Landré has received little attention in modern leprosy historiography until recently (Menke et al., 2007; 2009; 2010).

In this preliminary paper we shall describe the Surinamese leprosy control system, with emphasis on the 19th century, within the framework of: a) the social/economic context of the slave colony, and b) the scientific concepts about the cause of the disease. We shall pay special attention to the opposing view of Drognet-Landré, and the way he influenced European debate and research; we shall also demonstrate that the knowledge about leprosy, originating in Suriname, encountered serious opposition in The Netherlands. Finally, the possible connection between the Surinamese colonial political system, the leprosy control strategy and the scientific conceptualization regarding the cause of leprosy will be discussed in brief. This paper is largely based on a study of primary and secondary sources that were published on this subject together with an analysis of the leprosy laws of the colony Suriname and to a limited extent a research of original archival sources.

**Economy, slavery, and disease**

In the year 1667, Suriname definitively fell into the hands of The Netherlands in ‘exchange’ for what was to become New York, as part of the Breda Peace Treaty with the British. The colony on the ‘Wild Coast’ became the centre of a modest West Indian empire (den Heijer, 2002, p. 140). In spite of a chronic shortage of African slaves - a ‘limited’ annual supply of about 2000 slaves from the West Indian Company meant a high price per slave - the economy was booming. The export of sugar, coffee, tobacco, and wood increased tremendously, with, at the same time, the number of plantations increasing from approximately 25 in 1667 to 400 a century later, and the number of slaves increasing from a few thousand to sixty thousand. But Suriname proved to be an unhealthy environment for the newcomers, European settlers as well as African slaves, who were troubled by a variety of disorders. Intestinal, venereal, lung, and eye diseases as well as various skin disorders occurred frequently, including the very contagious ‘jas’, ‘jaas’, or ‘jaws’ (*framboesia tropica*), a devastating tropical illness. The death rate of the slaves on the plantations showed a steep increase, estimated at over 40 per 1000 inhabitants; thus, Suriname had an infamous reputation among the Caribbean plantation colonies (Oostindie, 1989, pp.1, 131). Maintaining the number of slaves needed on plantations was a continuous worry for the planters. It was in their own and utmost interest to deploy their slaves as long and as efficiently as possible. But to maintain high productivity, excellent health of the slaves was a prerequisite.
Leprosy legislation

The first attempts in the 18th century to get the ‘jaas or other inconveniences and disabilities’ in Suriname under control through legislation, were apparently meant to prevent infection of the white settlers. In the year 1728, the Surinamese government took the first legal measures against infectious diseases, based on strong suspicions that the slaves were a significant source of infection (Schiltkamp en de Smidt, 1973, pp. 395-396). Thus, it was legally proclaimed that a slave suffering from a contagious disease was not allowed to be on a public road because of the health risk for elderly people and especially innocent white children. The owner of the slave risked a penalty for breaking this rule of law. In practice, however, the rule appeared to be ineffective in controlling disease. Therefore, more stringent laws were needed. Leprosy was first mentioned in historical texts in Suriname in 1759 (Oostindie, 1989 pp.140-141). With the revision in 1761, of the above-mentioned law, ‘boasie’ (leprosy) was mentioned for the first time in a legal ordinance (Schiltkamp en de Smidt, 1973, pp. 707-708). Higher penalties were imposed on slave owners for ignoring the health regulations concerning contagious diseases. Leprosy was now definitely recognized as a serious and important disease. Yet it was not until 1790 when new and stricter rules, including the isolation of lepers, were propagated to control leprosy (Schiltkamp en de Smidt, 1973, pp. 1144-1147). In that year, the government decreed that the plantation Voorzorg on the Saramacca River was to serve as a location for the isolation of slaves and ‘free mulattoes and negroes’ suffering from leprosy. White residents who suffered from this disease were allowed to stay in their own houses, and they were permitted to have contact with others only if strictly necessary. Another article of this law (modified in 1791 and 1792) was meant to prevent newcomers with leprosy from spreading the disease: slaves who had just arrived from Africa and were diagnosed with boasie by the surgeon who inspected the ship, had to be taken into custody, and if they were not shipped back, they were transported to Voorzorg (Schiltkamp en de Smidt, 1973, pp. 1159-1160 and pp. 1167-1168). But again, in spite of these rules, leprosy was not to be defeated.

In the year 1823, the Government concluded that Voorzorg had to be abandoned as a leprosy colony, because of its close vicinity to plantations and to the only town, Paramaribo. By decree, the lepers were transported to the remote plantation of Batavia, situated on the right bank of the Coppenname River, much further away from Paramaribo. However, the deportation was not an easy task. The leprous slaves resisted to the very end, despite the fact that their huts were set on fire (Klinkers, 2003, p 51). It was not until the year 1831 that the legal obligation became effective to intern in Batavia all the slaves and a part of the free population, viz. those who were infected with leprosy. This was based on a new law, in fact a profound revision of the earlier laws concerning leprosy (Gouvernements Blad, 1830). According to this new law, slave owners were also obliged to report to the authorities, slaves who were suspected of suffering from leprosy. Anyone who did not do so could be punished with a fine of 200 guilders for each infected slave. Besides, an ‘informer’s system’ of secretive reporting was formally introduced: a premium for reporting suspected cases and a penalty for wilful lacking to report. A special leprosy commission, called ‘Commissie tot onderzoek naar de ziekte der Melautschheid en besmettelijke Elephantiasis’ decided, after an elaborate examination, whether a slave was infected or not. This commission was composed of six members, among them the Stads Genesheer (Town Medical Doctor) and the Stads Chirurgijn (Town Surgeon). If a slave was found to be infected, then his final destination was Batavia. In case of doubt, the slave was categorized as ‘suspect’ and was allowed to return to his owner under the condition that he not set foot in the public domain. After one year, an obligatory second examination was performed. Annually, thirty to fifty slaves were found to be infected, with a peak of 194 cases in 1831, the year of introduction of the law. For ‘free’ persons the same categorization prevailed, but only when a ‘free’ person with leprosy disobeyed the rules of ‘confinement’ by going outside the house into the public domain; or in case of housing problems, the authorities could decide on deportation to Batavia. The brutal consequences of the leprosy laws of 1830 are further demonstrated by pointing out that a slave who was found to be ‘suspect’ by the commission and was seen on a public road was given a cane beating as punishment; a free resident who was ‘suspect’ and seen in public was reported as such in the newspapers. Not only was a house search conducted on suspicion of harbouring an infected person, it was a formal procedure to systematically search all houses in Paramaribo once every three years. Yet one could wonder how far the legal arm actually reached in daily practice.

The ratio between ‘free’ and ‘non-free’ in Batavia was about 1:25, for a total population of 486 inhabitants in the year 1847 (ten Hove, 2003, pp. 42-43). Assuming that the number of inhabitants of Suriname was approximately 50,000, this meant that seventeen years after the introduction of the law slightly less than 1% of the population was kept in isolation at Batavia. At first sight, the total of 2,813 examined cases that were suspected of being infected with leprosy in a period
of fifteen years (since 1831) and the 1,289 persons found to be actually infected, seems to be impressive (Duchassaing, 1858). But further sharpening of legal measures in 1845 and again in 1855, leaves serious doubt as to the effectiveness of the legal measures taken by the government (Gouvernementsblad, 1845; 1855). The abolition of slavery in 1863 did not bring about much change in the severe policy of fighting leprosy. Fourteen years later, the renowned English leprosy expert, G. Milroy, characterized Suriname as an example of a country addressing leprosy in a barbaric way, and thus not deserving of replication: “They [the leprosy patients] are treated as outcasts, being expelled and rigorously excluded (for the rest of their lives) from society, deprived not only of personal liberty but also of sundry civil rights” (Edmond, 2006, p. 55). By the way, Milroy’s view was not surprising, as he was a fierce opponent of the isolation policy for leprosy patients.

From the above it is clear that the fight against leprosy was not an easy task. To implement the leprosy laws, the authorities had to rely on the collaboration of the entire community. Both economically and socially there were conflicting interests on an individual and a collective level. Just as in British Guiana and other parts of the Caribbean, stigmatization and myth-building around leprosy as well as the intrinsic tension between deterrence and compassion, played an important role in coping with the disease (Edmond, 2006 pp. 1-23). Leprosy patients were considered by the Creole population to be impure, sinful, and/or bearing a curse (‘kunu’) (Klinkers, 2003). The patients, irrespective of their ethnicity, were considered a threat to the entire community, and to protect cleanliness and moral order, they were excluded from social intercourse. At the same time, care for lepers was seen as the highest form of cleanliness and was appointed a member of the Leprosy Commission. As such he assisted in the enforcement of the Surinamese leprosy tracing and

From the foregoing it can be concluded that in the 19th century Suriname was gradually toughening its policy of coping with leprosy; a policy imposed and executed by a government which consistently believed that the disease was contagious. With this policy the colonial government in fact seemed to ignore the complexity of the scientific concepts about the cause of leprosy proposed by physicians, including those (see next paragraph) who had worked in Suriname and formulated contingent contagionistic models in the 18th century and the first half of the 19th century.

The origin of a Dutch West Indian or Surinamese leprosy contagionism

Ideas based on observations in Suriname played a major role in the Dutch scientific debate on the cause of leprosy in the 19th century. The authoritative book on leprosy by the Norwegians Danielssen and Boeck (1848), defending the thesis that leprosy was a non-infectious, hereditary disease, was discussed in the Dutch medical literature by Israëls (1857, pp. 162-166). According to this influential professor of epidemiology and medical history at the University of Amsterdam, the idea of leprosy being infectious was not necessarily at odds with the principle of heredity, proposed by the Norwegians as the cause of leprosy. Israëls suggested the unifying view that infection, together with environmental factors and predisposition, caused the disease. In formulating this concept, he continued in the tradition of physicians who had worked for the colonial authorities in Suriname: P. Fermin (1864) and G. W. Schilling (1771) in the 18th century, and A. van Hasselaar (1835) and J. P. ter Beek (1841) in the 19th century. These investigators described the cause of leprosy in terms of all sorts of mixtures of infection, heredity and sanitation, fitting in the concept of contingent contagionism. Indeed, Fermin and Schilling can be considered the physicians who in the 18th century laid the foundations for a ‘Dutch West Indian or Surinamese leprosy contagionism’. This ‘malleable contagionism’ of the 18th century and first half of the 19th century was also encountered in the work of the physician Charles Landré. He summarized his findings as follows: “The disease can spread through infection. Europeans who - due to the conditions in which they generally live here - remain more than others, free from infection and they are rarely affected by leprosy; they are probably less predisposed to this disease” (Duchassaing, 1858). Charles Landré was a Dutch medical doctor of Huguenot ancestry, who emigrated in 1840 from Amsterdam to Suriname. He held a position as First Town Medical Doctor and was appointed a member of the Leprosy Commission. As such he assisted in the enforcement of the Surinamese leprosy tracing and
isolation strategy. The life of the Landré family was strongly intertwined with leprosy. One son (with the first name Drognat) developed leprosy and another son (with the first name Charles Louis) studied medicine and published on the cause of leprosy. Charles Landré, who at first was a contingent-contagionist, would modify his view regarding the cause of leprosy in the direction of pure contagionism (Menke et al., 2009).

A sharp debate between contagionists and anti-contagionists

In the 1850s and 1860s, anti-contagionism had been the dominant scientific movement in the European debate on the cause of leprosy. In 1867, the young physician Charles Louis Drognat-Landré (Landré Jr.), born in Suriname, initiated a sharp scientific debate with his PhD thesis at the University of Utrecht, in which he defended the view that leprosy is a contagious disease (Drognat-Landré, 1867). In conjunction with this, he rejected the heredity concept of Danielssen and Boeck (1848); according to Landré Jr., the Norwegians drew an incorrect conclusion due to inaccurate interpretation of the data from the pedigrees they used to support their view. Landré Jr. based his opposing view (which he had constructed in close collaboration with his father), on the one hand on publications by the Dutch physicians Schilling (1871), Hasselaar (1835) and ter Beek (1841) who had worked in Suriname; on the other hand on his own observations. According to him, Europeans in the colony contracted the disease from African slaves or their descendants, who were infected with leprosy. Because of their isolated way of living, American Indians, however, rarely came into contact with Europeans and Africans and therefore remained practically free from leprosy. Landré Jr. expressed this observations in his thesis as follows (quote, translated from Dutch by the authors): “It is nearly impossible for Europeans in the colony to avoid contact with leprosy patients, and even though the affected have to eliminate themselves from society, this law is often violated, and therefore people are not often exposed to lepers, especially when the disease occurs with few signs; moreover, the majority of Europeans present in the colony mix with the female negro population. So contact between Europeans and lepers can invariably be demonstrated; but between American Indians and lepers hardly ever. Children of European parents who have never known leprosy among their ancestors are not infrequently infected by the disease; however, it can be demonstrated in the majority of cases that they, without their parents knowing, or because of ignorance, have been in contact with lepers or were breast fed by wet nurses suffering from leprosy. In these children - so it seems to me - the development of their disease can only be explained by contagion” (Drognat-Landré, 1867, pp. 74-75). Landré Jr. also rejected the idea - defended (among others) by Schönfeld (1857), - of the existence of two etiologically different types of leprosy: a European hereditary type and an overseas (colonial) contagious type. His view was that in all cases leprosy is an infectious disease, transmitted by contagion. Thus, Landré Jr. in fact abandoned the (multi-causal) etiological hybrid models (which included contagion) proposed by his Dutch predecessors who had worked in Suriname, in favour of a pure mono-causal contagion model.

However, the pure contagionism of Landré Jr. was rejected in The Netherlands. B. Carstens (1867), Inspector of Public Health in The Netherlands, defended with ardour the hereditary origin of leprosy in the same year, referring to the anti-contagionistic report of the British Royal College of Physicians (1867). Regarding Landré Jr.’s thesis, Carstens noted that despite the arrival in The Netherlands of leprosy patients from the colonies, no new cases of this disease had occurred, which could be considered to be an argument against contagion. Graver objections came from H.J. Vinkhuijzen (1868), physician and writer. He fuelled the debate with the provocative comment that Landré Jr.’s conclusions were of no value, because they were founded on an inaccurate interpretation of the observed epidemiological facts. According to Vinkhuijzen (quote, translated from Dutch by the authors) “Spreading of leprosy in Suriname was not the result of infection, but could simply be explained as being a combination of heredity and the specific living conditions such as the messy way of life of the Negro population and their unhealthy, unbalanced diet, together with the climatic conditions”.

With his challenging ideas about the cause of leprosy, Landré Jr. was completely ignored in The Netherlands, because his scientific position contradicted the prevailing paradigm based on heredity and sanitation. Although he defended a strong academic thesis, he was not taken seriously. However, he did not acknowledge defeat and tried to find an international audience. In 1869, he published a French monograph in Paris, in which he enforced his ideas with new facts, using the provocative title: “De la contagion, seule cause de la propagation de la lèpre” (Drognat-Landré, 1869).3 By presenting his theory of infection in French, Landré Jr. could now reach the European medical and scientific society (no longer facing a language barrier caused by using Dutch). The Norwegian physician G.A. Hansen, who worked with Danielssen at the leprosy hospital in Bergen, had his doubts about the Norwegian/British heredity concept. He studied Landré Jr.’s book and
found arguments and inspiration to elaborate on the idea of leprosy as an infectious disease (Harboe, 1973, p. 417). Within the framework of a profound analysis, he translated Landré Jr.’s twelve ‘keynote’ leprosy cases from French into Norwegian and published them in 1872 in a Norwegian medical journal (Hanssen, 1872, pp. 23-27). Cases in point are number 5 and 6: “The two elder of four daughters of a member of the court of justice developed leprosy and they died at the age of 19 and 22. The firstborn was breastfed by a Negress who developed serious signs of leprosy and later on died from this disease. The second girl was suckled by her mother. A few years later the girls were obliged to leave school, because the disease manifested itself in such a way that they had to be isolated immediately; thus unfortunately the girls were separated from their mother; afterwards it appeared that the first signs of the disease had already been present for a long time. The father of the children was Dutch. Their mother, born in the colony, was of European ancestry. They were of distinguished Dutch families and invariably led a comfortable life (Drognat-Landré, 1869, p. 52). According to Landré Jr., neither disposition nor environment could explain the development of leprosy in these patients. It could only be explained by direct physical contact with lepers. Hansen stated that it was Drognat-Landré’s book that opened his eyes to the fact that they (the Norwegians) had not paid enough attention to the issue of infection (Irgens, 1973, 193). Hansen continued his investigations in this direction and discovered the culpable micro-organism (Mycobacterium leprae) in 1873 (Vogelsang, 1978). Almost twenty five years later, at the first international leprosy conference in Berlin in 1897, the concept that leprosy is an infectious disease was generally accepted. At this meeting, Broes van Dort (1897), who represented The Netherlands, pointed out that in 1867, when leprosy anti-contagionism was at its summit, father and son Landré had been strong advocates of leprosy contagionism.12 And so in the last decade of the 19th century, with by then an international zeal in favor of leprosy contagionism, in Suriname the practice (prevailing ever since the 18th century) of chasing and isolation of leprosy patients, was continued and three new leprosaria (this paper, p. 171) opened their doors.

Discussion and conclusions

The Surinamese leprosy control system, started in the first half of the 18th century, and gradually became more relentless in the course of the 18th and 19th century. It is tempting to assume that the system of detecting and isolating people with leprosy was based on the concept of the disease being infectious (whether or not in addition to other etiological factors). But adherence to this concept of contagionism may not have been the only reason for the development of the prevailing system. The system might very well have been facilitated by the political structure of the slave society. The medical-historian E.H. Ackerknecht (1948) points out that there is a close connection between a nation’s political system and culture and the approach it takes to epidemic diseases, a correlation, in other words, between politics and prophylaxis. In Ackerknecht’s view, an ‘autocratic’ ideology favors a view of epidemic diseases as contagious, and consequently applies tactics of quarantine. In contrast, ‘liberals’ who resent state intervention, approach the issue from a variety of local perspectives, particularly social problems, and seek to prevent disease by correcting deficiencies of the environment through hygienic reform. Of course, as Baldwin (1999) has argued, Ackerknecht grossly underestimated the complexity of national responses to epidemics. Yet Baldwin (1999, p. 557) agreed that the political system roughly correlates with prophylactic measures. So Ackerknecht’s essay remains a cornerstone publication and can be considered to be a valuable starting point in examining the relationship between political structure and measures taken against diseases (Hamlin, 2009; Stern and Markel, 2009). The colonies in the West Indies, including Suriname, had an authoritarian system of government that did not care much about the rights of the non-white residents in particular. This autocratic political structure of the slave colony Suriname is therefore to be considered as an additional precondition to explain the leprosy control measures that were taken in this country.

The segregation of leprosy patients in Suriname was maintained throughout the 19th century, notwithstanding the fact that in Europe, including The Netherlands, the anti-contagionists had the upper hand in the debate and strongly opposed the use of quarantines and isolation. The Dutch East Indies, however, adhered to anti-contagionism and the leprosaria there were closed down (Sitanala, 1940). In the framework of our analysis, it is important to point out that the Dutch East Indies, in which the native population had a far larger extent of autonomy, seems to have been more inclined to less restrictive local health policies. This was quite a contrast with Suriname. The diverging developments of public health policies and theories regarding leprosy in the West and the East reflected the distinct characteristics of the societies and cultures, including the political system in which they evolved.

With this paper we have lifted only a corner of the veil that covers the history of leprosy in Suriname, which is inextricably bound up with the history of the country and its inhabitants. This is in

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line with the way in which the ‘biography of the disease’ is contextualized within the social, economic, and cultural structure of the society, and connected with the development of legislation and public health. It goes without saying that for a better comprehension and explanation of the historical dynamics and interrelationship of leprosy control, scientific concepts, and political system, a comparative investigation is required, comparing Suriname on the one hand with the Dutch East Indies (in the context of Dutch colonial history) and on the other hand with neighbouring countries in the Caribbean and South America.

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Endnotes

1. This paper is in part based on earlier papers by the authors (references: Menke et al. 2007, 2009 and 2010).

2. The leprosy incidence was roughly calculated from data provided by Leslie Sabajo, head of the Dermatological Service of Suriname, the subdivision of the Ministry of Public Health responsible for leprosy control.

3. Regarding the bacterial load and hence the infectiousness, two types of leprosy are recognized: paucibacillary and multibacillary leprosy. Patients with paucibacillary leprosy are non infectious or far less infectious than patients with the multibacillary type.

4. Information received from Leslie Sabajo; also see note 2.

5. Schilling in his 18th century academic thesis was (one of) the first to point to the African origin of Surinamese leprosy (Schilling 1771).

6. The presented view on the origin of leprosy in Suriname is partly based on hypotheses and indirect evidence. The exact origin of Surinamese leprosy requires additional research.

7. Regarding the West Indies, we refer to the dissertations of Schilling (1771), Hasselaar (1835), ter Beek (1841) and Drognat-Landré (1867).

8. The name of this commission in English reads: “Commission for examination of people in order to detect Leprosy and contagious Elephantiasis”.

9. According to Gussow (1989), an American anthropologist, missionarism is next to colonialism, contagionism and racism, one of the cornerstones of the construction, in the 19th century, of leprosy as a colonial tropical disease.

10. We have used the term “malleable contagionism”, which reads in Dutch as “kneedbaar contagionisme” (Menke et al 2007). “Malleable contagionism” can be seen as being identical with “contingent contagionism”.

11. In English this title reads: “On contagion, sole cause of the spread of leprosy”.

12. T. Broes van Dort was a leading Dutch dermatologist, who studied leprosy and published many papers and a book on this disease.