LEPROSY

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PREFATORY NOTE.

In the following work, which is a reprint from the *Edinburgh Medical Journal* (September 1876 to November 1879), I have endeavoured to show—1*st*, That the idea that Leprosy is increased by vaccination is groundless; 2*d*, That it is communicable; and, 3*d*, That its probable primary cause is a want of salt combined with a deficient vegetable diet. During these three years I have become less inclined to admit the influence of heredity in propagating the disease, and more inclined to look on human intercourse as the only means of propagation. My opinion as to the action of ammonium chloride on the liver (p. 69) is of course modified by Rutherford's experiments.

102 Earl Street, Manchester,
18th November 1879.

ERRATA

Page 25, line 2, *for* "sixteenth century," *read* "seventeenth

„ 47, line 32, *for* "richesse," *read* "richesses"

„ 54, line 27, *for* "5 in 1000" *read* "5 in 10,000"

„ 58, footnote 1, and p 60, footnote 10, *before* "Durand" *insert* 'Mare of

„ 91, line 29, *for* "inconsistent," *read* "inconstant"

ON THE

ETIOLOGY AND HISTORY OF LEPROSY.

Introductory.—Some years ago, while in St Kitts, my attention was called to the subject of leprosy, by the grave allegations made against vaccination by Dr Bakewell, of Trinidad, before the Vaccination Committee of the House of Commons. He stated[1] that leprosy was conveyed by vaccination, and that it was on the increase in Trinidad, and in Demerara, Barbadoes, and Jamaica, quoting the opinions of medical men in the several islands, as given in the Leprosy Report of the Royal College of Physicians (1867), in support of his extraordinary statements, and also that the increase was a direct consequence of the introduction of compulsory vaccination. As Dr Bakewell was at the time Vaccinator-General of Trinidad, and had been physician to the Leper Hospital there, his evidence was calculated to do more harm than such random statements by another man in a lower official position would have done. I therefore thought it worth while to show that he was wrong, and before I left St Kitts did all in my power to prepare myself for doing so. Since then the subject has occupied my attention more or less, although domestic affliction and other causes have delayed the publication of the present work.

In the first place, to dispose of Dr Bakewell's statements, I may say, 1st, he is contradicted by the very Report he refers to, at least in regard to Trinidad[2] and Barbadoes; while, in Jamaica, although two medical men then thought it on the increase, their opinion is contradicted by a comparison between the censuses of 1861 and 1871, showing that in 1861 there were 778 lepers among 441,264 of a population, while in 1871 there were only 749 in 506,154;[3] or in 1861, 1 in 567; in 1871, 1 in 676 of the population. In other islands it is held to be decreasing, as Tobago. Only in Demerara is it unanimously decided[4] (so far as *mere opinions* can decide such a matter) that it is on the increase; but this is distinctly attributed to the free intercourse of lepers with the healthy since 1838.

[1] Report, p. 213.
[2] Leprosy Report of the Royal College of Physicians, pp. 14, 33, 40.
[3] See Milroy's Rep. on Lep. and Yaws in the West Indies, 1873, p. 31.
[4] Coll. Phys. Rep., p. 47.

Immigration of coolies from India is also blamed for the increase; but I can hardly understand how the importation of picked labourers from a country with, so far as the census returns show, 1 leper in 1864[1] can *increase* the number of lepers in a country with 1 in 280.[2] Besides, Demerara is a badly vaccinated colony,[3] so how could vaccination increase leprosy in it?

2d, Dr Bakewell admitted that Trinidad was not well vaccinated,[4] only one-half of those born being protected in some districts, and in others none at all. *A fortiori*, a better vaccinated island would show a greater increase of leprosy, yet, in Basseterre, St Kitts, I found that from 1st January 1867 to 30th June 1871, the average living births were 280, the average vaccinations 137, the average age when vaccinated 18 months (calculated from 300 vaccinations). But I found that 98 children born died in the first 18 months, thus only 182 were left; so that of these, 137 being vaccinated, 45, or 25 per cent., were left unvaccinated under the ordinary operation of the Act of 1854. But in 1854, 504, and in 1862, 355, were vaccinated, so that the real number unvaccinated would be less than that given above.

In 1871, from examination of the schools in my district of Basseterre, I found 127 out of 555 without any marks of vaccination;[5] and from careful calculations, made from examination of old documents from 1849, as also of the inmates of the Cunnynghame Hospital, and of the Registrar's books from 1854 to 1870, making proper allowance, according to the St Kitts' rate of mortality, for those who had died in the interval, I concluded that about 6500 to 6700 in a population of 8417 were vaccinated. This of course was unsatisfactory as compared with Scotland with 96 per cent. vaccinated,[6] but is better than Dr Bakewell represents Trinidad to be. I may say that I have every reason to believe that the other districts of St Kitts have been almost, if not quite, as well vaccinated as Basseterre.

Yet in St Kitts, taking three periods, 1817, 1854, and 1872, I find that there has been both a relative and absolute decrease in the number of lepers since 1817.

In 1817, every slave was registered according to law; I found, by a careful search through these registers, and a reference to old slaves in every estate where there was any doubt, and also old estate-books, in which slaves registered as "sickly," "diseased," "useless," in the *public* registers, were entered "lepers," that there

[1] Calculated by myself from the census returns for India. As many lepers are doubtless omitted in these returns, 1 in 1500 would probably be nearer the truth.

[2] Coll. Phys. Rep., Appendix, pp. 48 and 214. [3] Milroy's Rep., p. 35.

[4] Vacc. Rep., pp. 208 and 215.

[5] This was an official examination made by order of the President, in consequence of my having represented to him that the Vaccination Act was not sufficiently operative.

[6] Calculated by me from the Registrar's books.

were in 1817, 95 lepers at least in 20,149, or 1 in 212; in 1855, the census returns showed 53 in 20,700, or 1 in 390 (no doubt under the real number, as a few would be omitted in taking the census); while in 1872, by special inquiries, aided by my colleagues, the clergymen, the police, and friends living in each district, I could only discover 72 in a population of 28,000, or 1 in 389.

But as it is a fact, which I have no doubt of, from information received from the late Dr Swanston (who began practice in St Kitts in 1815), confirmed by examination of old estate-books, and of a number of old slaves (only giving a percentage of under 10 per cent. unvaccinated), that the slave population *was better* vaccinated than that of the present day, leprosy ought to have increased most from 1817 to 1854, while the very contrary was the case. Indeed, had the strict seclusion so much insisted on by slaveowners been carried out to the present day, I firmly believe that there would have been a much greater decrease from 1854 than there has been.

Vaccination was introduced into St Kitts about 1815; I find it charged for in estate-books in 1819; but, besides that (what Dr Bakewell seemed to know nothing of), inoculation (in itself quite as much calculated to cause leprosy as vaccination) was practised in the colonies at the beginning of the century; I find it charged for by medical men in estate-books of 1805.

I think I have said enough to show the absurdity of the statement that leprosy has been increased in the West Indies by vaccination.

3d, Dr Bakewell admits (p. 212) that mosquitoes may convey the poison of leprosy; yet, because a child, the son of English parents, becomes a leper (p. 207), turns round on vaccination as the *only* explanation. But cases are numerous of Europeans and their children taking leprosy in the tropics when no such cause was possible.

In conclusion, I may say that in my inquiries in St Kitts, I made special notes of this point in every case, and never could see the least reason for believing that vaccination had anything to do with the conveyance of the disease.

I have said somewhat more than I intended on this point, but in Trinidad itself, the practical result of bad vaccination was seen in 1871, when several thousand deaths took place from smallpox, though, in 1867, Dr Bakewell thought it 'would hardly be worth the trouble" to vaccinate his own children there (p. 214), while in 1872, the island of St Kitts was exposed for five days to the infection—from a ship whose captain got pratique by saying that there had been no sickness on board—yet not one of the population took the disease. By that time all those found unvaccinated in 1871 had been protected.

I am thus anxious that all that lies in my power should be done to do away with the bad impressions likely to be created by such rash statements as Dr Bakewell's among those living in countries where leprosy is common. For other arguments against these statements, I may refer to Dr Milroy's Report, p. 32 *et seq.* I may

say, however, in conclusion, that in the case quoted by Dr Bakewell, in which vaccination is blamed, as given in the College of Physicians' Report, p. 235, by Professor Wilson, there is not one word about the native child (the vaccinifer) having been a leper, and leprosy did not appear till the child was eleven years of age.

Etiology and History.—I think it best to consider the history of leprosy along with its etiology, as I hold it to be a communicable disease from man to man, but that there must always be the greatest difficulty in proving such communicability from cases, from the great length of the period of incubation, extending from under two[1] to eight years.[2] Even cholera, with an incubative stage of a few hours, was long held to be non-communicable; and Pruner, one of the strongest opponents of the contagion of leprosy, would perhaps change his opinions now that the communicability of cholera is admitted, as he founds his opinion against the spread of leprosy over the world being a proof of contagion, on the supposition that cholera is non-contagious, and yet had so spread.[3] Now, I believe that a careful study of the history of leprosy, along with that of the commerce of all nations from the earliest periods of history, shows most irrefragably that leprosy is a communicable disease. It has always arisen in certain parts of the world within the tropics, and spread thence by communication to other parts; and the marked periods of spread have always followed, at an interval of one or two centuries, some great event, by which communication between a formerly infected and a formerly uninfected country was thrown open.

The earliest reference I can find to leprosy is in the Histoire d'Egypt by Henri Brugsch,[4] in which, from a great medical papyrus discovered at Memphis, is mentioned a passage which contains a number of receipts for the cure of diseases of the nature of leprosy (du genre de la lèpre). "This manuscript," says Brugsch, "was composed during the reign of Rameses II. (1350 B.C.), but there is a passage in it which throws back a part of the work to the fifth king of the table of Abydos." The text (of the papyrus) says, "This is the beginning of a collection of receipts for curing the exanthemata (uχet)." Following the quotation, Brugsch shows that it is as ancient as the reign of Husapti, the fifth king of Egypt, who, according to him, reigned about 4200 B.C.[5] But, even at this early period, *neyroes* were

[1] E. Wilson, in Coll. Phys. Rep., p. 240. Macnamara on Leprosy, Calcutta, 1866, pp. 21-24.

[2] Danielssen and Boeck, Traité de la Spedalskhed, p. 338.

[3] Pruner, Krankheiten des Orients, p. 172.

[4] Histoire d'Egypt, par Henri Brugsch Bey (Leipzig, 1875), p. 42.

[5] *Ibid.*, p. 179. Of course, this is only an approximative date. Wilkinson only dates Menes, the first king, at probably 2000 to 3000 B.C. (Ancient Egyptians, vol. i. p. 307); but, from Brugsch's calculations, this appears too late. However, these dates are all long anterior to the time of the Jewish exodus—an important point.

already "carriers of wood" (p. 16) to the people of Egypt. They were already slaves to them, and this communication between the Egyptians and the negro races has always been kept up. At p. 8, Brugsch says, "At the highest antiquity, these tribes extended to the frontiers of Egypt." During the sixth dynasty (about 3200 B.C.), Brugsch (p. 71) quotes from monumental records to show "the names of several negro countries which already at that period were under the Egyptian rule," and that negroes were in the Egyptian army, as the officers, "se sont efforcés de dresser militairement les negres."

There can be little doubt that the Egyptians, even under their earliest kings, undertook expeditions, and carried back captives from both Africa and Asia,[1] and that negroes even came, of their own accord, to Egypt as servants[2] as far back as Amenemhat IV., about 2250 B.C., and later on, during the nineteenth dynasty. Moses, long before he led out the Israelites, conquered the city of Meröe, in the heart of Ethiopia.[3] Herodotus (lib. ii. c. 31) says that the Nile was known for four months' journey above Elephantine (itself about 1100 miles from the sea), and describes a river in the interior of Lybia "flowing from west to east" (c. 32), which was probably the Niger. His description of the sources of the Nile also, as known to the Egyptians, shows some real knowledge of the lakes lately discovered.

Thus, there was evidently abundant, early, and continuous communication between the interior of Africa and Egypt, that from the northern interior chiefly consisting of an influx of black servants or slaves. The Egyptians never colonized to the southwards, with one exception,[4] but penetrated to great distances, and returned. Traces of ancient communication still remain, in the similarity of means of hunting, and other things, among the inhabitants of Southern Central Africa to those used by the ancient Egyptians.[5] Even at Lake Ngami this influence is seen, but this must probably be attributed to long subsequent migrations of the tribes of the interior. This communication probably began soon after the arrival of the Egyptians from Asia, whence they originally came,[6] probably as conquerors, and from a more or less nomadic state of life—a state which I have noticed does not seem compatible with the existence of leprosy, as I am not aware that it has ever been observed among any nomadic tribe. Although seen

[1] See Brugsch, p. 75, 91; Wilkinson, vol. i. p. 416, 417; and Lepsius, quoted in Humboldt's Cosmos, p. 488, footnote.
[2] Brugsch, pp. 109, 149.
[3] Whiston's Josephus, p. 70.
[4] Herodotus, lib. ii. p. 102.
[5] See Anderson, Lake Ngami, p. 522; Livingstone's Zambesi and its Tributaries, pp. 168, 509; and Last Journals, vol. ii. pp. 117, 206; and Wilkinson, vol. i. pp. 69, 241.
[6] Brugsch, p. 6. Wilkinson, vol. i. pp. 302–309.

among town Arabs,[1] it is not known in Oman,[2] among the wandering tribes, nor is it mentioned by Layard among the Arabs of Mesopotamia,[3] or by Huc, or Prejevalsky[4] among the Tartar hordes, or by Kenuan among the Koriaks of Eastern Siberia, while in Africa no notices of it appear in any books of travel except among the settled tribes. Hirsch, in his Historisch. Geograph. Pathologie, does not refer to leprosy among any nomadic tribe. The Bedouins in Egypt are said to be free from it.[5] But as the Egyptians could not have carried leprosy with them to Egypt, and, as I shall hereafter try to show, there is nothing to uphold the idea of Lucretius that it was "morbus qui *propter* flumine Nili gignitur;" as in Northern Central Africa everything has remained *in statu quo* for ages, while in Southern Central Africa migrations have taken place from north to south; as leprosy only exists to a very limited extent in the countries immediately south of Egypt, in some, as Senaar, not at all;[6] as the immigration of negroes from Northern Central Africa to Egypt would be infinitely more likely to cause the propagation of a chronic disease to the Egyptians than the mere inroads of the latter to carry such disease to that centre, I am led to the belief that Egypt first received leprosy from the Soudan and Darfur. This belief is strengthened by noting, from perusal of the works of Livingstone, Baker, New, and Stanley, that leprosy is by no means so common in Southern Central Africa as in Northern, and that those tribes, as the Manganja, among whom it does prevail, are those who have been longest in contact with the Portuguese—themselves a much tainted people. It is unknown around Lake Tanganyika, as Stanley (How I Found Livingstone, p. 532) mentions twenty-five diseases known at Ujiji, but not leprosy; and Livingstone does not speak of it as existing there, or anywhere from the Nyassa people,[7] and the Barotse and Banyai,[8] until the Manyuema, near the Lualaba, were reached.[9] But the Nyassa people came from the west-north-west, and may have

[1] Buckhardt's Arabia, p. 447, in a Bedouin, but he elsewhere speaks of town Bedouins; and Dr Claude Bernard of Algeria—private letter.

[2] Wellsted's Arabia, p. 312.

[3] Niebuhr's notice of it only refers to the towns of Yemen and Baghdad, etc.

[4] Mongolia, London, 1876.

[5] Coll. Phys Rep., p. 53.

[6] Hirsch, p. 311. Aubert Roche (Annales d'Hygiene, January 1846) only speaks of it among the mountains of Samen. Pruner, p. 164 (so far erroneously quoted by Hirsch, p. 311), only mentions having seen it "an Abyssiniern," *i.e.*, among Abyssinians, not in Abyssinia, where Pruner never travelled; but Baker never mentions it; and the elephantiasis mentioned by Bruce at Massuah (vol. iv. p. 232) is most certainly Barbadoes leg. By the kind courtesy of Dep.-Surg.-General Munro, I am informed that it was not mentioned among the army reports of the late Abyssinian war. It is not mentioned among the Gallas by New (Wanderings in Eastern Africa, 1873), though he mentions it among the Wasuahili on the east coast further south.

[7] Zambesi, etc., p. 119, and Last Journals, p. 131.

[8] Missionary Travels, pp. 503 and 629.

[9] Last Journals, vol. ii. p. 40.

brought leprosy with them;[1] and the fact that the Manyuema and Becuana dialects are similar,[2] proves that they also, separated by about 1500 miles, have migrated to great distances. But they have had at least some communication with Portuguese, and doubtless with negroes in their employment, as shown by beads from the west coast being found among them.[3] Between Lake Nyassa and the Manyuema country is about 1000 miles, clear of leprosy. A reference to works of travel in Northern Central Africa shows that it is much more common there.[4]

Passing to India, the earliest record of leprosy I have discovered has been through the kindness of Captain Waterhouse, Secretary to the Asiatic Society of Bengal, who most kindly referred questions put by me to Babu Rajendrálála Mitra, who as kindly answered them very fully. From these answers I gather that "kushta," or leprosy, was written about by the sage Atreya, son of Atri, who is quoted in the Vedas. Atreya is also quoted in the Rig Veda Sanhitá, dating from the fourteenth or fifteenth century B.C.[5] The date of the ancient Vedas has been placed by Colebrooke in the fourteenth century B.C.,[6] in which he is supported by Professor Cowell of Cambridge, who says that the Rig Veda is certainly not later than 1400 or so, B.C.[7] The word "kushta" was evidently meant to indicate leprosy by Atreya, as quoted by Charaka (600–400 B.C.), but by later authors was used in a generic sense, as by Susruta, about two centuries after Charaka.[8]

Kushta is not mentioned in the ancient Vedas,[9] but as they were religious poems, and, as Roth says, leprosy has never played as prominent a rôle among the Indian population as among some others—for instance, the Jews—its absence cannot be used as an argument against its existence when they were written.

From all the information I have been able to obtain from the best Sanscrit scholars of the present day (whose kindness in answering my queries I am very grateful for), leprosy, known then, as it is now, by the name "kushta," has existed in India from the earliest period of history, and was a common disease either before or cotemporaneously with the conquests of Sesostris of Egypt, who is said to have reached India in the thirteenth century B.C., fully half a century before the exodus of the Jews, so that

[1] Last Journals, p. 117.
[2] *Ibid.*, p. 117.
[3] *Ibid.*, vol. i. p. 180.
[4] See Caillé, Central Africa, pp. 225, 402; Park's Travels, p. 275; Du Chaillu, p. 390; and also Hirsch, p. 311.
[5] My questions and the Babu's answers are published in the Society's Transactions for 1875.
[6] Wise, Commentary on Hindu System of Medicine, p. 17. Calcutta, 1845.
[7] From private letter, for which I have to thank him.
[8] The Babu's letter. Roth of Tubingen says, "Kushta ist der name der Gattung."—(*Private Letter.*)
[9] Cowell, Roth, and the Babu.

these events could have nothing to do with its introduction into India. There was undoubtedly commerce between Egypt and Arabia 1000 years before this,[1] and probably communication thence to India; but of direct communication between Egypt and India there is no trace that I have discovered.

The Babu Rajendrálála says positively, "There is no indication whatever of leprosy having been imported from the west."

The weight of evidence, therefore, goes to show that leprosy is an indigenous disease in India.[2]

The next country in which it is important to discover whether leprosy is indigenous or imported, is China. In regard to its history there, I am much indebted to several Chinese scholars, who have most fully answered my inquiries, which were necessary, as I could not find any information as to its early history in any book I referred to. Dabry (La Médecine chez les Chinois) has a chapter on "Ta-ma-fong," as described by Chinese authors, but gives no references as to dates.[3]

Leprosy is not mentioned in Marco Polo's travels; but this may be because he resided chiefly in the north, while it prevails mostly in the south. Davis (The Chinese, vol. i. p. 357, and vol. ii. p. 14) mentions its prevalence, and the existence of a leper hospital at Canton. It is mentioned by Holman (Travels in China, pp. 152 and 272). In the College of Physicians' Report, p. 74, it is stated that the Leper Asylum at Macao is 200 years old.

Hirsch,[4] quoting Hobson and others, speaks of its enormous spread in China at the present day. I am informed that Dr Hobson, in a paper in the Chinese Repository, 1851, which I have not been able to get a reference to, mentions, as the first notice of leprosy, the case of a disciple of Confucius (about 500 B.C.); but the Rev. Dr Legge tells me that the name in the original was "lai," which is now used for itch, not leprosy. Mr Scott, of Edinburgh, mentions that leprosy is not spoken of in the earliest extant Chinese records, the Shoo-king, dating about 2000 B.C., nor in several other very ancient works, but chiefly in the Tcheou-li, referred to about 1500 B.C., which gives an account of the court of the third dynasty, and "a special account of the medical staff and its duties," and of ulcers of different sorts, but "it does not describe anything which seems to have any likeness to leprosy." Dr Dudgeon says, "Leprosy goes back to a period in China before our era;" and Dr Lockhart, "It has prevailed in China for some

[1] Brugsch, p. 80.

[2] This conclusion I have come to from my correspondence; the authors I have consulted, viz., Wise and H. H. Wilson (Hist. of Lep. by Sanscrit Writers, in Calcutta Med. Trans., vol. i.), only tracing it back to Charaka and the Ayur Veda, about 900 B.C., so that from them I could not conclude that it had not been imported from Egypt.

[3] I wrote to Captain Dabry at Canton, but have as yet received no answer.

[4] Lib. cit., p. 314.

centuries B.C." Le Marquis d'Harvey St Denys, of Paris, writes, that, on consulting his Chinese treatises on medicine, "Ils sont muets sur l'histoire de le lèpre," except one, which says that it was formerly called "li-fong," and *was very rare* in China."[1]

So far as etymology can assist us, the foundations of the words for leprosy are all regular Chinese, with no evidence of foreign origin.

So far as the imperfect evidence goes, there is no proof at present available of the existence of leprosy in China more than one or two centuries before Christ. As far as mere negative evidence goes, it did not exist there 1500 B.C. This is supported by its being formerly very rare, and now very common, although of course it *may* have been indigenous, but increased by commerce with other leprous nations, as the Indians and Portuguese, and by the change in habits of the people, the ancient Chinese being compelled by law to be cleanly, which the moderns certainly are not. It is not possible, however, to trace its introduction to any historical event, as in western countries. It is a significant fact, however, that it is almost entirely confined to the south-eastern provinces, which have been from the earliest times in constant communication with India by sea,[2]—a much more likely mode of introduction than by land, considering the mountains that intervene.

Seeing that leprosy can be traced in India further back than it has hitherto been in China, I am inclined to the opinion (though I express it with very great reserve, in the face of discoveries that may yet be made by Chinese scholars) that it is not indigenous in China, but has been introduced, probably from India, by commerce. Had it been indigenous, it would probably have always been as common as it is now. Yet, as I have said before, it may have been so, and been increased during the present era, partly by importation, partly by the changed habits of the people.[3]

We have thus examined into the early history, so far as known, of leprosy in the three parts of the world where there might be any reason to believe that it is of indigenous origin in remote periods. In only two other places could it have arisen of itself, and there may be some doubt as to the reality of the leprosy in both, viz., New Zealand and Fiji; but as these have only become

[1] Private letters, for which I thank the authors.

[2] Humboldt's Cosmos, vol. i. p. 173.

[3] It is possible that it may have been introduced into China by some of the lost ten tribes, who appear to have reached China about 200 B.C. (Davis, The Chinese, vol. i. p. 16; and Hetherington, in Christian Miscellany, 1843, The Lost Ten Tribes of Israel.) The facilities for increase by subsequent commerce were great, the Chinese not being an exclusive nation themselves until the accession of the present Tartar dynasty, in 1644. In the ninth century of our era there were 120,000 Mahomedans, Jews, Christians, and Parsees in Canton, and Chinese ships sailed to the Persian Gulf. (Travels by an Arabian Merchant, in Kerr's Collection, vol. i. p. 52, *et seq.*; and also Davis, p. 20.)

lately known, the description of leprosy in them will be best considered further on.

One question in regard to the origin of leprosy among the Egyptians, Hindoos, and Chinese, may be asked. Is it not possible, seeing that all these peoples had their origin in Central Asia, that leprosy existed among them as among one people, and was carried with them in their migrations southward? What I have already stated, as to its absence among nomadic tribes, answers this.

I may say also, that the idea of the Chinese being a colony of Egyptians, or having very early commerce with them, partly founded on the finding of some porcelain Chinese jars in ancient Egyptian tombs, has been disposed of by Wilkinson, who shows satisfactorily that they must have been brought by Arabs.

It being impossible to trace the transmission of leprosy from India and China in early times to any other part of the world, in the present state of our knowledge (though it is possible that the leprosy of Thibet[1] may have been carried from China, and that of Yarkand[2] from India), we now return to Africa, the centre to which can be traced the leprosy of Asia Minor, Europe, and the New World, to note its spread thence.

Were the idea correct which has been mooted by some,[3] that Job's disease was leprosy, this would be the earliest notice of it out of Egypt; but seeing that the word *shachyn*,[4] used for a boil (Job ii. 7), is also used to indicate Hezekiah's disease, in which there was only one severe boil, probably a carbuncle, an acute disease (2 Kings xx. 7), and "the boil with blains on man and BEAST" (Exod ix. 9), an acute disease breaking out suddenly on a whole population in Egypt; it is also translated "botch" in Deut. xxviii. 27; thus, to those who have seen the severity of such diseases in the tropics, or experienced them in their own persons (I have had thirty boils on my body at one time, destroying sleep for a week), there is no need for believing that Job suffered from anything else than is stated in the English text—viz., that he had sore boils or a carbuncle. This agrees also with the Septuagint ἕλκος, and the French "ulcère malin," and was quite enough to try his patience, and even more calculated to do so than leprosy, which is by no means a painful disease, but the reverse.

There has been much discussion as to whether the leprosy of the Jews really was true leprosy, elephantiasis Græcorum, and the most varied opinions have been expressed in regard to the point; Dunbar[5] thinks it is now an extinct disease; Balmanno Squire[6]

[1] Travels in Tartary, etc., by Huc, vol. ii. p. 199.
[2] Lahore to Yarkand, by Dr S. Henderson, p. 118.
[3] As Wortabet in Brit. and Foreign Med.-Chir. Rev., 1873.
[4] Gesenius (p. DCCCIV., שחין) translates it an inflamed boil, an ulcer, but thinks it was also used for elephantiasis, with "the feet swelling up," *i.e.*, Barbadoes leg. Jahn, in his Biblical Antiquities, thinks it was black leprosy or psora, but ψορα is itch or scab, not leprosy.
[5] Brit. Med. Jour., vol. i., 1873, p. 313.
[6] *Ibid.*, p. 141.

thinks "white as snow" meant psoriasis; and Kitto,[1] from differences in the description, concludes that it was not identical with modern leprosy; but Jahn[2] believes that it is identical with the leprosy of Guadaloupe, but influenced by change of climate. Daniellssen and Boeck[3] say there can be no doubt that it was the elephantiasis Græcorum, modern leprosy. Schillingius[4] of Surinam is of the same opinion, and strongly opposed Reill, who held that "Lepra Judaica" was "omnino diversam a lepra Americana." H. V. Carter of Bombay[5] holds that it is an undecided point, though he seems inclined to identify the eruption of leprosy with Mosaic lepra. Erasmus Wilson,[6] Kaposi,[7] and Tilbury Fox,[8] consider that Jewish leprosy, "tsaraath," included modern leprosy along with psoriasis and other skin diseases. Fox points out that different kinds of "tsaraath" were differentiated by the Jews, as such cases as that of Naaman[9] were allowed to come in contact with others, not being unclean, as only suffering from lepra vulgaris, while others, as Uzziah,[10] was at once thrust out, being unclean, suffering from true leprosy. Jahn also identifies the white spot, "berat," spoken of in Levit. xxii., as morphea, or the eruptive leprosy of the present day. I do not believe with Wilson or Kaposi that all cases of morphea are really cases of a remnant of leprosy, but consider that their views as to Jewish leprosy are undoubtedly correct.

From the broad meaning of the word "tsaraath,"[11] "a stroke," and from the ideas that the Jews had of it as a stroke sent in anger by God—an idea shared in by other ancient nations,[12] and even by some at the present day[13]—the term would certainly be used to indicate any severe disease, being in fact a popular term, and as such used in a vague manner.[14] Thus, while from the symptoms mentioned in Leviticus, which it ought to be distinctly remembered (as it has not by Kitto and others,[15] who have looked at the want of symptoms seen in modern leprosy as proving the non-identity of the two) are only the premonitory symptoms indicating the beginning of the "plague of leprosy," other diseases being indicated, as one with the characters of leucoderma, mentioned under the

[1] Art. Leprosy in Dictionary.
[2] Biblical Antiquities, p 86.
[3] Traité, p. 2.
[4] Dissertatio de Lepra, 1778, Prolegomena, by Hahn, p. 14.
[5] Trans. of Med. and Phys. Soc. of Bombay, 1861, pp. 4 and 21.
[6] Lancet, 26th April 1856.
[7] Hebra, Dis. of Skin, New Syd. Soc. Trans., vol. iv. p. 189.
[8] Ed. Med. Jour., March 1866. [9] 2 Kings v. [10] 2 Chron. xxvi. 6.
[11] From נגע, Tsare, to strike, or, supplying the ellipsis, the stroke of the Lord (see Jahn, p. 186, and Gesenius).
[12] As by the Assyrians (Smith's Assyrian Account of Genesis, p. 124), the Persians (Herodotus, lib. i. c. 138), and the Hindoos. See Wise, pp. 196, 207.
[13] Leared, 1876, Morocco, p. 148.
[14] Josephus, quoting Moses, says, "*if any one* of their diseases" (*vide* Jos. against Apion, lib. i. c. 31).
[15] As Mason Good's Study of Medicine, vol. iv. p. 453.

name of "bohak,"[1] which do not cause uncleanliness; from a comparison of the description of those symptoms with those of the eruption of leprosy by Schillingius,[2] Cazenave and Schedel,[3] Kaposi,[4] Erasmus Wilson,[5] and chiefly Carter;[6] and also as the first symptom spoken of is a rising[7] (or tubercle), which is the most prominent manifestation of tubercular leprosy; I have no doubt that elephantiasis Græcorum was one of the forms, and probably the chief form, of the leprosy of the Jews. The description of one of the forms of Kushta by Atreya corresponds also to that of Jewish leprosy.[8]

Dr Milroy points out[9] that anæsthesia, the most important, but by no means the most prominent, symptom of leprosy, is not mentioned by Moses; but this argument is of no value, as anæsthesia is not found in the earlier stages, the only stage Moses required or desired to describe, and besides, might even, when present, be easily overlooked, as I found when trying to discover its existence in cases of joint evil or anæsthetic leprosy in St Kitts, in which I could hardly detect it; nor is the patient often aware of it himself till its existence is called to his notice,[10] as I have myself observed. It might just as well be said that the ἐλεφαντίασις described by Celsus was not true leprosy, which would be absurd, yet he says nothing about anæsthesia.

I may hereafter return to this subject, which requires much more space than can be spared for it here, but I think I have said enough for the present, except that had real leprosy arisen as a *new* disease among the Jews in historical times, we have too full an account of them for such an event to have passed unnoticed.

Thus, from the Bible, as well as from Josephus,[11] we can be certain that leprosy existed among the Jews and Egyptians at the time of the Exodus, or from about 1550 to 1300 B.C.[12] We hear of it again among them when the "four leprous men" discovered the flight of the Syrian army at Samaria,[13] about 892 B.C., and who

[1] בהק, Sept. ἀλφός, Lev. xiii. 39, translated freckled spot. This name is still retained by the Arabs (Niebuhr, Travels through Arabia, p. 278).

[2] Dissertatio, p. 7.

[3] Maladies de la Peau, p. 350.

[4] Hebra, vol. iii. p. 182; and vol. iv. p. 139.

[5] Diseases of Skin, p. 674; Lancet, 12th Jan. 1856; and Coll. Phys. Rep. on Lep. Appendix

[6] Trans. of Med. Soc. of Bombay, 1862, p. 4.

[7] Lev. xiii. 2, שאת, Shat, a rising or tumour (Duncan). It has often been supposed to mean an eruption, but this is erroneous. The primary meaning is a rising up, something elevated. The French translation "tumeur" is a good one.

[8] Wise, *l. cit.*, p. 260, six severe kinds, and the Babu Mitra in the Asiatic Society of Bengal Proceedings, Aug 1875.

[9] Coll. Phys. Rep., p. 230.

[10] Carter, *l. cit.*, p. 33.

[11] Against Apion, b. i. c. 26, 31, 34.

[12] Josephus makes it about 1550, the Bible 1491, and Brugsch (p. 175) 1300. The latter appears the most correct date.

[13] 2 Kings vii. 3; and Josephus, Antiquities, ix. 5.

were, be it observed, put out of the city; then the case of Uzziah, about 765 B.C., showing its continuous existence.

Passing from the Jews, we find it among the Persians, who had laws for the expulsion of lepers before the time of Herodotus[1] (about 450 B.C.); cases of leuce (λεύκη) or leucoderma were also expelled these cities. This *may* have had some connexion with the captivity of Israel in 721 B.C., and of Judah in 605 B.C., when they were carried into Assyria and Babylon, neighbouring nations to Persia.

I have searched for mention of leprosy among the descriptions of the remains of Nineveh and Babylon in the works of Layard, Smith, and Rawlinson, but have found none. As the records they translate, however, are entirely in the cuneiform character, which was looked on as sacred, and only to be used for commemorating great events, while writings in the common running character were used for ordinary purposes,[2] and as it is only the great deeds of conquerors, or the creation of the world (Smith), that is spoken of in such writings as are extant, no conclusion can safely be drawn as to the non-existence of leprosy among the ancient Assyrians and Babylonians from its not being mentioned.

Plutarch mentions, in his Life of Artaxerxes II., that his wife (and daughter) Atossa was a leper, about 380 B.C.

We have now considered all that is at present known of the ancient history of leprosy in Africa and Asia; we must now pass to the consideration of its introduction into Europe, and its slow spread north and west, trying to connect such spread, so far as may be possible, with events likely to cause it.

Passing to Greece, the first country in Europe in which it appeared, we look in vain for any proof of its existence there at the time of the Hippocratic writings There can be no doubt that by λεπραί (which always occurs in the plural), psoriasis, or some disease nearly allied to it, and not lepra Græcorum or true leprosy, was meant. The term simply meant scurf or scales. In the first place, ὄνυξι λεπροίσι (translated unguibus scabies), or scruffy nails, are mentioned,[3] while the expression ἐλέπρα τὴν κύστιν,[4] or scabby bladder, could apply to no symptom of modern leprosy. Secondly, λεπραί are always mentioned along with other slight diseases, such as lichen and impetigo.[5] Again, Celsus, in quoting, and almost translating, a passage in which λεπραί[6] are spoken of, uses the term pustulæ[7] as including them along with lichen and alphos, which he describes elsewhere[8] as the mildest form of

[1] I. 138.

[2] Layard, Nineveh and its Remains, vol. ii. 342.

[3] Hippocrates Opera, ed. Kuhn, tom. ii. p. 160, Περι ὑγρων χρῆσιος.

[4] Tom. ii. p. 554, Επιδήμιον το πέμπτον. Although this is not a genuine work of Hippocrates, still it shows the use made of the word about his time.

[5] Προρρητικον, ed. Kuhn, vol. i. p. 232, book 2d, last paragraph, and Αφορισμοι, tom. iii. p. 724.

[6] Aphorisms, as in last reference. [7] Lib. ii. c. 1. [8] Lib. v. c. 19.

vitiligo or leucoderma. Now, as he uses pustula to indicate a wheal or blain, he could not have been thinking of such a disease as leprosy when he wrote the passage referred to.

Galen also makes use of the term lepra as indicating a disease distinct from leprosy, as in the passage in which he says, "At pruritus in precipue affliget infectos psorâ vel leprâ,"[1] while elephantiasis is his name for leprosy; but as he follows Hippocrates, we can hardly think he would have changed the use of the word so completely. We are thus, I believe, on these grounds, entitled to conclude that by "λεπραί" the Hippocratic writers did not signify leprosy.[2]

Some have thought that the word "φθινικὴ" (phthinike), used by Hippocrates for phthisis,[3] should be read φοινικὴ (phœnice) or Phœnician, so that the Phœnician disease would be referred to; and Galen, in a passage I have seen quoted, but cannot find in his works, thinks that ἡ νουσὸς ἡ φοινικὴ meant elephantiasis. But then Galen himself did not seem to be sure of the meaning, while Kuhn's and other good editions give the word φθινικὴ, or phthisis; and even if it did mean leprosy, the mere mention of it in such a way does not in the least prove its existence in Greece at the time. I do not believe, however, that such a writer as Hippocrates could have passed over a disease of such importance, had it been known to him, with such slight notice.

H. V. Carter,[4] one of the best living authorities on leprosy, says that "Hippocrates and Galen state, that leuce sometimes passed into elephantiasis." I merely mention this to point out that it is an error, which may mislead others, as it did me for some time, and make them think that Hippocrates spoke of elephantiasis, which I have already stated is not mentioned by him. Carter gives no references; but the only passage in Galen he seems to refer to would hardly bear out his statement, as nothing more seems to be indicated than that leuce and elephantiasis arise from the same causes, Galen's words being,[5] "Nec aliter affectus hic (elephantiasis) gigni solet quam leuce." The cause he mentions

[1] De Symptomatum Causis, lib. ii. (Argentorata, 1604, p. 682).

[2] Against these arguments it may be said that Herodotus used the word λεπρήν, as also the Septuagint authors, for what was evidently leprosy; but they were not medical authors, making a strict use of the term. Herodotus simply used a name of a disease known to him to indicate a disease probably unknown to him except by description; while the Septuagint translators used the generic term, to translate sufficiently clearly for their purpose, another generic term in the original Hebrew. The same loose use of the word "leprosy" is seen in the writings of Cook, who mistook sea-salt on the skin for leprosy (Voyages, ii. p. 56); and of Ellis (Polynesian Researches, vol. ii. p. 19). I suspect that the leprosy spoken of by Dr Seeman in his Mission to Fiji, p. 338, so wonderfully cured by roasting with the sinugaga wood, was also simply psoriasis, or some allied disease, or leucoderma, as in Ellis's leprosy.

[3] Prorrhetics, at end.

[4] Trans. of Med. Soc. of Bombay, 1861, p. 16.

[5] De Symptom. Causis, p. 687.

is poverty of food. He elsewhere[1] says that they are "melancholic" diseases, arising from black bile. The first Greek author who decidedly mentions leprosy, under the name of satyria, is Aristotle. I may translate his words here,[2] to leave no doubt that it was really leprosy he meant: "The disease called satyria . . . . . from a plethora of humor or air breaking out in the parts of the face, the countenance is like that of any beast and satyr." I cannot imagine myself any other disease these words could apply to as a general description, except tuberculated leprosy. Hirsch[3] doubts this, but gives no reasons for his doubts; while Daniellssen and Boeck say it could only be elephantiasis.[4] Aristotle does not say *where* he saw this leprosy; but as he spent all his life in Greece or the adjacent coast of Asia Minor, as from the description he gives (a very full one, considering that it is only given by way of illustration, in a work on the generation of animals) he must have seen the disease, it can hardly be doubted that when he wrote, about B.C. 345, or fully half a century after Hippocrates, leprosy, although still probably a rare disease, had found its way at least to the coasts of Asia Minor near Greece, and probably to the latter.

From this time onwards it seems to have spread slowly but surely in Greece and the adjacent countries of Europe. The words of Celsus, written in the early part of the first century after Christ, show this, and that at his time it was, and had evidently long been, common in Greece. He says—"Ignotus autem paene in Italia, frequentissimus in quibusdum regionibus is morbus quem ἐλεφαντίασιν Græci vocant! isque longius adnumeratur."[5]

Thus, we find that leprosy was known under the names elephantiasis and elephas, from a fancied resemblance to that animal,[6] and that this became its common name among them. Now this, I think, is a very strong argument in favour of its having been introduced into Greece only a very few centuries before Christ, as neither Homer nor Pindar use the word elephas (ἐλέφας) to indicate an elephant, but ivory, they not being acquainted with the animal itself. (Pindar's writings preceded those of Hippocrates by fully half a century.) Herodotus, writing about 446 B.C., or four years before the death of Pindar, was the first to use the word for the animal, and he only refers to them as existing in Ethiopia among other wild beasts.[7] He mentions horses, camels, and asses in the great expedition of Xerxes,[8] but no elephants.

Aretæus says that *some* call it leontiasis, but it is evident that elephantiasis was its common name in Greece.

To sum up, then; leprosy appears to have been quite unknown

[1] De Tumoribus præter naturam.

[2] Περὶ Ζώων Γενέσεως, iv. c. 3, from "τὸ νοσήμα τὸ καλοῦμενον σατύριαν."

[3] Lib. cit., p. 304. [4] Lib. cit., p. 3. [5] Lib. iii. c. 25.

[6] See Aretæus, De Lepra et Causis Anat. Morb., lib. ii. c. 13, ed. Kuhn, Lepsiæ, 1828, pp. 174 and 182. Elsewhere, Aretæus calls it ἐλεφάντος and ἐλεφάντα.

[7] History, lib. iii. s. 114 [8] Lib. iv.

in Greece up to the time of Hippocrates, or about 400 B.C. By Aristotle's time (345 B.C.) it had become slightly known, but was still rare; and before our era, probably two centuries or so, it had become common.

Now, up to the time of Cambyses, 525 B.C., Egypt, the country where we have seen leprosy had been so long endemic, was as much closed to the Greeks as Japan was to a very recent date to Western nations. In 650 B.C. the first Greek factory was opened in Egypt, and a hundred years later Greek mercenaries served in the Egyptian army; but till the conquest of Egypt by Cambyses, 525 B.C., Egypt was practically closed. Close after this came the conquests of Darius, and then of Xerxes, 480 B.C., who led, according to Herodotus,[1] a host of about six million people into Europe from all the nations of Asia and Africa under his rule, and when he retired left thousands behind. These two historical events, and especially the latter, have a close connexion with the spread of leprosy, which it can hardly be too much insisted on, is essentially a slow disease. We will see hereafter that it seemed to take between one and two centuries to spread fully over Scotland, after it was in England; and it has taken nearly a century, even in these days of quick travel, to reach the Sandwich Islands. It cannot, therefore, be a matter to cause surprise that it was not known in the south of Greece in the time of Hippocrates, only three-quarters of a century after Xerxes had entered it, and abandoned part of his army in the north, while just what has taken place in modern times took place then. Half a century after Hippocrates it was noticed, and then gradually spread, and a century or two later was a common disease.

We will now trace the spread of the disease throughout the world.

From the time that leprosy fairly gained a footing in Europe, it can be clearly traced in its onward course. The words of Celsus and Lucretius, already quoted, and the positive testimony of Pliny[2] to the effect that it was unknown in Italy until *the return of Pompey's soldiers from the East* (B.C. 62), show that it reached Italy in the first century before Christ.[3] Galen,[4] writing about 180 A.C., speaks as follows (after speaking of its existence in Alexandria): "At in Germania et Mysia rarissima hæc passio videtur, et apud Scythias lactes potatares, nunquam fere apparuit;" showing that it had spread beyond Italy in his time, although it was still very rare in Germany; but four centuries after this, from the researches of Virchow,[5] we learn that it had already spread so much, that, in

[1] Lib. vii.

[2] Pliny, lib. xxvi. c. i. of the Historia Mundi; and p. 154 of vol. v. of Bohn's edition.

[3] See also Hirsch, p. 305; and Hebra, vol. iv. p. 121.

[4] De Arte Cur. lib. ii. cx.

[5] Granulations geschichte (p. 506), which I thank him for sending me; also in Archiv, B. xviii. and B. xx.

636, leper houses were already established in Italy, Verdun, and Mæstricht. In 757 Pepin of France, and in 789 Charlemagne, made marriage of lepers illegal, and leprosy a sufficient cause for divorce.[1] In the ninth and tenth centuries, leper houses were probably established in Bremen and Constance. Thus, by the end of the tenth century, the disease had already spread to some extent all over France and Germany.

Now let us glance at some of the great historical events which had taken place in Europe during the period of fully ten centuries during which we have just traced the progress of leprosy. Just before, and at the time of Christ, Rome had become the mistress of the civilized world, and communication between all parts of the empire was, for those days, easy and constant; armies were constantly leaving and returning to it, *bringing captives from all parts*. Jerusalem had fallen, and the Jews were scattered. All these facts may be kept in view when we read the words of Galen, already quoted, and show, along with what we are told by Pliny, that Rome, in gaining an empire, paid as one penalty the infliction on generations then unborn of the most loathsome scourge, the most hideous death in life that has ever afflicted the human race. This scourge was not only carried to Rome by her returning armies, as Pliny states, and as we know also it was to France in modern times from Egypt,[2] but also without doubt by those of the numerous captives taken to Rome. From Rome, again, it was carried by the constant communication going on between it and all parts of the empire, including France, Germany, and Spain,[3] up to the time of the fall. In the fifth and sixth centuries the disease would be still further spread by the conquests of Alaric and others, and the return of *their* armies with captives to their native places.

We are informed that leprosy was first carried into Spain by the Roman troops soon after its outbreak in Italy,[4] and that it was common there in the tenth century. This, I may point out, is of importance in connexion with its spread in France, where it appears to have somewhat increased in intensity about the time of Pepin, as he in 757 promulgated the law already mentioned, this being *about forty years after France had been invaded by the Saracens from Spain*, in which country leprosy was increased after their invasion of it;[5] an increase, be it remarked, noticed about two centuries after that invasion. Doubtless the frequent invasions

[1] Lepers were also looked upon as dead.

[2] Larrey, Relation Histor. et Chir. de l'Armée en Orient, 1803, p. 236. Only a few cases returned, as Napoleon's occupation of Egypt was short. This chiefly, and also the improved condition of the French as compared with former times, may have been the reason of its not spreading. Nor, be it remembered, were any Egyptian captives taken to France.

[3] Marejon, quoted by Hirsch, p. 305.

[4] Ozanam, quoted by Hirsch, p. 305.

[5] Hirsch, p. 305.

by the Lombards in the sixth century, they having the disease among them, would also spread it in France.

From the late Sir James Simpson's classical papers on the subject,[1] we learn that leprosy had reached Wales in 950, as Noel Dha, then king, passed a similar law to that of Pepin in that year.

We now come to a period in the history of leprosy when all authors who have investigated the matter agree, however differently their opinions may have led them to explain the reason, that there appeared to be a great increase in leprosy all over Europe,[2] and when it first really seems to have become very severe in England—I mean the period of the Crusades. Hirsch does not certainly consider this absolutely proved, thinking that the apparent increase may have been only from a more scientific spirit in the medical world of the time, and an increase in the number of leper hospitals at the same time, partly no doubt as a consequence of that spirit;[3] but surely this is less likely than that the increased number of lepers then required more leper houses, as their decreasing number from the fifteenth century onwards required fewer, especially as Hirsch expressly quotes Mezeray (Histoire de France, vol. ii. p. 168), to the effect that all the towns and villages in France in the twelfth century had leper houses. He also quotes Muratori (Antiq. Ital. Medævi, iii. 53) to the same effect as regarded Italy.[4] He also as expressly states that many who went away healthy (der gesund ausgog) returned from the East afflicted with leprosy. His idea that many of the cases of so-called leprosy were really cases of syphilis is no doubt correct, but it does not seem to have occurred to him that such cases would be *less* likely to be confounded with leprosy after the uprising of the scientific spirit he speaks of than before, and so many the less lepers would there have appeared to be as compared with previous centuries, had there not been a real and absolute increase in their numbers. In conclusion, I may say that Hirsch speaks of leprosy as being between the twelfth and fifteenth centuries "one of the widest spread (verbreitetsten) chronic constitutional diseases, playing a no

[1] Ed. Med. and Surg. Journal, 1841, vol. p. 301. He also proves clearly in these papers that the lepra of the Middle Ages, the English leprosy of Gilbert (1270), and the elephantiasis Græcorum of Bateman, etc., were identical (vol. i. 1842, p. 135 to 140). And it is pointed out as clearly by Dr Charles Macnamara (Leprosy, Calcutta, 1866) that the leprosy of England of the middle ages described by Gordon, was identical with the "Kushta" or leprosy of India, as seen there at the present day.

[2] Dan. et Boeck, lib. cit., p. 182; Simpson, lib. cit., 1842, p. 395; Hebra, vol. iv. p. 122; Michaud, History of Crusades, vol. ii. p. 308, etc.

[3] Lib. cit., p. 306.

[4] Lib. cit., p. 307. Mezeray's words are—"Il y avait ni ville, ni bourgade, qui ne fust obligee de batir un hospital pour les (sil. lepreux) retirer;" and Muratori—"In Italiam vix ulla erat civitas, quæ non aliquem locum leprosis destinatum, haberet." Could anything be stronger, even if some cases of secondary syphilis were confounded with leprosy, as they doubtless were?

less important rôle than syphilis has in our time,"—an admission hardly compatible with his own scepticism as to its increase at that time. That it was widespread there can be no doubt, as, according to the testament of Louis VIII. (1229), quoted by Michaud, there were at his time 2000 leper houses in France alone, and it has been stated 19,000 throughout civilized Europe.

In England, the first leper hospitals established seem to have been that of St Giles, London, in 1101,[1] and York about 1110; and from that time to 1472, a hundred and twelve such hospitals, all richly endowed, were built in England. Leprosy reached Scotland before 1177, three-quarters of a century after the first leper hospital was built in England; and many such hospitals were built over the country, at Aldcambus, Gorbals of Glasgow (1350),[2] Greenside, near (now in) Edinburgh (1589), etc., and in Elgin in 1226.[3] The latter date is to be noted as being at least forty years before the establishment of a leper hospital at Bergen in Norway, the part of that country in which leprosy has always been most severe, and is so at the present day,[4] it being, as Simpson points out, the *nearest* to Scotland,—a point of no little importance.

It also appears to have spread slowly northwards to the Shetlands, Faroe Isles, Iceland, and Greenland. The last part of the British Islands to be reached was, I must here point out, the one to which, looking on its introduction as only possible through more or less prolonged intercourse of the sick with the healthy, and at the extremely rare communication with any other part of the kingdom, we should have actually expected it would reach last—I mean the remote island of St Kilda, which it only reached about 1680,[5] after it had died out in England, and when it was on the decline in Scotland, at least in the southern counties where records remain of it.[6]

It also spread to Holland and Denmark, as well as Sweden and Ireland.

We must now slightly turn back to trace it into Russia, the only country in Europe where it has never been universally prevalent, *except* (and this is a most important exception) in those parts which have been at all times within the historical period the highways of commerce throughout that country, viz., the southern provinces, and more especially the Crimea and the provinces lying to the east of it.[7] It has also been observed in Finland, Esthonia,

[1] Simpson, 1842, p. 428. [2] *Ib.* 1841, p. 329. [3] *Ib.* 1842, p. 148.
[4] See Carter's Rep. on Lep. in Norway, map, etc.
[5] Simpson, lib. cit., 1842, p. 141.
[6] Even yet St Kilda is in one sense more remote from Scotland than Australia. Letters may be months delayed going and coming, as appears from complaints in the *Scotsman* lately. I wrote to the minister fully five months ago, asking whether any cases of leprosy still existed, but no answer has reached me yet. Dr Latham of Cambridge visited the island last year, and (as he kindly informed me by letter) saw no cases of the kind. He was known to be a doctor, so any sufferer might have been shown to him; but it is just possible that such cases might have been kept out of sight. [7] Hirsch, p. 315.

and Courland; but its presence in them is only another proof of its communicability, they being rather Swedish and Polish provinces than Russian, only having become part of the latter empire since the time of Peter the Great. In the great interior of Russia leprosy is unknown,[1] and that is precisely the part of the empire which, up to three centuries ago, was practically cut off from direct communication with the outer world. Voltaire[2] does, indeed, repeat a fable of Pliny's of some *Indians* being wrecked on the Elbe, and tries to give colour to it by saying that they might have come through Persia to the Caspian, and thence by the Volga and Perm to the Baltic, and on this less than slender foundation seems to argue that there was some early commerce to these regions; but as the Elbe is 1700 miles from Perm, and as the ideas of tribes and nations outside of the Roman empire entertained by the Romans at the time of Augustus were of the most confused kind, I cannot see that there is any reason either to believe the fable, or Voltaire's deduction from it. It may be true that Perm, on the extreme east of European Russia, in 60° N. lat., was, at the time of the Caliphs, a depot for the furs of what is now North Russia, where they were exchanged for the goods of Persia; but such commerce would not by any means bring the *people* of these two remote countries into contact, except in the case of a few traders of each country who would meet at Perm, and could have almost no effect in the transmission of a purely contagious (if not simply an innoculable disease, as I am inclined to think leprosy is), although infectious diseases might be so carried. But it is remarkable that leprosy is said to have been observed in Kasan;[3] and if such were the case, it would be an instance of the disease going just as far as actual personal commerce with the south was carried on, and no farther. Kasan is traversed by the Volga, giving free means of commerce from the south. It and Perm were both included in the "Tatar" empire of Gengis Khan and his successors.

I have thus spoken at some length to point out strongly the absence of leprosy in the centre of Russia, and also in Lapland and the other northern provinces;[4] these being the very provinces having no contact with infected countries, as I believe that it is from such proofs that the communicability of the disease can be made clear.

The date of its introduction into Russia is unknown. Hirsch (p. 314), quoting Krebel, considers it likely that it was already endemic in the 15th century; and Richter[5] says that it only reached Russia

[1] *Ibid.*, p. 316.
[2] Hist. de Pierre le Grand, p. 35.
[3] Hirsch, p. 316, Note. Blosfield says that cases occur, *arising* from syphilis. Hirsch says there are doubts as to the correctness of this idea.
[4] Voltaire says of the Lapps that they were "sans maladies."
[5] See Virchow in Coll. of. Phys. Rep., p. 1867 (quoted from Richter's History of Med. in Russia, vol. i. p. 245). Virchow says that it is only highly improbable that Richter's idea is correct.

in 1426. But this last author is decidedly wrong, as leprosy is expressly mentioned by Rubruquis as having been seen by him in the Crimea in 1236.[1] As true leprosy is now very common in the Crimea, there is no reason for doubting that it was the same disease.

As additional proofs of the connexion between the spread of leprosy and communication with infected countries, the following historical events may be kept in mind:—1. The Crimea was a Greek colony, founded about 550 B.C., and communication was naturally kept up for centuries afterwards with the mother country. 2. In the Middle Ages it was the country through which all commerce passed from east to west, and that commerce passed along the Volga and through the south-eastern provinces of Russia, where leprosy is most common to this day.[2] It is just possible that leprosy may have been introduced into Southern Russia at a very remote period of history by either of the following events,—(1.) Rameses or Sesostris, king of Egypt, penetrated as far as Scythia about 1350 B.C., and left a colony of soldiers behind him at Colchis.[3] (2.) The Scythians under Madyes, the Ogus Khan of the Tartars, penetrated as far as Egypt, and remained in Persia twenty-eight years before they were driven back, 610 B.C.[4] (3.) Darius left a large part of his army in Scythia about 510 B.C., those left being chiefly the sick and their guards. Under the circumstances they probably fraternized with the Scythians.[5]

Thus there has been abundant communication between the south of Russia (and more especially the south-east) with countries infected with leprosy, and the consequence is seen in its existence there; while, as already stated, it is unknown in the parts which have had no such communication.

I may finish what I have to say about Russia by saying that the disease seems to be unknown in Siberia, with the exception perhaps of Kamtschatka;[6] in which, however, syphilis is exceedingly prevalent, so that Inozenzoff, the author whom Hirsch quotes, may, as Hirsch says, have been mistaken. Kennan also indicates the frequency of syphilis; and as neither Cook, nor King, who not only visited, but also travelled in the country (although the latter mentions a number of diseases he saw[7]), nor Beechy (1825), nor Kennan, who lived for some time in Kamtschatka (1868), mention

[1] See Travels of Rubruquis, in Kerr's Collection, vol. i. p. 183.

[2] Rubruquis, p. 196; and Marco Polo, 1260-95, in Kerr's Collection, vol. i. p. 272.

[3] Herodotus, lib. ii. 102 *et seq.*; and Lepsius, quoted in Humboldt's Cosmos, vol. ii. p. 487.

[4] Universal History, vol. xx. p. 31; and Herodotus, b. iii. c. 103.

[5] Herodotus, b. iv. 134. His whole army consisted of 700,000 men, so a great number must have been abandoned.

[6] Hirsch, p. 314, and Chapter on Syphilis, p. 358.

[7] King's Continuation of Cook's Journal (Voyages), vol. vi. p. 158. Second Voyage, 1772-74.

leprosy, it is probably a mistake. As the Kamtschatdales, however, were decimated by smallpox in 1767,[1] and the Empress Elizabeth compelled a great number of Cossacks to immigrate into Kamtschatka, it is possible that they may have taken it with them from their homes in the south of Russia. The same doubt exists as to its existence in the Aleutian Isles, where Dr Anderson, Cook's surgeon, makes no mention of having seen it, though he mentions cancer and syphilis.[2]

Let us now return to Western Europe. The decline of leprosy as a great epidemic, lasting for centuries, took place very much in the order in which it attacked each country; although countries *in which the condition of the people improved most rapidly, in which the strictest segregation and the most severe laws against leprosy existed,* got rid of it at a comparatively earlier period than those labouring under the reverse conditions; a marked instance of which is Norway, in which it lingers to the present day, the country being still poor, and only lately having passed laws encouraging segregation, and never having had severe laws affecting lepers.[3] Thus, in England it was disappearing by the end of the fourteenth century, and appears to have entirely disappeared about the early part of the sixteenth;[4] while it still existed, though rare, in Italy (where it began so many centuries earlier than in England) at the end of the fourteenth century,[5] but still lingered until the sixteenth century, and even thereafter remained in a few spots: it only exists in one spot now.[6] In France it was still common at the end of the sixteenth century,[7] though *tubercular* leprosy may not have been so common, and it had not entirely died out in 1789: a very few cases even exist in some parts of the south coast at the present day.[8]

In Germany it was still common in the middle of the sixteenth century, though dying out. In the Netherlands it died out in the middle of the seventeenth.[9] In Denmark it was gone in the middle of the sixteenth century. In Sweden a leper hospital was established as late as 1631, and many cases still remained at the beginning of the present century.[10] In Norway, as already stated, it still exists, though chiefly confined to the district of Bergen.

In Spain it almost disappeared later than in France, but it still remains in a few places up to the present century.[11] It still exists in Portugal.[12]

Returning to our own country, we find that it remained longer

[1] King's Continuation of Cook's Journal, vol. vi. p. 327.
[2] Vol. vi. p. 390, *et seq.*; and pp. 470 and 480 of Cook's Voyages.
[3] See Danielssen and Boeck, p. 184. [4] Liveing, p. 25 *et seq.*
[5] Beneveni (quoted by Hirsch, p. 308) and Dan. and Boeck, p. 182, says, in speaking of a case seen elsewhere, "morbus qui in Italia pene numquam visus." [6] Hirsch, p. 308. [7] Ibid., p. 308.
[8] Dan. and Boeck, p. 185, and Hirsch, p. 318. [9] Hirsch, p. 309.
[10] Dan. and Boeck, p. 182. [11] Hirsch, p. 318.
[12] See Peacock in Lancet, vol. ii., 1870, p. 775, and Hirsch.

in Scotland than in England, only being stamped out in the former country about the end of the sixteenth century, but effectually when it was so.[1] It lingered on in the Shetland Islands till about 1742, and one case was seen in 1798, in a man in whom it appeared to be hereditary.[2]

It disappeared about the end of last century from the Faroë Isles.[3] It was seen in Iceland by Robert Chambers[4] about seventy miles inland in 1855, but appears, from what Burton says, to have become extinct now.[5]

Thus we see that England was one of the first countries from which it disappeared, it being a country in which lepers were strictly kept apart from the healthy. They were driven out of London in 1346.[6] At the same time, from the time of the Norman Conquest, it was one settled kingdom in which no very long-continued intestine wars, with their direct consequences, famine and pestilence, ravaged the country. As a constitutional State, it encouraged by its laws internal improvements among the people, which were impossible in the down-trodden petty States of continental Europe, with their splendid kings and dukes and starving peasantry. Compared with France, which up to 1355 was a mere jumble of petty States, in which for five hundred years "anarchy and ignorance"[7] prevailed, and in which famines were frequent, and the poverty of the people, as compared with England, was horrible to think of,[8] and in which many were really slaves up to the time of the great Revolution;[9] as also with Germany, where famines were in the Middle Ages by no means unfrequent,[10] England was far advanced in comforts and civilisation, while they were still in a state of extreme wretchedness. No doubt, compared with the present day, the condition of the English people was poor enough, even the courtiers often eating bad food, as mentioned by Philip de Blois, and dearths being by no means unknown. I only wish to point out decidedly that the English, as a people, were better off, in the period under consideration, than Continental peoples, and rose much more rapidly above constant want; and I believe, as a direct consequence, leprosy disappeared earlier from among them. I may as well say here that I do not think it possible that simple want could ever *create* leprosy, but, as a secondary cause, it lays a population open to the attacks of the disease when the contagion is brought into a country; and, on the contrary, as I shall hereafter try to show, a flesh-fed population is protected from such

[1] Simpson, lib. cit., p. 325.
[2] Simpson, lib. cit., and Edmonstone's Ancient and Present State of the Zetland Isles, vol. ii. p. 102.
[3] Liveing, quoting Hjort, p. 31.
[4] Tracings of Iceland and the Faroe Isles, Chambers' Journal, Oct. 1855, p. 261.
[5] A Summer in Iceland, 1875, vol. i. p. 153.
[6] Simpson, 1842, p. 419.
[7] Hallam, Hist. of Middle Ages, p. 120.
[8] *Ibid.*, p. 19 *et seq.*, and p. 125.
[9] *Ibid.*, p. 106.
[10] *Ibid.*, p. 103.

contagion. Scotland, again, was always a much poorer country than England, and in a much more unsettled state. The English soldiers are said to have been astonished at the poverty of the Scottish camp after the battle of Pinkie (1547),[1] and oatmeal is still in a great measure the staple food of Scotch farm labourers (although by no means their exclusive food, as many English people think), just as it was two and more centuries ago in England.[2] This extreme poverty, combined with the disturbed state of the country up to and even after the Union, would tend in Scotland to counteract the good effects which, so far as stamping out the disease was concerned, were certainly calculated to be produced by the rigorous and barbarous laws enacted against lepers. At Greenside, near Edinburgh, they were not allowed to leave the hospital under pain of death.[3] A leper woman quick with child was buried alive.[4] The state of slavery[5] in which many of the people were steeped, with the abject poverty accompanying it, are also to be kept in mind.[6] Such laws would, no doubt, along with the general avoidance of lepers, assist in its total suppression; and we have to thank our forefathers for acting as they did, that we have not a remnant at least of the disease among us at the present day.

Going slightly back, and comparing the food of the Italians with that of the English, the latter will be found to have always been much superior, and, it may be remarked, included an abundance of one article which the Italians do not use—milk; yet of the value of which as an article of diet calculated to prevent the spread of leprosy even Galen seems to have had some idea, when he remarked on the absence of the disease among the Scythians—"lactis potatores," in a passage already quoted. Now, I have already shown that leprosy lasted a much longer time as an epidemic in Italy than England, where it seems only to have come in with the Saxons (at least the Saxons had words for it, though the first leper-house was built in 1101); at least five centuries after it began in Italy, yet it was as late, if not later, of dying out there, and did not do so entirely even then.

[1] Hugo Arnot, Hist. of Edinburgh, 1779, p. 55; yet it had improved from the fourteenth century. He states, p. 194, that Fletcher calculated 200,000 as the number of beggars in Scotland in the sixteenth century.

[2] Dodd, the Food of London, p. 77. The whole historical part of the work shows that from the fifteenth century, and even earlier, the English were a well-fed people on the whole, although partial local famines might occasionally take place from want of communication. Going further back, Matthew Paris is quoted in reference to a famine in which great numbers died in 1258 (p. 27). No doubt also much of the food eaten was salted, unless near the sea.

[3] Hugo Arnot, Hist. of Edinburgh, 1779, p. 194.

[4] Simpson, 1842, 418 *et seq.*; (Dan. and Boeck, p. 118, mistakenly quote this "burned" (brulé).

[5] Hugo Arnot, Hist. of Edinburgh, 1779, p. 52.

[6] By an Act of the Scotch Parliament in 1427 all lepers were to be secluded (Geo. Munro in Preface to Public Health Act 1867, p. 1).

Spain, where leprosy still lingers, has never been a settled country.

I have seen it stated that the present yearly consumption of flesh meat in Spain is only about 24 lbs., as compared with over 180 lbs. in England.[1] Dodd (pp. 248-50) gives the London consumption of 1750 as 70 lbs. per head; in 1840 and 1852, 120 to 140 lbs.; that of all the great towns of England, 100 lbs.: all far above that of Spain, while the increase in the English consumption is noticeable.

Now, passing to Norway, we find that at the present day the food of the people is as poor as that of England was some centuries back. Fresh meat is never eaten, and the greater part of the nourishment is from oatmeal, potatoes, and sour-milk.[2] Besides this, segregation has only been carried out lately; during the last twenty years, the disease up to that period, "as in other countries, showing no natural tendency to subside,"[3] but now diminishing rapidly since proper asylums were erected.

In Sweden, where the food of the people is now abundant but coarse, and milk, butter, and cheese plentifully used, as I have myself observed, and where meat is more common than among the peasantry of other countries, though unfortunately it is chiefly used salted,[4] there being an aversion to fresh meat, the disease has died out during this century.

In both these countries the food has somewhat improved of late.

In the Shetland Islands, where it lingered on for three-quarters of a century after becoming extinct in Scotland, Edmonstone,[5] in 1809, tells us that "the lower class lived chiefly on bread, milk, and fish," the fish being dried, and putrefaction favoured, it being in that state esteemed a delicacy. From what I myself saw in Shetland fifteen years ago, the condition of the poorer classes is now much improved, fowls and meat being plentiful and cheap, and I never saw any used that was tainted.

I need hardly say that Iceland is essentially a poor country, famines not being unknown in the present century, and driving many to America.

As to Greenland, there is a want of late information on the subject.

Thus, I think I have shown that England, in which the improvement in the condition of the people was most rapid, while segregation was carried out at the same time, got rid of the disease most rapidly; while the opposite conditions prevailing to a greater or less extent in other countries, it prevailed accordingly a greater or less time in them.

[1] This is from a newspaper cutting. I should be greatly obliged to any one who could refer me to the original calculations.

[2] Carter's Report, p. 31. [3] *Ibid.*, p. 28.

[4] Harper's New Monthly Magazine, Jan. 1871, "Folk Life in Sweden."

[5] Lib. cit.

We will now follow the disease to the Western Hemisphere, its introduction among the indigenous tribes of which is one of the strongest proofs of its communicability.

All authors writing on the subject agree that leprosy was first carried to America by the negro race: Schillingius[1] says, "Cum mancipiis Africanis in Americam pervenisse videtur," and "endemium Americæ morbum fuisse non puto;" and further on, "igitur non dubito quin ex Africa in novum orbem primum venerit."

Hillary[2] quotes from Town, an earlier writer, who says that the negroes brought it from Africa, and he was the first English writer who noticed joint-evil after Haly Abbas the Arabian. Peyssonel traced the importation of the disease into Guadeloupe in 1730[3] to negroes; and, later on, Brunel[4] mentions that it was brought from the coast of Africa to the Parana and Uruguay; while Bates[5] mentions (vol. i. p. 238) that numerous importations of negro slaves had taken place to the banks of the Amazon during eighty years back, and (p. 241) that a body of Portuguese immigrants came from Morocco in 1769.[6] Further on (vol. ii. p. 15), he mentions the great prevalence of leprosy at Santarem, a prevalence quite explained by his notice of the immigrations in the first volume.[7]

In support of the idea that negroes carried the disease to America, I may point out that they are known to have carried it to the Cape of Good Hope, where it was totally unknown among the Kafirs.[8] They also carried it of late years to India, to Tranquebar, about 1830.[9]

I may say here that at the time of Columbus leprosy appears to have reached the Canaries, at least they were discovered in 1344, and a leper hospital was built in them in 1542; in Madeira, discovered in 1420, a leper house was built in 1656.[10] In both these instances, no doubt, the disease existed some time before the leper houses were built, but both are examples of the long time required for the importation of the disease. The Canaries were, no doubt, infected from Africa, Madeira from Portugal, which,

[1] Lib. cit., c. xx. and xxi.
[2] Dis. of Barbadoes, p. 335.
[3] See Jahn, lib. cit., p. 84.
[4] Obs. topograph. dans le Rio de la Plata, etc., Paris, 1842, p. 46; and Hirsch, p. 311.
[5] Naturalist on the Amazon, 1863.
[6] This immigration was of Portuguese masters, belonging to an infected race, with their negro servants, coming from Morocco, a country where leprosy is an exceedingly common disease, and appears long to have been so. (See Jackson, "Account of the Empire of Morocco, 1809," p. 155, leprosy mentioned as jeddam); also Rolfe, in *Chambers's Journal*, Aug. 1874, p. 503; and Leared, lib. cit. (1876), p. 146.
[7] This prevalence is noticed by Liveing (p. 56), who quotes Bates's description of the glorious climate of Santarem, as showing that neither bad climate nor want of food could have caused that prevalence. Strangely enough, he does not point out *the* evident cause I have mentioned above.
[8] It is now most prevalent among the Hottentots (see Coll. Phys. Rep. p. xxx.) who are, and always have been, a filthy race, as Dampier, who in 1686 speaks of them as "Hodmandods," calls them.
[9] Hirsch, p. 313.
[10] Liveing, p. 52.

however, it can hardly be questioned, was largely infected about the year 1500 by the great number of negro slaves (about 700 yearly) who were then imported[1] from Morocco, an importation which doubtless has had a great effect in causing Portugal still to suffer so severely from leprosy, as it was continued till nearly 1730.[2]

As further proof that leprosy was carried to America by the negro races, we have the fact that it has never been mentioned as existing among any of the aboriginal races *until after they had come in contact with negroes.* In fact, from Boothia Felix to Cape Horn it was an unknown disease, and still remains so among peoples and tribes who have never come in contact with negroes, or races directly or indirectly infected by them; for instance, it is not mentioned by any Arctic voyager as existing among the American Esquimaux. Anderson and King never speak of it in their notices of the natives of the Pacific side of North America. Hewit, after a life of some years among the natives of Nootka Sound,[3] says nothing of it, and no voyager from Magellhaens to Darwin has ever seen it in Tierra del Fuego or Patagonia, nor does it exist, I am informed by an old resident, high up the Orinoco. As to its former absence in parts of the Western Hemisphere in which it is now common, I may say that no mention is anywhere made of it by Prescott in his works on the Conquest of Mexico or Peru, or by Diaz de Castillo, who fought and plundered by the side of Cortes; nor is it mentioned as existing there in any of the works on the life of Columbus I have read; nor is there any mention of it among histories of the Aztecs, in which their migrations have been traced from far beyond California to Mexico, long before the time of Columbus.

This is a great contrast to the present time, when many parts of America suffer more or less from it, and when *there is more leprosy in the British West Indies than in any other part of the British dominions*, in comparison with the amount of population, and perhaps more than in any part of the world,[4] except the Sandwich Islands.

It is now found all over the West Indies, though more severe in some islands than in others. Thus it is hardly known in

[1] Helps's "Life of Columbus," p. 27. He also mentions, p. 212, that negro slaves, "born in the power of Christians," were first allowed to pass to the West Indies in 1501. Columbus saw leprosy in St Vincente, Cape de Verde Islands.

[2] Moore's Travels into the Inland Parts of Africa, 1740, p. 9.

[3] Captivity among the Nootkas in 1804.

[4] It is possible that the great relative numbers I have quoted in my introduction, ranging from 1 in 280 in Demerara to 1 in 676 in Jamaica, may be exceeded in South China and Africa, but the want of statistics makes comparison impossible. However, the fact already mentioned that there was at least 1 person in 212 a leper in St Kitts in 1817, half a century *nearer* the time of their leaving Africa, and when there were many native Africans in the population, would tend to show that such is the case in Northern Africa.

Dominica,[1] while it is more common in St Vincent and Barbadoes, and also in Jamaica and the Bahamas. It also exists in Antigua, Montserrat, and Nevis, although no statistics of the actual numbers in these islands are obtainable. Again, in Grenada,[2] Tobago, St Lucia, and the Virgin Isles, it is said to be rare. Dr Liveing states that it is less common in St Kitts and Antigua than in Jamaica; but by the statistics I have already given, this is shown, at least as far as St Kitts is concerned, to be in all probability an error. As to islands not under British rule, it is known in Cuba and Porto Rico, in St Domingo,[3] in St Thomas, St Bartholomew, and St Martin's. In Martinique and Guadeloupe, Dr Brassac of Basseterre, Guadeloupe, *estimates* about 150 cases in each island,[4] or *about* 1 in 860 of the population, or (if the estimate be correct) less than half of the relative number of St Kitts.

In North America the disease is almost unknown, except in one isolated spot, the Bay of Chaleurs, New Brunswick, and chiefly among some poor French families; although it is to be remarked, that one Scotchman has been known to take it, and that his family were affected after him, and some few English settlers have also been attacked.[5] It is uncertain how it was first carried to New Brunswick. Had it spread only among the French colonists, it might have been considered only hereditary, but its passing to other uncontaminated families proves that it is communicable, while its descent from one who got it by contagion may have either been from heredity or from the constant contact of his family with him. The question as to the spread in such cases being caused by the disease being *endemic*, I will consider further on.

It has been stated to me that cases occur in the Southern States of America, and such cases are seen among the blacks at Baltimore, but whether of blacks from the West Indies or natives of the States, I have no information.

In Central America, leprosy is known in Mexico,[6] where it was certainly brought by the negro races, but where the present miserable poverty-stricken state of the population[7] makes them ready for its attacks. It is unknown in Nicaragua, and apparently also on the Mosquito coast.[8]

[1] Milroy's Rep., p. 2.

[2] Bakewell, in Rep. on Vacc. Act, p. 208, says the Governor of Grenada told him there were only five or six lepers in the island.

[3] Cazenave and Schedel, p. 355.

[4] Private letter. He states that there are 50 or 60 lepers at la Desirade, where the lepers from Martinique and Guadeloupe are sent, but that many remain at home concealed (cachés). There is not now any law for their compulsory segregation, though there was formerly.

[5] Coll. Phys. Rep., pp. 1–3, and 29.

[6] Simpson quoting Cheyne, lib. cit., 1842, p. 410, and Hirsch, p. 320.

[7] Canon Kingsley (*Good Words*, 1873, p. 559) on "Spring in Mexico." He says, there are "magnificent churches all over the country, surrounded by two or three dozen huts, more fit for pigs than human beings, leaving the Indians around sunk one step lower in poverty, superstition, and ignorance."

[8] Hirsch, p. 320.

In South America, it is known in Guiana[1] (where many authors agree that it was carried by the negroes), in the Brazils, New Grenada, Parana, Uruguay,[2] and Venezuela,[3] these being the very states into which negroes have been imported most directly from Africa.

Now, had the disease remained known only among the African race, the fact of its being prevalent in the New World would have been no argument in favour of its communicability; but the very contrary is the case, *for wherever the indigenous tribes have come into constant contact with the blacks or Portuguese they have become infected.* The case of the North American Indians is no exception to this rule, for they have never been affected by leprosy, but they have always kept aloof from and despised the blacks, while the whites they have come in contact with have been chiefly English and Germans—races unaffected by leprosy at the time of their first contact with the red man. And, besides, they were a nomadic, and, consequently, flesh-eating people—another reason for their immunity.[4] But among tribes in contact with infected races the case is different. In regard to the indigenous tribes of Surinam, Schillingius expressly states,[5]—"Nam licet hodie aborigines eo (i.e., morbus) passim laborent, sinit tamen integræ gentes ab eo prorsus immunes, atque in illis etiam tribubus quas jam attigit *eos tantum affectos esse deprehendimus qui cum Ethiopibus corpora sua miscent, aliarumve rerum commerciis jungantur*," showing, especially by the part I have italicised, that, although some tribes remained free, those tribes brought most in contact with the blacks were most infected. Bates, again, in speaking of the disease at Santarem, says that all races were affected alike, white, Indian, and negro.[6] The same may be said of Mexico. Brassac also speaks of several *Indians*, natives of Venezuela and Trinidad, who were lepers.[7] I am aware that Milroy[8] notices that the Indian tribes in Essequibo (Guiana) are exempt from the disease, and their freedom from it is attributed to their not eating salt fish, and to their dwellings being cleanly and well ventilated; but as these Indians seldom or never mix with the negroes, and the country was originally settled by the Dutch, the immunity is much more likely to have been the result of absence of communication with the blacks, while the Dutch were, as a nation, nearly free of leprosy before Guiana was colonized,[9]

[1] Hirsch, p. 321. [2] *Ibid.*, p. 326.

[3] Brassac, Report Addressé au Directeur de l'Interieur (on Beaupathuy's treatment), Gaudeloupe, 1869.

[4] It is possible that to the fact that no nomads *can* be solely vegetable-feeders that their immunity from leprosy can be traced.

[5] Dissertatio xx. [6] Lib. cit., vol. ii. p. 15.

[7] Rep., p. 28, etc., also quoted by Bakewell. [8] Rep., p. 9.

[9] Guiana was colonized in 1580, and the Netherlands were quite free of leprosy in the middle of the next century, so that the cases must have been at that time (1580) few and far between—while it is to be remembered that it was only hardy men who would go as colonists to such a place.

unlike the Portuguese, who still remain affected. The North American Indians are a filthy race,[1] yet they are not affected, so that simple dirt cannot produce the disease; so that no arguments can properly be founded on such instances as those mentioned by Dr Milroy.

I may here notice, as another strong proof of the communicability of leprosy, its spread to whites who have either themselves come from uninfected parts of Europe, or are the descendants of such,[2] and who consequently can have no hereditary taint, yet become affected after a more or less prolonged residence in the West Indies or other places where leprosy is common. The value of such proof has been questioned, on the ground that such cases are only examples of certain endemic influences causing the disease, or of the disease occurring in those with a personal predisposition.[3] But such a supposition only adds one difficulty to another, for if there is such a personal predisposition why does it never show itself except in persons living in countries infected by leprosy? As to the contagiousness of the disease being explained away on the idea that leprosy is endemic, and that cases arising in Europeans in the West Indies and such places are caused by endemicity simply, I would simply ask those advancing such a theory, how it is that in the West Indies, and all over the Western Hemisphere, and all over the Pacific Ocean, with the exception of New Zealand, a possible exception which only proves the rule, no such endemicity has existed until an infected race was brought into contact with, or took the place of, the original inhabitants. It may doubtless be said that the habits of the inhabitants have changed, and that these changes have tended to make the disease endemic; but this is so far from being proved, as I will hereafter show, that every one of the causes, such as filth, want of ventilation of houses, and the like, which have been put forward as their cause, are, on careful consideration and a broad view of the subject, quite inadequate to the *production* of leprosy, whatever may be their power in assisting in its propagation when it once has taken hold of a population; such supposed causes having, in fact, existed among many populations where leprosy has or still remains unknown, so long as no infection has been introduced among them.

One remarkable instance in which leprosy was acquired without the possibility of any endemic influences acting, is that quoted by Hutchinson, referred to by me above, of a Scotch sailor who became a leper after trading for about thirty years to Barbadoes, where he only spent about six weeks each time, and lived in his

[1] Paul Kane, in *Household Words*, 26th March 1859.

[2] I have seen such cases myself among *poor* whites in St Kitts. Many are also referred to among works on the subject, as in Hebra, vol. iv. p. 184; Milroy, Rep., pp. 3–10; Coll. Phys. Rep., xxxviii., pp. 3, 20, 85, 198, etc.; Virchow, lib. cit., p. 507; Macnamara, p. 56; Bakewell, Rep., p. 61; and Hutchinson in New Sydenham Soc. Catalogue to Atlas of Skin Diseases, p. 96.

[3] Carter, Rep., pp. 24 and 26.

ship almost the whole time.[1] Now leprosy is essentially *slow*, and it cannot be imagined for one instant that this man's occasionally (twice or three times at most each voyage, or perhaps six separate times a year) spending a night on shore could have caused the disease to arise through the slowly-acting endemic influences which have been put forward as causing it. I have already shown how absurd it is to argue that because in such cases no history of contact with a diseased person can be obtained, owing to the long period of incubation, therefore it could not have been caused by contagion —the only cause I consider we can really look to to explain away all difficulties. In short, it is, in my opinion, an utter misuse of the word endemic to attach to it necessarily the idea of causation of disease, as certain diseases may exist now among a people, and so be strictly endemic, and yet have been imported, as I believe leprosy has been, to the New World, so that it cannot be looked on as an indigenous disease—which, after all, is what a disease capable of arising at any time, simply from the existing conditions of the soil, unless those conditions themselves are changed, must always be, as for instance malarious fevers. Leprosy is *not* an indigenous disease in the West Indies; no sufficient cause or causes for its origin there have ever been shown to exist; therefore, the proposition that certain cases may have been affected with it, simply because it was an "endemic" disease, amounts to nothing, and is of no value whatever. No doubt the conditions of life of the present inhabitants are different from those of the Caribs, the huts of the negroes being close and ill-ventilated, and they being anything but cleanly; but the houses of the Esquimaux, or the Yourts of the Koriaks, as described by Kennan, are far worse in that respect, while within the tropics the houses of the Mexicans and Peruvians, as described by Prescott, were equally "civilized," in so far as they were close, not mere open huts like those of the Caribs, yet among neither of these peoples was leprosy known.

From the Western Continent we now pass to the Isles of the Pacific, in none of which, with the exception of New Zealand, and, it is possible, Fiji,[2] was leprosy known up to the year 1848.[3] I make this assertion after a careful perusal of the

[1] Within my own knowledge, it is very rare for sailors to spend a night on shore, and I have known of them spending weeks in harbour at St Kitts without being ashore at all.

[2] *Ibid. ante*, Nov. 1876, p. 436, footnote.

[3] I make this assertion in the full knowledge that Bougainville called one of the Friendly Isles the Isle des Lepreux (Kerr's Collection, vol. xi. p. 503), that Cook speaks of a case of leprosy, *or some scrofulous disorder*, at Anamooka, one of the same group (Voyages, vol. iv. p. 19), and that Ellis ("Polynesian Researches," vol. ii. p. 19) speaks of "a kind of leprosy" at Tahiti. The last, from his description, as I have already said, is evidently simply leucoderma, as "it turns the skin of the parts affected white." Bougainville probably saw cases of scruffy skin caused by ava-drinking, which every voyager, from Cook to Boddam Witham, writing in 1876, has described as producing fishy eyes and scaly skins (p. 156); but as Bougainville never actually landed on the

accounts of the voyages of Magellan (1522), Schouten and Le Maire (1615), Dampier (1685), Clipperton (1705), Rogers

island, except for a very short time, and only saw some of the natives *while fighting with them*, his statement is of no value, even had it been likely or even almost possible that *real lepers could have fought;* besides, he was a colonel, not a medical man. As to Cook's case, it must be examined rather more in detail to show that it was *not* one of leprosy, but really a case of syphilis or scrofula. Firstly, Cook mentions that, "on his first visit, entering a hut, he saw a man with his nose eaten away," and describes the disease thus, as seen on his third voyage :—"It is very frequent, and appears on every part of the body in large broad ulcers with thick white edges, discharging a clear thin matter, some of which had a very virulent appearance, particularly those on the face, which were shocking to look at. And *yet we met with some who were cured of it*, or in a fair way of being cured, but with loss of nose." "*Notwithstanding the similarity of symptoms*, it cannot be the effect of the venereal contagion" (vol. v. p. 403). Thus, in the parts I have italicised, Cook himself disproves his own idea, for, leprosy being very rarely a curable disease, the number of cured cases, or cases in process of cure, he met with, showed it was not leprosy ; and as he only founds his assertion that it could not have been syphilis, on his belief that he was the first voyager who had reached these islands, and therefore his crew the only means of communicating it to them, it is quite clear, from the expression I have italicised in regard to the symptoms, that, had he known of such former communication, he would have thought it syphilis. But such communication had actually taken place, the very same spot having been the landing-place of Tasman in 1643, a date at which the great epidemic of syphilis, which began in Italy in the end of the fifteenth century, had not yet died out in Europe Again, Bougainville actually visited Anamooka fully a year before Cook, and spent some days there. This seems not to have been known to Cook ; and although Bougainville accuses the English of conveying syphilis to the Society and other South Pacific isles (Kerr's Collection, vol. xiii. p. 502), as he had his men examined before allowing intercourse between them and the natives ; yet, as Cook himself says (vol. vi. p. 180), "however confident we may be of the health of our men, we are often undeceived too late,"—an observation the force of which can now be seen in the light of the newest investigations, which show that every secretion of the body may convey syphilis (see for instance Morgan, in *Br. Med. Jour.*, 14th March 1874 ; also see *Lancet*, 13th June 1868 ; Drysdale on Syphilis, p. 48) ; thus it is quite possible that Bougainville's crew carried it. Besides, Wallis, in 1767, more than a year before Cook, visited some of the same group, the Friendly Islands ; and he remarks (Kerr's Collection, vol. xii. pp. 222–23), that though there was no kind of metal in any of the islands, the natives knew that *iron* could be *sharpened*, showing that even previously to his time there had been communication with nations using iron, and consequently opportunities for the introduction of syphilis.

Even so early as 1522, Magellan, in speaking of Luzon, says that the lues venerea was common there "and in all the islands of this great archipelago." Mendana also passed through the Georgian Isles in 1567. Of course Magellan only refers to such islands as he had seen, but the fact of the disease then existing makes it more than probable that, considering the constant communication between those islands, of which there is an almost continuous chain, from those visited by Magellan and Mendana to the Friendly Isles, apart from the possibility that these islands had been infected before from China, where syphilis is stated to have existed centuries before Christ (Dabry, La Médecine Chez les Chinois, p 229, *et seq.*), syphilis had spread to the Friendly Isles before 1768. Thus there had been abundant opportunity for the introduction of syphilis into the Friendly Isles even from *recorded* voyages ; but as Cook himself mentions that syphilis was introduced into Queen Charlotte Sound (vol. v. p. 194) by an unknown ship, and as Carteret

and Courtenay (1708), Roggewein (1722), Carteret and Byron (1764), Wallis (1766), Bougainville (1766), Cook (1768), etc., and the accounts of different islands given by Mariner (Tonga or Friendly Islands), Herman Melville (Marquesas, 1842), "Dash (Six Years among Savages in the Marquesas"), also, "Rovings in the Pacific, by a Merchant," and Ellis's "Polynesian Researches," being, in fact, all the works I could obtain giving me any information about those islands up to the date I have mentioned. In many of them notice is particularly taken of the good health of the natives, especially of those islands furthest from the Asiatic continent, and which had had least communication with strangers. Thus, Wallis says that, at Otaheite, "we saw no appearance of disease" (Kerr, vol. xi. p. 216), and distinctly states that there was no *syphilis there* at the date of his visit. The same is said by Dash and Melville in regard to the Marquesans, among whom, according to the latter, "sickness is almost unknown," there being "on their

speaks in the same manner as Wallis of finding natives of the Carteret's Isles acquainted with the use of firearms, though he was the first *recorded* visitor (1765), there might have been many opportunities, besides those known to the world, for the introduction of the disease.

Thus, Cook's conclusion that the disease he saw was *not* syphilis, simply because there had been no possibility of that disease being introduced, falls to the ground. That it *was* syphilis might be more difficult to decide. On this point, the evidence of Ellis ("Polynesian Researches," vol. ii. p. 14) is of value, as it is in regard to the effects of syphilis in the same race, though in another group of islands. He says of the South Sea Islanders, "There are many cases of deformity arising from a disease of foreign origin affecting the features of the face and muscular parts of the body." This was written after ten years' residence in the Society Isles from 1816, and reminds us at once of the disease described by Cook, in which the *nose* was eaten away.

On the other hand, Thomson, in describing the diseases of New Zealand, and among them Ngerengere, the leprosy of that country, expressly states that he *never saw a native without a nose* (*Med. Chirurg. Rev.*, Ap. 1864). As to the possibility of its being scrofula, as Cook admits, however, the following description of a disease called "palla and cei," in which the generative organs are never affected, to which the natives of the Friendly Islands were very subject about 1810, is very much to the point. The people are very subject to scrofulous indurations, glandular enlargements, and *ulcers*, chiefly in groin, axilla, and neck, "sometimes to such an extent that some travellers have mistaken them for lues venerea (it is possible Magellan did so); and it is certain that some individuals with palla have been obliged *to submit to* the loss of the nose, the cartilaginous and softer parts of that organ becoming completely destroyed." (See Martin Mariner's "Account of the Natives of the Tonga (Friendly) Islands," London, 1817, p. 267.) He was among them several years. He mentions that "palla" gets well spontaneously, thus agreeing with Cook's description of the "leprosy or scrofula" he saw at one of the same islands. Thus, I have no doubt that the disease was *not* leprosy, but probably scrofula, and possibly syphilis or the latter acting on scrofulous subjects.

In conclusion, I may remark that Cook's ideas of leprosy were evidently confused, as he mistook dried salt on the skin, caused by constant immersion in salt water, for leprosy. This was among the New Zealanders (vol. ii. p. 46); and he also speaks of the Otaheitans having "cutaneous eruptions of the scaly kind, very nearly approaching to leprosy." Thus, he evidently looked on psoriasis as a kind of leprosy.

smooth, clear skins no blemish or mark of disease" ("Residence in the Marquesas," p. 141).

I have also carefully noted in all these works all remarks made about disease of any kind, and have found elephantiasis, scabby eruptions, albinism, ulcers, and deformities of various kinds mentioned, but nothing except those few instances I have noted indicating anything like the existence of true leprosy in any of the Polynesian Islands.

At the present time, however, leprosy has reached the Sandwich Islands. The history of its introduction is as well told as it possibly can be by Dr Hillebrand, for it must be kept in mind that such an inquiry has to be made *years after the first contagion is introduced into a country;* such being of necessity the case, from the slow action of the disease; and, besides, it is among an uneducated people, little inclined to make observations in regard to such matters. He says:[1] "In the Sandwich Islands leprosy was unknown before 1859, and, after close scrutiny, cannot be traced farther back than the year 1852, or, at the most, 1848." From a Government census, there were, about 1865, 230 lepers among 67,000 natives, or $3\frac{1}{3}$ per 1000; but Dr Hillebrand thinks that anæsthetic cases were generally omitted from this, and calculates the real ratio as 4 per 1000. He first recognised the disease in 1859, but remembered cases as far back as 1853, and says, "Further inquiry among the natives at length brought to light that a few had been observed in 1852 and 1851; and an old chief, well versed in everything pertaining to his countrymen, referred the first case known to him to the year 1848." In 1859, when he first drew the attention of the Government to its existence, only a few cases became known, but in 1864 and 1865 it was common. "Soon after the character of the disease became known, the natives began to call it 'Mai pake,' the Chinese disease." He "was not able to ascertain whether this was from a belief that the disease had been imported through Chinamen, of whom there have been a considerable number settled at the island

[1] Letter to Dr Ch. Macnamara, appended to his paper on Leprosy, p. 53, dated at Calcutta 3d Feb. 1865. Dr Boeck (in Carter's Rep. on Lep. in Norway, p. 45) says that "a whole series of observations would be required to establish a scientific proof that the disease was imported by the Chinese in 1848; and, from observations in Norway, considers Dr Hillebrand's report a total misunderstanding. But in this Dr Boeck demands what, from the very nature of the circumstances, we can never really expect to obtain, and it is surely more unscientific to reject such evidence, because it does not come up to some impossible-to-be-reached standard, than to accept it for what it is worth, when carefully compared with that obtainable from other countries. I must confess also that I think Dr Boeck's assertion very vague, and that I cannot understand how observations made in Norway—a country in which leprosy has been known for centuries, and is now on the decline—could have any bearing on the question of the mode of origin of the disease in the Sandwich Islands, where it has begun so lately, and is rapidly on the increase.

Dr Boeck seems also to be in error as to a matter of fact, when he says that the disease spreads among particular families, as in Norway. Dr Hillebrand points out that the very opposite is the case.

for years," or simply because the Chinamen had told them that the disease was common in China.

Thus, leprosy has been introduced and spread without the possibility of hereditary taint. Dr Hillebrand only saw one child under six years of age, and only one case of father and child. Yet he points out that the state of the people has in every way improved from their former state: food of all kinds is abundant, "but I would like to remark here," he says, "their food is the same as it used to be, a paste formed of the tubers of the *Colocasia esculenta*, richer in gluten than any other." This is, as it was when Cook discovered them, still their *chief* diet, and is eaten when partly putrid.[1] Athough animal food may now be, as Dr Hillebrand says, within the reach of every one, a national taste is not easy to change, and the Sandwich Islander will still prefer his dish of pooee or poi, as the paste was called, to animal food, which for ages has been tabooed to him, for, when they were discovered, the use of such food was almost entirely confined to the chiefs.

As regards their houses, Dr Hillebrand says, "Their former dark and damp straw huts are rapidly making room for pretty wooden structures, raised from the ground, and *well aired*." He mentions that their constitutions have been sapped by syphilis. Many of those affected are well off.

As to its diffusion, he first saw it in 1853, about twenty miles from Honolulu; in 1861 this case was far advanced, and six persons *in his immediate neighbourhood* had been taken ill with it. "The natives are of a very sociable disposition, *much given to visiting one another*, and hospitality is considered a sacred duty by them." The greatest number of cases are at Honolulu, the capital, while "at the time the census was taken, one or two of the remotest districts of Hawaii, *which have but little intercourse with the rest of the group*, were yet exempt from the disease. When asked, about one-fourth avow contact with other lepers as the cause—a proportion which may be considered high, considering the shortness of time that the disease has been known, and the long period of incubation, during which the poison must lie dormant in the body before it manifests itself." "In one family, I hear, a brother, sister, and all individuals between fourteen and thirty-five years, hereditary taint is out of the question." Dr Hillebrand's observations refer to tubercular leprosy. In almost all the cases there was anæsthesia, and generally squamous eruption.

I have referred at some length to Dr Hillebrand's most interesting letter, which, in my opinion, gives as complete an account as can possibly, from the nature of the circumstances, be expected, of the introduction of leprosy among a previously healthy people *some time after* they had first come in contact with the Chinese, an

[1] Boddam Witham, "Pearls of the Pacific," 1876, p. 33. The same remark is made by many other travellers in regard to the Marquesas and other islands. He mentions that pork is still reserved for the chiefs in Fiji (p. 345).

infected people—exactly what had taken place in other countries of which I have already spoken—although in them we have not so clear an idea, as a rule (except in Gaudeloupe), as to the exact date within a decade of its introduction. I hold that it matters not that it is now utterly impossible, and was so at the time Dr Hillebrand began his inquiry, to trace the exact source of contagion, the exact individual Chinaman from whom the first native was infected, seeing that the time of contact must have been many years previously; but the broad fact remains, agreeing with similar facts elsewhere, and until some other reasonable hypothesis can be advanced, and I candidly confess I can think of none, I consider that we are forced to the conclusion that the disease, being carried by human intercourse between two distinct races, was conveyed by contagion. This conclusion is supported by the words quoted which I have italicised; the second six cases (noted in 1861) seen, were all in the *immediate neighbourhood* of the first case seen in 1853; and he remarks that other instances of the same kind came under his notice. Again, we have the fact that those places with little intercourse with other parts (like the centre of Russia) remain longest or totally free from the disease.

The spread of the disease has been like that of all epidemics at their commencement—fearfully rapid—and reminds us of its behaviour in Europe after the Crusades, when everything was in its favour. There were last year no less than 700 lepers in the Leper Settlement which has been established by Government 20 miles from Honolulu,[1] so that, even supposing that to be the whole of the lepers in the group, which is most unlikely, there is (even if the population is the same, 67,000, as formerly, which is hardly likely, as the tendency throughout Polynesia has been to a decrease of population through the ravages of syphilis and other causes) no less than 1 leper in 97, or the greatest relative number in any population in the world—far surpassing even the West Indies.

Again, I would call attention to a remark of Dr Hillebrand's I have italicised, as to the natives being sociable, *and much given to visiting*. Here we have, I have no doubt, at least one explanation of the rapid spread of leprosy, and another argument in favour of its being contagious.

Having already said what I think about the so-called leprosy of Fiji, so far as we can at present speak of it,[2] we will now pass on to consider that of New Zealand.

[1] Boddam Witham, lib. cit., p. 63.

[2] I do not *deny* that Dr Seeman's case, or rather Mr Moore's, for Dr Seeman only reports it at second hand, was one of leprosy. I only consider our evidence on the matter defective. It is much against the idea of its being leprosy that Dr Forbes ("Two Years in Fiji, 1875") makes no mention of the disease, though he mentions several diseases, as dysentery, the effects of "kava" drinking, etc. (pp. 170 to 194). I have written to Fiji.

Note.—While the foregoing sheets were in the press, I have read for the first time Landré's excellent work ("De la contagion seule cause de la propagation

The only account we have of the leprosy (?) of New Zealand, called by the natives "ngerengere," or "Tuwhenna," is that given by Dr Thomson.[1] His description resembles closely that of West Indian joint evil, the so-called anæsthetic leprosy, except that, in the six cases he saw, there was no anæsthesia. From my experience, however, of that kind of leprosy, I cannot but coincide with the opinion emitted by the Royal College of Physicians, to the effect that the term "anæsthetic" is a misnomer, as anæsthesia, or rather as I prefer to call it "analgesia" (for it is the sense of *pain* that is lost, not that of feeling entirely[2]), is much more readily distinguished in tuberculated than in non-tuberculated leprosy or joint evil, in fact—in many cases of the latter I could not discover it at all, while in advanced cases of the tuberculated kind it was always present, and I have even discovered it in a case of only about seven months' standing. In some advanced cases, not only the tubercles but the whole body is analgesic. Thus, as Dr Thomson's description of the blisters on the fingers, followed by dry ulceration, but preceded by an eruption which appears and disappears exactly as described by Carter,[3] while the face becomes swollen and shining, and the eyeballs exposed—as his description tallies in a great measure with what I have seen in the West Indies, although in cases of joint evil there, there is *no* swelling of the face or nose, and there *is* paralysis of the orbicularis, and consequent falling of the lower eyelid, and exposure of the conjunctiva (symptoms not shown in Thomson's plate of one case, which has rather the appearance to me of the portrait of a sufferer from mixed leprosy), and as I do not attach very great importance to the mere absence of anæsthesia, or rather, as I should prefer to call it, analgesia, analgesia being, although *when it is present in conjunction with other symptoms*, undoubtedly pathognomonic of leprosy, yet much more easily discovered in the tuberculated than the non-tuberculated form[4]—I am inclined to admit that "ngerengere" *may* be a peculiar variety of leprosy, but, like Virchow,[5] do not consider that it is decidedly proved to be the actual specific disease,[6] to

de la Lépre," Paris, 1869). He gives (p 20) a case of an *Indian* who contracted leprosy after constantly frequenting the leper-house, showing an instance of what I have referred to at p. 31. His work as a whole shows what I myself have tried to demonstrate—that the proof of the contagion of leprosy is more to be sought for in its history than in mere cases.

[1] "Diseases of New Zealanders," *Med. Chir. Review*, April 1854, p. 496, *et. seq.*

[2] This remark only applies to the *cutaneous* nerves; pain may still be felt during destruction of deep-seated structures.

[3] "Trans. of Med. Soc. of Bombay, 1862," "Report on Leprosy and Elephantiasis, 1874."

[4] Brassac mentions that anæsthesia is not always present, and Carter (Mem. on Leprous Nerve Disease, in Path. Soc. Trans., 1877) speaks of individuals in whom the nerve affection is very limited, as to the hand below the wrist.

[5] Granulations geschwulste, p. 528, footnote.

[6] It would be of great interest, if the disease is not yet extinct, for some

which, however, it is the nearest approach of any yet seen, and between which and ergotism it may be a connecting link. It is worthy of note that, as stated by Thomson himself, Dr Shortland looked on it as a variety of such a disease as ergotism,[1] and other medical men thought it was a kind of scrofula; so that, as even observers on the spot have not agreed as to its nature, I am inclined to look on positive opinions, such as have been expressed by Liveing,[2] who says that "no one can possibly doubt its identity with elephantiasis Græcorum," after reading Thomson's description, as being much too hastily arrived at, especially as while calling it "lepra gangrenosa," Thomson himself distinctly states that all the patients he saw were highly scrofulous—showing possibly some doubt even in his mind as to the real nature of the disease. In conclusion, as to this point, I may remark that the duration of the disease is much shorter than that of anæsthetic leprosy, which it most closely resembles, being one to eight years, while in St Kitts I found the average duration of such cases to be seventeen and a half years, some living to periods far beyond that average—thus showing a difference from ngerengere too great to be accounted for simply by difference in the conditions of life in the two countries.

As to the history of ngerengere but little seems to be known. Thomson seems to have ascertained to his own satisfaction that it was much more common twenty years previously, and that it existed before Cook's discovery of the country. It is found chiefly in the *interior* of the North Island, but also on the coast, and in the Middle Island, and probably was formerly only known in the interior, as Cook himself is most emphatic as to the good health of the natives. After several weeks' visit to the coast, he says, "In all our visits to their towns, we never saw a single person who had any bodily complaint."[3] Savage, whose account, as he was a surgeon, is of special value, says also, "Neither the accounts nor the appearance of the natives indicate the prevalence of disease."[4] Thus, it does not, a century and half a century ago, appear to have been known on the coast, and Thomson himself mentions as a proof that it is not a *syphilitic* disease, that he had heard of fewer cases at the Bay of Islands than anywhere else, whereas, had it been so, there ought to have been more, as Cook landed there. In

New Zealand practitioner to try to settle this point. Perhaps the presence or absence of the temporarily so called leprous elements, described by Carter (in the Path. Soc. Transactions, 1876 and 1877), might assist in clearing up the diagnosis—that is to say, if any cases still exist in the North Island. The disease was dying out when Thomson saw it. Of course, the simple presence of these brown leprous elements would not of itself be sufficient, as Carter has seen them in other skin diseases, but, taken in conjunction with the other symptoms, their presence would go a great way in deciding the matter.

[1] "The absence of intoxication, and of the anæsthesia which generally precedes gangrene, is against this." (See Lasegue, Arch. Gen. de Med., May 1857; and Hirsch, p. 456, on Ergotism.)

[2] Lib. cit., p. 63.

[3] Vol. ii. p. 46.

[4] "Account of New Zealand, particularly the Bay of Islands," by John Savage, Surgeon, 1807, p. 88.

the present settled state of the country the few cases on the coast might have migrated from or become infected with the disease in the interior.

As to whether the Maories brought the disease with them when they reached New Zealand about four and a half centuries back,[1] or if it has been developed among them since, we have no positive knowledge, but I am inclined to the latter opinion, as, had it always existed among them, it would not have been *entirely* confined, as it evidently was last century and the beginning of this (if, as Thomson's researches would seem to show, it then existed), to the interior; had it not been so confined, either Cook or Savage must have seen something of it.

I will consider hereafter the etiology of the disease in New Zealand.

Turning to Australia, we find that the disease is known in Victoria, but *only* among the Chinese.[2] Up to last year this was still the case, as I am informed by a letter from the Central Board of Health (for which I have much pleasure in thanking the officials), with which I may say was also enclosed some conclusions come to, I think rather hastily, by the medical officers of that body, to the effect that its not having spread among other races proves its non-contagious nature. I think this can be much more reasonably explained by the fact that, in Australia, mutton has of necessity to be used three times a day by the great mass of the population—in fact, no population in the world, taken *en masse*, is so thoroughly flesh-fed as the Australians. Besides, there is no predisposition through bad health to take the disease, Australian immigrants being necessarily as a mass healthy individuals; so that, as bad general health predisposes to leprosy, they are not exposed to it. I may here observe, that there were only 15 lepers among 10,385 Chinese in Ballarat, Castlemaine, and Beechworth, or 1 in 692, probably, from the accounts given of its enormous spread in China already referred to, much less than the relative number which obtains in that country,—a result probably brought about by the fact, that it is only people who are at the time in good health who can emigrate.

Leprosy is found in Japan,[3] but nothing is known at present of its history in that country.

It is also seen among the natives of Java and Sumatra both in the interior and on the coast,[4] also in the Malay Islands, and in

[1] Trollope, "Australia and New Zealand," 1873, vol. i. p. 302.

[2] Coll. Phys. Rep., 1867, pp. 14 and 80. Liveing says (p. 92), writing in 1873, "Lately it has been reported that the disease has spread beyond the Chinese population." It is evident, therefore, from what I have stated, that this report is an error, and may have arisen from the disease having been seen by Hutchison in one European, who, however, contracted it in India.

[3] Ashmead in *Doctor*, May 1875 (quoted from *Phil. Med. Times* of 14th January.

[4] Hirsch, p. 314.

Singapore, Penang, and Malacca, where the Chinese are chiefly affected, though it is common among the Malays and Dyaks.[1] It is not seen among the Arab races *there, they having no communication with other races.*[2] In British Burmah it is chiefly seen among the immigrants from Bengal.[3] On the west of the Indian Ocean it is common in Madagascar, the Mauritius, where it was imported in the middle of last century,[4] and Mozambique.[5]

We have thus historically completed the circuit of the earth, but as I formerly had occasion chiefly to speak in reference to its ancient history only, in Asia, to be more complete, I must speak of it as it exists there at present, so completing the geographical history of the disease

I have already spoken of it in China, Thibet, and Yarkand, but as to India, where, though so little attention has been paid to it (it never having, as among the Jews, had any great notice given to it among the religious codes or laws, possibly because the relative numbers affected are small, though the aggregate is hideously large), it is known from Ceylon to the Himalayas, I would wish to say a little more.[6] I find from the censuses of the following provinces of 1871 and 1872, comprising nearly the whole of India, viz., Bengal, Madras, Bombay, the Central Provinces, the Punjab, the North-West Provinces, Oudh, Coorg, and Mysore, that the total number of lepers then enumerated was 99,639, or 1 in 1864 of the population; but as I have already stated, about 1 in 1500, or 120,000, would be I believe nearer the truth. My grounds for this belief are that the proportion of females to males, as enumerated, is so small, being only 1 in 5 in the whole of India, a proportion which obtains nowhere else in the world, while it varies from 10 to 67 in Bengal, 10 to 66 in the North-West Provinces,

[1] Macnamara, p. 13, and Coll. Phys. Rep. p. 31 Ida Pfeiffer ("A Lady's Voyage Round the World") states that there are in the island of Singapore 40,000 Chinese and only 10,000 Malays in 55,000 of a population, and that the Chinese and Bengalese are *almost exclusively males*— a fact of some importance.

[2] Landré, p. 40. This is a strong negative proof of its communicability, the Arabs being by no means exempt, *as a race*, from the disease when *exposed to communication with an infected race*, as with the negroes in Cairo and Western Arabia, or the Moors and Kabyles in Algeria.

[3] Coll. Phys. Rep., p. 194, and Macnamara, p. 9. It is probable that the facts that at Akyab, where the above remarks chiefly refer to, the Burmese population is omnivorous, unlike the vegetarian Hindoos, and that the Burmese laws treat lepers as outcasts and malefactors, have much to do with its rarity among the natives. I may say that I have not been able to find mention of it in two or three books of travel on the "Land of the White Elephant," as Siam is called, that I have consulted.

[4] Coll. Phys. Rep , p. 219.

[5] Hirsch, p. 311 ; Cazenave and Schedel, p. 355 ; and Coll. Phys. Rep., p. 83 and Appendix.

[6] It being impossible in a work of this kind to do full justice to the subject of leprosy in India, those desiring fuller information must refer to the references I quote.

10 to 26 in Bombay, 10 to 20 in Madras, to 10 to 18 in the Central Provinces.[1] Now, in the census of the North-West Provinces,[2] the difficulty of obtaining true census returns in regard to the *number* even of the females is mentioned, and it is stated that there "seems to be uniform concealment of females between 10 and 13." So much the greater difficulty would there be in discovering what the relatives would be anxious to conceal—the existence of leprous females. This is borne out by the statement (p. 63) that the numbers given are not "a correct representation of the extent to which persons afflicted with these infirmities (insanity, leprosy, etc.) are to be found in the various localities in the province." Thus, such returns are only useful as showing a certain number who *are* affected, but must not be taken as showing the *whole* number. It is highly probable that such returns as those of Bombay and the Central Provinces are nearer the truth, while I think we must accept it as proved from the agreement of the censuses of all the provinces on the matter, that male lepers *are* more numerous than females—a fact speaking strongly in favour of the communicability of the disease, when the seclusion in which the greater number of the females of India live, *so that they are less exposed to contact with the sick than the males*, is taken into consideration.[3] Some allowance, however, must be made for female infanticide, especially of diseased children.[4]

Passing from India, we find leprosy in Persia,[5] Bokhara,[6] Eastern Arabia,[7] and Syria,[8] in the latter chiefly in the south and the mountains of Lebanon. It also exists among the eastern islands of the Mediterranean,[9] the Ionian Isles, Crete, Rhodes, etc.

I may also add that Sonnini mentions its existence in Turkey early in this century; but Thomas,[10] who quotes from him, does not decidedly say whether he meant European or Asiatic Turkey; he appears to mean European. We have no information in regard to it

[1] Census of Madras for 1872, Part II. p. 215. [2] Pp. 36 and 54.

[3] In connexion with the great number of lepers in India (which, however, it must be remembered, is *relatively* in its 190 millions, a smaller proportion than obtains elsewhere), I must here emphatically protest against the manner in which the disease is ignored by modern text-books and many teachers of medicine. The latest text-book in which I find it described is Good's "Study of Medicine"—a masterpiece of its kind, published in 1840. As a consequence, students who are to be spread over the whole world may go into practice *in total ignorance that leprosy even exists in modern times;* and the study of the disease is even neglected by medical men living in the tropics.

[4] See Macnamara in Coll. Phys. Rep., p. 45; and Census of North-West Provinces, p. 3.

[5] Coll. Phys. Rep., p. 71, and Hirsch, p. 313. [6] *Ibid.*, p. 313.

[7] Niebuhr, lib. cit.

[8] Wortabet, "Memoir on Leprosy in Syria," in *Brit. and Foreign Med. Chir. Rev.*, July 1873; and Coll. Phys. Rep., p. 54 *et seq.*

[9] *Ibid.*, p. 58, *et seq.*, and *Once a Week*, 1863, p. 143.

[10] "Modern Practice of Physic" (1813), p. 548.

of a later date in the interior of the country, though it does exist among Turks, Greeks, and Jews at Constantinople to a limited extent.[1] Virchow[2] mentions its existence in Moldavia; in Greece, where it began in Europe, it is still known in some parts.[3] It is worthy of notice that the diet of the people where it has lingered so long is a vegetable one, with salt fish—poor food, when compared with that of the English, among whom it has so long ago disappeared.

As a kind of appendix to the history, I must notice cases of so-called leprosy arising among natives of Europe who have never been abroad.

Hebra mentions[4] cases of "pigment lepra" or morphœa, in Europe, and confuses it with macular leprosy; and Erasmus Wilson[5] looks on morphœa "as a faint trace still existing among us of leprosy;" but with all due respect to such high authorities, sufficient grounds for such an opinion seem to be wanting. In the first place, as Dr Hilton Fagge points out,[6] nearly all the cases of morphœa are in females (20 out of 25 reported by E. Wilson), while in leprosy as many males are affected as females. Secondly, the anæsthesia is only secondary to the destruction of the skin and contained tissues, in morphœa; while the skin is intact there is hyperæsthesia, as I have myself observed; on the other hand, there may be, and generally is, complete analgesia in cases of leprosy, while the skin, with the exception of some scruffiness, is perfectly intact. I have bored pins and thorns into such skins, while the patient was looking straight and unconcernedly into my face. Thirdly, the patches of morphœa are rarely symmetrical, leprosy is almost always so. Besides, morphœa is unilateral, leprosy symmetrical; morphœa is a purely local disease, leprosy, as Wilson says, is a blood disease. Again, Wilson states (Lecture 8, May 1856), that mercurials do good in some cases "where a syphilitic affection had to be controlled," while all authors from Schillingus agree that mercurials are hurtful in leprosy. The character of the deposit also differs in the two diseases, being gelatinous in leprosy, lardaceous in morphœa; and, lastly, the eruptive form of leprosy appears and disappears; morphœa progresses continually from its first appearance.

Wilson's opinion, therefore, "that the pathognomonic characters of the disease (*i.e.* morphœa) are such as to point directly to elephantiasis as their source," appears to me to be without any foundation, and his admission quoted already as to the syphilitic affection of some of the cases, points out a much more likely general cause in this country. At the same time, I can understand that in countries where leprosy is endemic, its poison there,

[1] Coll. Phys. Rep, p 70.
[2] Lib. cit., p. 529.
[3] Coll. Phys. Rep., p. 68.
[4] Lib. cit., p. 156 of vol. iv.
[5] *Lancet*, 26th Ap. 1856.
[6] Guy's Hosp. Catalogue, p. 203. All the facsimiles are of females. A woman was shown at the Path. Soc. this year. (See Transactions, 1877.) Wilson himself notices this fact.

as that of syphilis in England, *may* act as an exciting cause of morphœa in some cases, and thus morphœa may come to be looked on as a first stage of leprosy.

Besides these cases of morphœa, however, a number of cases have from time to time been reported in the journals of so-called leprosy. Having carefully examined into all of these to which I could possibly obtain a reference, I can only say that, with the exception of one, Dr Rees' case, none of them can with the least confidence be pronounced cases of leprosy,[1] although some of them may come under the head of leproid, as described by Virchow,[2] having, as he says, analogies with leprosy, but differing from it in the absence of anæsthesia and of diseases of the mucous membranes. Some of these cases are really cases, however, of syphilis. I will try shortly to review all the cases I have referred to, noting as shortly as possible their points of difference from true leprosy. In Nourse's case,[3] not only was there no anæsthesia after *nine* years' illness, a period long after it is a distinctly marked symptom in leprosy, but there was actual tenderness of the *reddish* (not dusky) tubercles, which besides, even up to that period, came out in crops, instead of being permanent. I have seen just such a case in Scotland, in which, from some vaso-motor disturbance, large temporary tubercles formed on the eyebrows and cheeks, the patient being subject to severe pain in the stomach, but I never looked on it as a leprous case. In Erasmus Wilson's case,[4] the sudden invasion, emaciation, and contraction of the skin are all unlike leprosy, while the contraction of the fingers was only caused by the skin disease; there is no mention of real anæsthesia, only that the sensibility was somewhat deadened, as it must have been with the skin in the diseased state described, nor are any of the characteristic bullæ of leprosy, which appear on the fingers, mentioned. From the description, the case does not appear to me to be one of *bona fide* anæsthetic leprosy. I have now under treatment a case of pemphigus of the fingers of one hand, in which, while the pemphigus lasts, there is contraction, but that disappears whenever the bullæ have burst and healed up,—there is a deadened feeling in the finger now affected, but not analgesia. Such a case, while it resembles anæsthetic leprosy in some of its symptoms, and might be *supposed* to be caused by the poison of leprosy still remaining in this country, is to my mind simply a case of local disease, or at most a local manifestation of some slight temporary derangement of the system.

Broadbent's case,[5] in a young man from Stornoway, was unlike leprosy in so far as there was no anæsthesia or nodules on the *ears* after nine years' illness, and there *was* profuse sweating and emaci-

[1] Landré (p. 71) and Kaposi say the same of three such cases reported by Steudener.

[2] Lib. cit., p. 540.

[3] *Med. Times and Gazette*, 2d Sept. 1865.

[4] *Lancet*, 19th Jan. 1856.

[5] *Ed. Med. Jour.*, 1855, p. 434.

ation—very uncommon symptoms, to say the least of it, in the tubercular disease. The ulceration of the palate would point to syphilis. Had this been proved to be a true case, it might have been looked on as the last case in a remote district where it had not quite died out. Priestley[1] quotes two cases, but the first is evidently Dr Broadbent's case, although slightly erroneously quoted; the second is really a case of dry gangrene of one finger, which is mentioned elsewhere by Sir James Simpson[2] as a "curious amputation, which is sometimes a mark of elephantiasis."

Dr Gull's case[3] is so clearly from the history, symptoms, and post-mortem appearances, a case of syphilis, only resembling leprosy in the presence of tubercles, that I am astonished to find the facsimile of it in the museum of Guy's Hospital still marked as a case of leprosy. There was no anæsthesia, and the face is of a coppery colour, not the *dusky* colour shown in model 423, a typical case. A similar case is reported in the *British Medical Journal* of 24th January 1874 by Victor de Meric as a case of syphilis; and I have myself seen a man in St Kitts whose face was covered with tubercles, but in whom there was no analgesia, and whose illness was looked on by Dr Boon of that island, who had known him for many years, as undoubtedly the effects of syphilis. Mr Gaskoin's case,[4] headed "tubercular leprosy," has not a symptom indicating the presence of that disease, unless to one holding to the old confusion between it and elephantiasis Arabum, or Barbadoes leg.

Dr Rees' case[5] was undoubtedly one of true leprosy, and I am surprised that any question as to the fact should have ever been raised. Johanna Crawley, Irishwoman, aged 54, had lived thirty years in Stepney. In 1866 had lost part of the first finger of the right hand, and had her body and limbs stained with large brown patches; there was decided anæsthesia as far up as the elbows. The face was puffy, and the lips and ears swollen. In that state she left the hospital. Having made inquiries at Stepney, I have been informed by her daughter, a woman of 25 years of age, that Johanna died in 1874, after losing a part of *all* her fingers and toes, the blisters and destruction of bone causing great pain. She died of inflammation. A Dr Hayden saw her in her last illness, and at once recognised the case as one of leprosy. It may be worth recording, that her daughter's name is Mrs Suckling, 8 Salmon's Street, Salmon's Lane, in case *she* should in time be attacked, as she attended her mother throughout her illness.

[1] *Med. Times and Gazette*, Jan. 1860.
[2] *Ed. Med. Jour.*, Jan. 1855.
[3] Guy's Hosp. Reports, 1859, and Catalogue, p. 210, model 435.
[4] *Br. Med. Jour.*, 6th Dec. 1873, p. 655; I fully criticised the case in the Journal of 3d January 1874, p. 36.
[5] Guy's Hosp. Reports, 1868, p. 190, and Catalogue, p. 214, models 446 and 447. A similar case is said to have occurred under Dr Gale some years before, but no reference is given to it. (See *Lancet*, 5th Jan. 1867, p. 17.)

Although undoubtedly indigenous, I cannot look on this case as necessarily an antochthonous one, although, from the information given me by Mrs Suckling, I could not trace any actual source of infection, as her mother never, that she remembers, kept lodgers, and her father had never been abroad,—but Johanna was a sail-maker, working in a factory with many others, and, living in a district crowded with people *in constant communication* with the East and West Indies,[1] and in which there are many coloured people; so that, even before her daughter was born, or while the latter was a mere child, she might have lived in contact with some leper, and the circumstance have entirely passed out of memory before the first appearance of the disease in 1865, so long is the period of incubation. I look on the case as strictly analogous, though not demonstrably so, to cases of yellow fever occurring at parts in direct communication with the West Indies among subjects who have never been abroad, but who are infected from those arriving sick. This is much more likely than that such a case would arise of itself.

Having thus, in what has preceded, completed the history of leprosy, I will now consider the etiology of the disease by itself, in considering which our knowledge of its history will greatly assist us.

I know of no question in regard to which more rash opinions have been ventured than the one I am now entering on, or in which opinions have been more utterly opposed to one another. The great error I have observed to pervade all such opinions, is that they have almost invariably been founded on local observation only, each author emitting an opinion as to the causation of the disease mostly in accordance with the conditions of the locality in which he happened to observe it. This remark certainly does not apply to the conclusions come to by the Royal College of Physicians, who had quite an "embarras de richesse" to choose from in the shape of confident opinions coming from all parts of the world, this especially applying to the non-contagious nature of the disease; but unfortunately, to any one acquainted with the manner in which such questions were answered, such returns for the most part are more worthless than the paper they are written on. It is to be particularly remarked that in countries where the most leprosy prevails, as Demerara, medical men are most convinced of its contagious nature;[2] where there is least (comparatively), the

[1] Her house is just beside the Limehouse basin, not quite half a mile from the West India Docks, and a corresponding distance from the East India Docks.

[2] Drs Manget and five others—*all* those whose reports the Coll. of Phys. publish (p. 45)—consider that it *is* contagious, and speak of such cases known to them. It is therefore surprising to find Dr Milroy quoting (p. 10) six other gentlemen, "some of the most experienced men in the colony," and *omitting all mention of the contagionists* except Dr Manget, whose cases, he simply says, are meagre in their details.

The chief argument of the non-contagionists is that they have not seen the

opposite is the case, as in India, where *the disease is not studied;*[1] and a medical man may spend years without having more than a passing glance at a few cases, *unless he searches them out.* The

disease produced during cohabitation for years, yet he takes no further notice of Dr Manget's first case,—an Englishman who became a leper after living with a leprous coloured woman, who had a child by him. Yet at p. 29 Dr Milroy boldly quotes the "*expressed opinion*" *of the medical men of Demerara against* contagion, and goes on to say, "My own personal observation and inquiries have all tended to the same general result, namely, that the spreading of leprosy is not due to contagion;" but at p. 41, he admits "leprosy appears to me neither more nor less contagious than scrofula, and what Dr Williams says of consumption *is equally applicable* to the other cachexy," viz, "both reason and experience indicate that such a noxious influence may pass from a patient in advanced consumption to a healthy person in close communication, and *may produce the same disease.*" I can respect a change of opinion in such an author as Erasmus Wilson, who in 1856 (*Lancet*, March 1st) wrote, "the doctrine of infection and contagion has long been abandoned," while in 1873 (*Lancet*, February 15) he strongly upholds the contagion theory, thinking "it may even be conveyed by exhalations given off by the leprous." Such a change was doubtless the result of *long* research, but for the vacillating indecision of Dr Milroy's self contradiction *in the same Report* there is no such reason.

[1] This is stated in regard to Demerara by Manget (see Rep., p. 48); as to India by Carter (p. 117), who repeats the remark in his later reports; and in the Mauritius by Regnaud (p. 83); and again, at p. 241, Erasmus Wilson speaks of a *medical officer* of the Indian army, himself a leper, who, *along with other medical men in India*, did not recognise the nature of his own case, but looked on it as one of syphilis, from which indeed he had suffered previously. This gentleman *had never seen a case of leprosy*, from which Wilson hastily and erroneously concludes "that the disease is not so widely distributed in India as we have been accustomed to believe,"—an idea disproved by the census of India, which shows its universal existence throughout the country, although in the North-West Provinces (in which this gentleman seems to have resided) there is less (next to Mysore) leprosy than in any other presidency, the census returns giving only 1 in 3046 (1 in 2500 being probably about the truth), so that unless a man searched it out he might easily spend years without seeing cases, though not one district is free from it. *Yet there is no part of India from which more positive replies, founded on negative evidence alone, are given as to the disease being absolutely non-contagious.* Only three reporters out of forty-two think it *is* contagious. One gentleman in Jansi candidly says he "only met with one or two isolated cases during a residence of eight years, and his attention was not called to it till the receipt of these questions" (Coll. Phys. Rep., p. 151). (It is to be remarked, however, that in a remote mountainous corner of these very North-West Provinces, in one division—Kumaon—there are more lepers to population than in any other division in India.) I specially notice that where the census returns show *fewest* lepers, there the conclusions against contagion are strongest. Thus from Banda, a district with a very high proportion (1 in 717), there is an uncertain report; from Budaon (1 in 2174) a positive opinion in favour of contagion; in Benares (1 in 2777) the opinions are against it; while in Etawah (1 in 12,500) the decision of expression is worthy of note, "I have never known the disease to be contagious either by proximity or cohabitation." No doubt, as Lewis and Cunningham point out, the *hospital* experience at Almorah, Kumaon, is against contagion, and they contradict (p. 58) two cases reported in the Coll. Phys. Rep. (p. 141) of attendants said to have become affected; but I beg to submit at once that a hospital is the worst place, with its *absolute cleanliness* both of persons and things, in which to draw any conclusions in regard to the non-contagion of the disease, which I have already said appears to require long-continued contact or inoculation and a

same is the case in the West Indies, where, to my own knowledge, men may and do pass years of their lives without even seeing or knowing or caring more of lepers or leprosy than to pass them on the road, yet it is on the negative evidence of men so situated that the conclusions of the College of Physicians were drawn up. For instance, from Grenada, *where the disease is very rare*, there is one negative report from Dr M'Intyre, who says, "I have met with no such instances" [1] (of contagion); and this and such like reports are allowed to overweigh as evidence such positive observations as are recorded on the same page by Dr Aquart, simply, it appears to me, because the negative witnesses were greater in number than the positive; the utter worthlessness of such negative evidence appearing more strongly when it is remembered that Grenada has but few lepers on which to make observations—only, it is stated, about *half-a-dozen in all.*[2]

The same tendency to accept and even extol evidence without sifting its true value (which, it must be said for the College of Physicians, it was hardly in the nature of things possible for them to do), is seen in Dr Milroy's report, where (p. 16) he gives extracts from an "able report on leprosy by Dr S. H. Harris," in which the latter gives *decided opinions* as to the causation of the disease, the said opinions being, in fact, a mere repetition of some of the most undecided and least valuable ideas extant on the subject. Dr Harris says, first, that the influence of climate is "the most potent cause;" secondly, diet; and thirdly, habits of the people. As regards climate, he says *malarial poisoning* is the *primary* cause, and says, "it may briefly be remarked that their dwellings" (of the subjects of the disease) "are generally situated in the vicinity of marshes and of low elevation, or in some *well-known malarial part.*" He also speaks of their filth, and their bodies "being only partially clothed." Dr Harris is quite as positive as to its being non contagious; his words are, "*I am of opinion* that further experience will teach us that it is not communicated by contagion if we confine ourselves to the strict definition of the term."

Reading such an "able report," and such positive opinions, one naturally inquires in what extensive field of observation, or over what period of time, did Dr Harris's observations extend. He *had only been appointed to Montserrat a very few months when the Report was written.*[3] The island contains about 8000 inhabitants; in the lazaretto, near Plymouth, are six or eight lepers, while as many more, so far as I could ascertain, were, when I was there (although I had no chance of making such extended inquiries as I did in St Kitts, such inquiries requiring actual residence), scattered over the

subject prepared for its attacks. That hospital dressers *do* sometimes contract the disease is certain, however, as in three cases reported by Hillebrand and Rose (Macnamara, pp. 22 and 57).

[1] Coll. Phys. Rep., p. 36. [2] Vacc. Rep., Bakewell's Evidence.

[3] Before being appointed to Montserrat, Dr Harris was in Cunard New York Liners and Liverpool Hospital.

island. Now, so far as Dr Milroy's extracts show, Dr Harris never inquired even into the number, much less the intimate individual history, of the cases either at the lazaretto (which, I believe, was under Dr Johnson's care) or elsewhere, such inquiries, which take both time and trouble, being absolutely necessary as a foundation for the formation of any opinions on the subject. Again, as to malaria being the primary cause, it is, to say the least, strange that such an opinion should have been adopted in an island having quite its fair share of the disease, *in which there is not enough level ground to form a croquet green*, the name Montserrat plainly expressing its topographical features. As to the value of Dr Harris's *opinion*, formed on such observations (!), I think I need say nothing. His remarks about clothing also do not apply.

If I have appeared to say too much in regard to this gentleman's "observations and opinions," it is because I look on such as a fair specimen of the "authentic evidence," as Dr Milroy calls it,[1] as supplied to the Royal College of Physicians and himself, leading them and him to conclusions as to the non-contagious character of the disease, and the non-necessity for segregation of those attacked by it, which I have not the slightest doubt will have, from their being acted on by the Imperial, Indian, and Colonial Governments, the most disastrous effects in the future, and tend greatly to the continuance of the disease, if not to its actual increase

It is to be hoped that such real evidence as has been advanced by Carter[2] as to the value of segregation, may come to be acted on, and the building of proper asylums or villages for the diseased may be encouraged, or at least not discouraged, by Government, as it has been since the College of Physicians published their Report.

The various causes which have been advanced as sufficient to produce or increase leprosy may be classed under Climate, Poverty and Malhygiène, Heredity and Contagion. The idea that certain races are more obnoxious to it than others has also been advanced.

Climate may be considered under Tropical Climates and Malaria. Residence near the sea and great rivers is another point which might be considered under this head, but may be more conveniently spoken of when treating of Food, such residence being chiefly

[1] Rep., p. 65. I may appear unfair to the non-contagionists, and, so far as those who *have* made *bona-fide* observations, would regret exceedingly being so, but I must remark that the simple fact that a man *can* point to cases of contagion, shows that he has paid some attention to the disease; when he cannot, it proves nothing, unless he has had years of observation and numerous cases, and even then he can only fairly say that it is not easily communicated.

[2] Rep. on Lep. in Norway, pp. 23 and 24, and Report of 1876, p. 20, where he points out a fact lately elicited in regard to the people of India, "that relatives of all degrees live together in little communities," and almost admits that it is contagious,—saying, however, "we should act as if the leprous plague were really communicable."

supposed to have some influence in causing leprosy, because fish is generally the chief diet in such situations.

Tropical Climates.—Some authors, as Hobson, who observed that leprosy only existed in South and not in North China, and Thomas, have looked on true leprosy as a disease which could only exist in hot climates, an idea that the whole of the history at once contradicts. At the same time, the history as clearly shows *that it has never arisen spontaneously, and attained its full development to the typical form of the disease, except in the tropics,* unless when conveyed out of them by contagion.[1]

This, and the decidedly beneficial effect produced on the disease in those who have contracted it in the tropics, amounting, in some cases, to an entire arrest of the disease, when they came to reside in a cold climate, would tend to show that, with other depressing influences, that of a tropical climate may be an adjuvant factor in the primary production of the disease.

Malaria.[2]—That malaria has no necessary connection with leprosy, although at first sight it would appear so from its great prevalence in such a place as Demerara, is shown by its prevalence in other places where no such influence exists. In St Kitts, on the windward, coolest, hilly side, there was in 1871 much more leprosy (1 in 306) than on the leeward, hottest, and lowest side, where the proportion was 1 in 452. This same proportion obtained in 1817. My own, the No. 1 district, once somewhat malarious (the only one that ever was so), now, at least in the town, well drained, but still exposed to slight malarious influences occasion-

[1] The Ngerengere of New Zealand is, at first sight, an apparent exception to this, but it must be remembered that that is not a *typical* form of leprosy, and it is far from unlikely that the differences I have already pointed out are the result of the climate of New Zealand not being a tropical one,—this adjuvant factor in the production of the disease thus being awanting, a want not sufficient to change the type of the disease elsewhere, when conveyed by contagion, but sufficient when the disease is, so to speak, struggling into existence, to do so. Again, the New Zealanders *may* have brought it with them from Samoa or Fiji, from which they appear to have immigrated to NewZealand.

[2] Liveing (p.67) says erroneously that leprosy is rare in dry localities in India, and points to its existence on the moist banks of the Nile and low-lying levels of South China as proof that malaria has a causal relation to the disease. E. Wilson also speaks of its "origin" on the "marshy" banks of the Nile, with the same view (*Lancet*, Mar. 1, 1856). Unfortunately for the theory of these authors, it has one fault—being opposed to facts. So far as Egypt is concerned, it has the driest climate in the world, and Larrey (*lib. cit.*, p. 243) distinctly states that leprosy is *not* seen on the coasts (where round the bitter lakes there may be some malaria), but is common (*règne*) in *dry* and arid places near the *deserts* (*not* near the Nile) in Upper Egypt. Liveing's statement in regard to India is contradicted by facts soon to be stated; and in regard to China, while the disease is almost unknown to the north of the Yang-tse-Kiang, the Hoang-ho, north of it, flows through a flat country *below* its own level, and parts of the banks of the Yellow Sea are highly malarious. The malarious theory has also been adopted without sufficient inquiry by others, as St Vel ("Maladies Intertropicales," p. 478), and Cazenave and Schedel (*lib. cit.*, p. 356), Holmsen of Norway (Landre, p. 75), etc.

ally, had fewer lepers (1 in 503) than any other district in the island. In Africa it is seen on the malarious coast at Sierra Leone, *but chiefly among natives of the interior*. It is unknown in Dahomey, a malarious place, and on the upper tributaries of the Amazon, which, as Bates mentions (p. 95), are highly malarious. It is common on the coast of Bergen, in Norway, where there is no malaria. In India, as already mentioned in a footnote, it is most common in a mountainous division of the North-West Provinces, and is known to be common in Nepaul; and another focus is in the district of Beerbhoom in Bengal, with 2872 lepers in 695,921 of a population,[1] or 1 in 243. This is a dry undulating district, with a semi-civilized population. Burdwan *District*, in the same Division as Beerbhoom (the Burdwan Division), has 1 leper in 442, it being partly malarious; while Hoogly, "a hotbed of malarious pestilence,"[2] another district of the same division, has only 1 in 1444.

In what I have already said in the history, it is easily seen that the disease has spread in all kinds of places, and in what I have to say on sea-coasts, further proofs will be adduced that malaria and leprosy have no necessary connexion. I may here mention its prevalence in the inland towns of Arabia, where there is no malaria, and that it is known only in the elevated dry districts of Persia, and unknown in the marshy swamps near the Caspian.[3] Finally, there is no malaria in the Sandwich Islands, where it has spread so rapidly.

Food.—The use of fish, and, with this, residence on sea-coasts and the banks of great rivers, have been put forward by many as causing leprosy.[4] That the opinion is erroneous the following facts show:—In Norway, leprosy certainly exists mostly along the coast-line (leading authors in that country to the opinion), but even there in varying intensities, being, for instance, unknown in the town of Bergen among the people of that place,[5] and very severe in the north of the province of that name, while there is little of it in the south of the next province to the north of Bergen. On the northern and southern coasts of Norway it is unknown. It is unknown among the pure fishermen in the islands of Norway.[6] It is unknown in Newfoundland, a great fishing country; indeed, the history has shown us that it was unknown throughout America before it was introduced, though on the west coast the natives were chiefly fish-fed. Menol, on the ground that in Madeira, on the northern side, there are only 15 lepers in 20,000 people,—that

[1] Census of Bengal, p. xcvii.
[2] *Ibid.*, p. 91.
[3] Coll. Phys. Rep., p. 71.
[4] Danielssen and Boeck, p. 138; Virchow, p. 507; Wortabet, *op. cit.*; T. Fox, Ed. Med. Jour., March 1866, p. 802; H. H. Wilson, *op. cit.* (quoting from Hindu authors); Peacock (Lisbon), *Lancet*, 1870, p. 770; Hutchinson, Atlas of Skin Diseases, Catalogue New Syd. Soc., p. 92; Coll. Phys. Rep., *passim*, etc.
[5] Carter, Rep. on Lep. in Norway, p. 10 and map, and Danielssen and Boeck, p. 372.
[6] Carter, Rep. on Norway, p. 10.

side being about 1000 feet above the sea, while in the southern side there are 73 in 25,000,—concludes that it never spreads far from the coast. That his conclusions are totally without foundation is at once apparent, on comparing these statistics with those I collected in St Kitts, where I have already shown that the conditions are exactly the reverse of those obtaining in Madeira, the northern, windward side, high above the sea-level and with few fishermen (16 in 9475, or 1 to 591 of population), having fifty per cent. more leprosy than the southern side, where fishing is constantly carried on, and where the census returns show 143 fishermen in 18,524, or 1 in 130 of population.

The prevalence of leprosy on the high table-lands of Central Asia already mentioned; in Bokhara and Samarkand; in the mountains of Samen in Abyssinia; in the mountains of Lebanon, and *not* on the coast of Syria;[1] in arid Cephalonia;[2] in Sicily, where the proportion in the interior, from a late inquiry, appears as 5 lepers to 9000, while on the coast there are only 2 in 9000 of population;[3] in the mountains near San Remo in Italy;[4] in Madagascar, 7000 feet above the sea-level;[5] in the interior of Africa, 100 miles from the Niger, the nearest large river;[6] on the table-land of Mexico;[7] while in Brazil it is seen *chiefly* in the *interior* provinces of Minas Geraes and Matteo Grosso;[8] in the Rio de la Plata States, chiefly in Parana and Uruguay, inland provinces;[9] in Bogota and Soccoro on the Andes,[10] and in Quito,[11] while it is unknown in Peru and Chili; all these facts show that the opinion as to the prevalence of leprosy being in any measure dependent on proximity to the sea or large rivers, *except in so far as these are great pathways of human intercourse*, is utterly erroneous.[12]

[1] Wortabet, *op. cit.* [2] Coll. Phys. Rep., p. 67.

[3] Profeta, Sulla lepra in Sicilia, 1875, quoted in Lewis and Cunningham's Report on Leprosy in India, 1877, p. 24.

[4] Report on Leprosy in North Italy, &c., 1876, Carter. It still lingers in San Remo and Varazze. [5] Coll. Phys. Rep., p. 220.

[6] Caillé, *lib. cit.*, p. 402, and map.

[7] Simpson (quoting Cheyne) *op. cit.*, p. 402.

[8] Hirsch, p. 321. [9] *Ibid.*

[10] *Ibid.*, p. 325. Humboldt (Travels and Researches, p. 325) notices that Santa Fe de Bogota is 8727 feet above the sea-level, higher than Mount St Bernard.

[11] I would here for a moment revert to a part of the subject already considered, to point out how much confined the disease has been to the eastern side of the Andes, where the aboriginal natives were not fitted for work, and it was necessary to import negroes; whereas in the western coasts, as any reader of Prescott knows, the aboriginal Peruvians and Chilians were, and are still, the labourers of the country, and negroes were not required.

[12] Just as highways might be said to be a cause of its prevalence, because it is sometimes noticed to affect severely a whole string of villages intervening between two larger towns (Carter's Report, 1876, p. 19). He says also, "A similar line may follow the banks of a main river." In China it is as common in the interior as on the coast (Coll. Phys. Rep., p. 78); and is necessarily so on the banks of the great rivers, *where nearly all the population is crowded*, the rest of the country being, in comparison, deserted. (See "All Round the World," p. 133.)

More proof, if more is wanting, is shown in the late census returns of India, to the same effect. No doubt, as I already stated was my belief, those returns greatly understated the true number, and this is confirmed to even a greater extent than I supposed by Dr Carter, in his last (1876) report already quoted, which I received a few days ago, in which,[1] quoting Major Watson's scrutiny of the numbers in four divisions or counties in Kattiawar, Gujerat, with 220,000 inhabitants, he states that not one-half appear in the census returns; yet, as the same difficulties were to be met with in the ignorance and suspicion of the natives all over India, for purposes of comparison of the numbers in one part with those in another, the census may be taken as correct enough to show where it is most prevalent. I have already spoken of its prevalence in Kumaon;[2] it is also common in Bangalore, 3000 feet above the sea-level.[3]

In Bengal, Orissa and Chittagong, both sea-coast divisions, have only about 1 leper in 3400, and Rashaye, an inland division, 1 in 1453; one of the districts in it near the Himalayas, Rungpore, having 1 per 1000.

Turning to Bombay, we find that the census returns show the less the seaboard and the fewer great rivers in each division, the *more* leprosy there is. This is contrary to the hastily-drawn conclusions of Liveing,[4] and to a certain extent to the more cautious estimates of Dr Carter, who thought there was more leprosy in the Konkan than the Deccan. The Deccan, with no sea-coast, and elevated, has 12 lepers in 10,000,[5] the Konkan 8 to 10,000, it having sea-coast on one side; in Gujerat, a peninsula, 5 in 1000; and Scinde, a country traversed by the Indus with its numerous large tributaries and mouths, has only 1 in 10,000.[6] No doubt, in some instances there may be, as Carter thinks, within certain districts, as Kattiawar, Gujerat, more leprosy near the sea-coast, though his figures by no means show that there is, they not being relative, but absolute. He says, "Limiting the coast district to five miles from the sea, I find the disease commonest towards the sea,

[1] P. 18.

[2] Lewis and Cunningham attempt to explain its prevalence in Kumaon by its proximity to Nepaul, because the greater prevalence of leprosy is seen in the *eastern* side of the division bordering Nepaul, forgetting, or not noticing, first, that in Gurvhal, the western division of the province of Kumaon, it is *more* prevalent than in Kumaon proper; in Dehra Doon, on the south-*west*, it is as prevalent, there being 19 in 10,000; while in Bignoor and Bareilly, to the south of Kumaon, the latter partly bordering Nepaul, there are only 5 in 10,000; secondly, in the districts *bordering Nepaul to the south* there is less leprosy than in almost any other part of India: thus, Barrackpore has only 23 in 100,000; Bustee, only 9 in 100,000, or 1 to 21 of the number in Kumaon (see census of N.-W. Provinces, p. 9).

[3] Coll. Phys. Rep., p. 188.

[4] *Lib. cit.*, p. 66.

[5] Census of Bombay, part 11, table vi. p. 7.

[6] Unfortunately, white leprosy, kod or simple leucoderma, was confused with "black" or true leprosy, except in four districts, two of these being in Scinde, one in Gujerat, and one in the Deccan (Census, p. 215); however, if these districts are examples of the others, what I have said above is only more fully borne out.

namely, in the proportion of 100 to 71 inland."[1] But then he admits that the *normal* populations *may bear the same ratio*. Even if he is right, his explanation, that it appears to have been introduced by sea and is spreading inwards, would fully account for the numbers being greatest near the coast, while the fact that the people are "pure agriculturists," fish being little consumed,[2] shows that the greater prevalence near the coast has nothing to do with a fish diet.

Evidently, then, proximity to the sea or great rivers, or a fish diet, are not factors in the production of leprosy.

Note.—In a former part (p. 42) I stated on authorities then before me that leprosy was, in British Burmah, most prevalent among the Chinese and Bengalese. That it is common among the population as a mass, however, is seen from the report on the census of British Burmah, recently published, and quoted by Drs Lewis and Cunningham in their Report (p. 9), which I received while writing the preceding pages, and from which it appears that there are 11·6 lepers per 1000, or fully double the number shown in the Bengal census.

Exposure.—The idea that exposure, can at least favour the production of leprosy, has been spoken of by Daniellssen and Boeck,[3] but there seems to be no proof sufficient to support it. It may be admitted that when once the disease is established, exposure may, and probably does, increase the mischief in a disease in which weakness of the circulation is a decided symptom; but, from its great prevalence in the most beautiful climates, as that of Rhodes and Santarem, and its rapid spread in the Sandwich Islands, with other examples which might be quoted, as well as from its absence from the most rigorous climates, it is clear that exposure is not a necessary factor in its production, although in some cases it may assist in aggravating its effects.

Salt Food.—This has been supposed to at least assist in the causation of leprosy by many authors, as Schillingius,[4] Larrey,[5] Wortabet,[6] H. H. Wilson, speaking of it among the Hindoos, and quoting their opinions,[7] and Cazenave and Schedel;[8] Daniellssen and Boeck speak as if they considered the use of either fresh or very salt fish assisted in causing it.[9] In the Coll. of Phys. Rep.,[10] and Dr Milroy's,[11] a number of opinions to this effect are expressed, and Peacock thinks so from observations in Portugal.[12]

By those writers it seems to have been left out of consideration

[1] Carter's Rep., 1876, p. 18. [2] *Ibid.* [3] P. 257.
[4] *Lib. cit.*, p. 21. [5] *Lib. cit.*, p. 221. [6] *Op. cit.*, p. 190.
[7] *Lib. cit.*, p. 3. [8] *Lib. cit.*, p. 356. [9] *Lib. cit.*, p. 342.
[10] *Vide*, pp. xxxiii., lxvii., 171, 201, 219. At p. 201 acids are mentioned as a cause, an idea derived from Hindoo writers.
[11] Pp. 9 and 16 in Dr Harris's "able report."
[12] *Lancet*, 1870, vol. ii p. 776.

that salt food is extensively used in many parts of the world where leprosy is unknown, as Newfoundland, Canada, the west coast of South America, and the greater part of the Mediterranean coasts, while in the Sandwich Islands, long before leprosy was known, salted pork was constantly eaten.[1] Again, in China most leprosy is seen in the south and in Shanghai, yet in the neighbourhood of the latter the people use very little salt;[2] while in the north, where leprosy is unknown, the enormous salt mounds in the neighbourhood of Tientsin, described by every traveller, show that there is no want of it there. In Portugal, religious orders using salt food exclusively have no leprosy among them.[3] In the Faroe Isles, where leprosy was common, fish was eaten "without any salt,"[4] and in India and Africa, the ancient foci of leprosy, salt is *a luxury* unknown to millions.

These writers also seem to have forgotten that salted food is seldom eaten as such, a taste such as that of the Sandwich Island chiefs being unusual, but is generally soaked before being cooked, unless when it is simply used as a relish with a large quantity of vegetable diet, and even in that case the blacks eat it with vinegar to take away the taste. In Norway, again, salt and semiputrid fish is eaten to as great an extent on parts of the coast where leprosy is rare or unknown as in those where it is common.[5]

It is clear then that salt food, as such, has no influence in either producing or increasing the disease, although, as being poorer food than fresh, the best part being lost in the brine,[6] and what remains, being rendered less digestible, its use may assist in lowering the general health of those using it, and so probably increase their susceptibility to the disease

Poor Food and Deficient Food.—From the time of Galen[7] and Aretæus,[8] a poor diet has been looked on by many as a cause of leprosy.[9] Others, in contradiction, have pointed to its existence among all classes, from kings downwards, in the Middle Ages,[10] and among all classes alike in Madagascar.[11] In India, rich Europeans are affected, as well as natives of the very highest classes.[12] So far, how-

[1] King, in Cook's Voy., vol. viii. pp. 119 and 128. Boils and ulcers common among them are attributed to the great quantity of salt consumed (vol. vii. p 113). [2] Coll. Phys. Rep., p. 78.

[3] Landré, p. 34. [4] See quot. from Debes in Liveing, p. 29.

[5] Carter, Rep. on Lep. in Norway, p. 11.

[6] See Blyth, Dict. of Pub. Hygiene, p. 363. One-fourth of the fibrine and ix-sevenths of the albumen is lost.

[7] De Arte. Cur., lib. ii. c. x. De Cancris. [8] *Lib. cit.*, p. 179.

[9] See Schillingius, § xxxi. Wortabet, *op. cit.*, p. 189. Virchow, *lib. cit.*, pp. 507 to 509. Nourse, *op. cit.*, and Coll. Phys. Rep., pp. 3 (New Brunswick), 64 (Crete), 72 (Persia), 93 (Ceylon), 134, 137, 144, 160, 171, 195 (India), etc., etc.

[10] Robert the Bruce of Scotland, and Baldwin IV., King of Jerusalem, died of it (Simpson, *op. cit.*, p. 394). [11] Coll. Phys. Rep., p. 221.

[12] Macnamara, p. 38 (Rajah of Parsee); Coll. of Phys. Rep., p. 158 (Rajah of Jansi).

ever, as such cases as King Robert the Bruce is concerned, it must be remembered that the food eaten by kings and courtiers of the Middle Ages was in many respects inferior to what is now used by the middle classes. Thus, Philip de Blois says, "I have sometimes seen wine so full of dregs put before noblemen that they were compelled to filter rather than drink it, with their eyes shut and their teeth closed, with loathing and retching—meat, whether sweet or not, is sold alike. The fish is four days old, yet its stinking does not lessen its price—indeed the tables (sometimes) are filled with carrion." Its existence among rajahs and well-to-do Europeans and others, contagion will account for; while it must be remembered that even the best-fed people within the tropics are more or less anæmic and debilitated. It may be true, as Carter says in his "Report on Leprosy in Norway" (p. 11), that leprosy attacks the robust as well as the weak and ailing; but there are no statistics available to show that they are attacked in as great a proportion, while, as I have already shown, the history of the disease shows that it has always declined where the food of the people and their general hygienic conditions have improved,[1] and has never spread among a flesh-fed population.[2] In India the condition of great masses of the people is, and for ages has been, that of chronic starvation, of which the late and present (1877) famines are only aggravations. In the Census Reports for the N.-W. Provinces, it is stated,[3] "hunger is a prominent symptom, and chronic starvation, both from a deficiency in the quantity of food, and in almost an entire absence of one or more of the ingredients essential to health, is by no means of uncommon occurrence." The people are chiefly fed on "behjur," a mixture of several kinds of grain, and the ordinary quantity consumed by the agricultural labourers *does not contain one-half* the nutriment that is just sufficient to preserve health, as supplied in the food given to

[1] A special instance of this is seen in the Faroe Isles, where, with great improvements in agriculture and diet, leprosy has disappeared.

[2] At first sight the prevalence of leprosy at Santarem might appear a local contradiction of this, as Bates specially says that flesh meat is cheap. It by no means follows, however, that it is used by all classes; it is much more likely that it *is* cheap, partly because the demand is confined to the better classes, while the great quantity of salt fish (vol. ii. p. 10) shows what, with vegetables, is the real staple food of the population as a mass. But Livingstone, in his Last Journals, vol. ii. p. 40, speaks of the existence of "partial" leprosy among the Manyuema, who seem to live in a great measure on "durra," the meal of millet, but who also have a plentiful supply of flesh food (p. 148). It by no means follows, however, that *all* the population enjoy its *use*, such food being often reserved for the chiefs and males. If further information should show that all do obtain it, we would then know that even a flesh diet is not an absolute protection of a population *within the tropics;* it being always remembered, however, that sedentary tropical races and tribes do not, as a rule, use as much flesh meat, even when obtainable, as natives of the temperate zone.

[3] Pp. lxiv. and lxxiii.

prisoners in the jails. In China matters are little better.[1] I wish it distinctly understood, however, that I do not in the least believe that any amount of starvation can *produce* leprosy. Its absence in North China, where, in Tien-tsin, death by starvation is by no means uncommon,[2] people living actually on the offal thrown from the ships; and in Tierra del Fuego, where the wretched inhabitants are often reduced by starvation to the horrible necessity of roasting and eating their old women,[3] as also from among the aborigines of Australia, next the Fuegeans, the most degraded of human beings, and from the entire western side of the Andes, where the food was poor and insufficient,[4] or among poor tribes of North American Indians, exposed to the most fearful famines, so that they would eat one another, even their own families,[5]—such examples of its absence as these show that something more than simple poverty of diet is required to produce the disease; at the same time, as I have already indicated, I have little or no doubt that, where such conditions exist, a population is more prepared for and more likely to suffer from leprosy when it is once introduced.

Poverty and Malhygiene.—Much that has been said in regard to poor diet also applies to this part of the subject. The history has shown that wherever a decided improvement has taken place in the general condition of the people, leprosy, even when prevalent formerly, has decreased. Carter, indeed, our leading author on the pathology of leprosy, expressed a contrary opinion early in 1873, then thinking that faults of diet, hygiene, or habit equally fail to account for its varied distribution;[6] but he seems to have hardly taken the history into consideration, and the example he quotes of the disease being "nearly as frequent"—(in reality, as I have already stated, 50 per cent. more so), in the elevated Deccan Plain, Bombay, with better food than in the Koncan—proves nothing, and may be ex-

[1] Coll. Phys. Rep., 78. Durand Fardel (La Lêpre en Chine, *Gazette Medicale de Paris*, 18 Août 1877) mentions that rice is the basis of the food of the mass of the population, salt fish and the like being only used as a relish. Some cannot even obtain rice, and use potatoes. The rice is always insufficient in quantity. He also states that the province of Canton is the chief seat of leprosy in China, yet is, on the whole, the best fed. If this is true, it increases the probability of what I have already said,—that leprosy has been at least increased in that province by its constant commerce with India, and the immense number of Mahomedans and others who resided there 1000 years ago.

[2] *Vide* "All Round the World," p. 149. Tien-tsin means "heavenly felicity" (!) the misery in it is a horrible caricature on the name.

[3] Darwin, "Jour. of Voy. Round the World," 1845, vol. ii. p. 214. They prefer eating the old women to eating their dogs, because the latter can catch fish, while the old women are useless; I ought to have quoted the Fuegeans as fish-eaters solely who have no leprosy among them. The Californians also used almost nothing but fish (Beechy, vol. ii. p. 74).

[4] Cartwright, in *Br. Med. Jour.*, 18th Dec. 1875, Lecture on the Scope of Dental Surgery. [5] Kane, in *Household Words*, 26th March 1859.

[6] "Observations (1869-71) communicated on Pathology of Leprosy." *Lancet*, Ap. 1873, and Rep. 1874, p. 20.

plained by his own observations, already quoted in regard to Kattiawar, unless it could be shown that improvement in hygiene *within these districts themselves* had not been followed by a decrease in the number of lepers; while, that such a state of matters is purely local and not to be taken as the foundation for any argument, is seen by a comparison with Syria, where leprosy is unknown in Aleppo and Beyrout, and other sea-coast towns, where people are comparatively well housed and fed, and cleanly, while it is common in the villages of the interior where they are dirty and live in poverty.[1] My own belief is that, so far as poverty is a depressant, leading to an anæmic state of the blood, and so far as malhygiene consists in or leads to overcrowding, so forcing the sick and healthy into *intimate contact*, as in the West Indies, where seven or eight people often sleep in a hut ten feet square, so far they tend to the increase of leprosy.

But *filth* has often been put forward as a cause of the disease. I agree with Carter, to a certain extent, however, that it has *per se* no influence, at the same time its non-removal, like overcrowding, may assist in the propagation of the disease by increasing the chances of inoculation, the contagious matter being allowed to remain in contact with the healthy skin, while with cleanliness it would be washed away.[2] I have no doubt that the condition of matters in England depicted in the sonnet, "The King and the Miller of Mansfield," in which the miller, thinking he is speaking to a royal page, asks—"Hast thou no lousen in thy shoon?" or in which the gentlemen of the Earl of Northumberland (of 1512) were allowed 2s. yearly to pay for washing their body linen, while those slightly under them appear to have had no body linen to wash,[3] would assist in the propagation of leprosy in the manner I have indicated. So in the present day the filthy state in which many of the Negroes and Portuguese in the West Indies live, almost equal in some cases to that of the old Manganja, who told Dr Livingstone that he *had* washed *once* in his life;[4] and of the Portuguese in the Brazils who, even respectable families, look on an Englishman's morning wash as an extraordinary and somewhat insane proceeding, would at least prevent the removal of the contagious matter when brought into contact with the skin, and is probably, along with the indifference in regard to coming in contact with the sick among these people, one cause for its excessive prevalence. The same may be said of the Chinese, and more emphatically of the lower class Bengalese, who let their filthy rags rot off their bodies.[5] This is especially noted in Dehra Doon, where leprosy is very prevalent. Thomson[6] notices the same disgustingly filthy state of the New Zealanders affected with "ngerengere,"

[1] Coll. Phys. Rep. pp. 78, 93, 133, 191, xvii. Wortabet, *op. cit.* p. 189.
[2] Rep. on Lep. in Norway, p 55.
[3] Hugo Arnot, "Hist. of Edinburgh," p. 61.
[4] Zambesi, p. 119.
[5] Coll. Phys. Rep., pp. 133, 15.
[6] *Op. cit.* p. 501.

and specially notices that the inhabitants who bathed frequently had not the disease among them.

But that no amount of filth can ever *produce* leprosy is clearly enough shown by its absence from the slums of London, and the Cowgate of Edinburgh, within the last two decades. The natives of uninfected parts of Norway, again, are neither more nor less cleanly in their homes or persons than those of the infected parts. The same may be said of Greece [1] where there are "vermin everywhere." The filth of the "noble savage" of North America I have already referred to,[2] while that of the Esquimaux,[3] of the natives of California,[4] and of Nootka Sound,[5] among none of whom has leprosy ever been seen, show that something more than simple filth is required to produce it; while, on the other hand, its spread among Europeans in hot climates, where to them the daily bath (often twice daily) is an absolute necessity; among the Foulahs of Central Africa, who "often wash the whole body,"[6] and are "neat and clean;" and among the Sandwich Islanders, also a cleanly people,[7] shows that cleanliness alone is not of itself sufficient to check its spread, once the contagion is introduced among a population prepared by other circumstances for its attacks.

Putrid Food.—The theory that the eating of food in a putrid or semi-putrid state is a *bonâ-fide* primary cause of leprosy has been advocated by several authors, whose opinions are worthy of being treated with respect, and has, at first sight, some plausible arguments in its favour. On more careful and extended examination, however, it will not hold good.

The use of tainted and putrid food by tribes of Central Africa, among whom the disease is prevalent,[8] also among Negroes in Surinam,[9] among the Chinese to a limited extent,[10] among some of the natives of India,[11] as also in Bergen in Norway,[12] in Portugal,[13] at Jerusalem,[14] and finally among the natives of New Zealand,[15] who purposely allow their maize to become putrid—all appear to prove that the use of such food has some essential connexion with the origin of leprosy; but we must remember that the description[16] of the food given by Schillingius can only be applied to Surinam,

[1] Roving Englishman, *Household Words*, 1853, p. 473.
[2] Welch (*Lancet*, vol. ii. 1874, p. 796) speaks of their filthy condition in New Brunswick. [3] Beechy, p. 365 (and others). [4] *Ibid.*, p. 76.
[5] Cook, vol. vi. p. 279.
[6] Caillé, pp. 223 and 225. [7] Ellis, vol. ii. p. 77.
[8] Du Chailu, pp. 390 and 457; Park, p. 51 (putrid fish; Livingstone, Zambesi, pp. 119, 305, and 373; Last Journals, vol. i. pp. 121 and 131, and vol. ii. pp. 41 and 149 (Manyuema Cannibals). Livingstone only speaks of "partial epers" in the last quoted instance. [9] Schillingius, p. 21.
[10] Davis, vol. i. pp. 235 and 404, and Durand Fardel, *op. cit.*
[11] Coll. Phys. Rep., pp. 136, 157. [12] Carter, Rep., 1876, p. 146.
[13] Peacock, *Lancet*, 1870, vol. ii. p. 776.
[14] T. Fox, *Ed. Med. Jour.* 1868, p. 802.
[15] Thomson, *op. cit.*, p. 501, and Cook, vol. ii. pp. 44, 46.
[16] They still sometimes eat putrid salt-fish in Surinam (Landre, p. 62).

the West India negro, at least of the present day in the British Islands, not using putrid food.[1] Among the Chinese, again, I have already said rice is really the chief diet, while in India it is also with pulses of various kinds the chief food, animal food being entirely prohibited to the Hindoos, while the use of putrid fish is by no means common, being only noticed a few times in the College of Physicians' Report. Nor do the places where the use of such food is noticed correspond in the least degree with the places where leprosy is most common, as for instance in Meerut, where there are only 2·9 lepers per 10,000 of population, and in Chittagong, *with the fewest lepers in the Presidency of Bengal* (1 in 3679), putrid fish being used as food is spoken of, while in Dehra Doon, with 19 in 10,000, no such habit is remarked on; and in Kumaon (21 in 10,000) Drs Lewis and Cunningham did not observe it. Dr Macnamara also says,[2] "the natives (of India), as a general rule, are not in the habit of consuming putrid fish," and points out that in Behar, where leprosy is common,[3] the people are cleanly, bathing daily, and are on the whole fairly fed, as described in the Report he quotes from.[4] The food is chiefly vegetable. The existence of the disease among Europeans in the tropics could have no connexion with the use of putrid food.

On the other hand, too, leprosy is *not* seen among many tribes and peoples who daily use putrid food—as the Kalmucks, various Mongol tribes,[5] in Dahomey,[6] and among the natives on the west coast of America, from Tierra del Fuego to Vancouver's Island,[7] as well as among the Esquimaux of the north coast, to whom a dish of whale's blubber, which has been buried some weeks or more and has become thoroughly putrid,[8] is as great a delicacy as "high" pheasant on the tables of the higher classes in England. The natives of Nootka preferred absolutely putrid whale flesh to fresh.[9]

Thus, there is no proof that the use of putrid food, as such, has any influence on leprosy, although it may, from its loss of nutrient power, play a very subordinate part in the manner I have indicated that "poor food" probably does.

The question may be asked, Can the use of fish, with famine, filth, exposure, and putrid food produce the disease? This is answered by

[1] By an Act passed in 1798 (see Antigua and the Antiguans, 1844), affecting all the Leeward islands, each slave was bound to receive nine pints of corn or beans, or an equivalent in wheat or other flour or meal, or double the quantity of potatoes or other esculents, or thirty pounds of plantains weekly; also one pound and a quarter of salted or double that of fresh fish, all of good quality; showing that three quarters of a century ago the Negroes were by no means starved or forced to eat putrid food.

[2] *Op. cit.*, p. 36. [3] 1 in 1800, Census of Bengal, xcviii.

[4] See Coll. Phys. Rep., p. 136. [5] Prejavalsky, *lib. cit.*, vol. ii. p. 240.

[6] Sketchley, pp. 59 and 491.

[7] See Bougainville, in Kerr's Collection, vol. xiii. p. 189; Wallis, *ibid.*, vol. xi. p. 140–2; Candlish, *ibid.*, vol. x. p. 76; Cook, vol. v. p. 295; also Darwin, *lib. cit.*, vol. ii. p. 214.

[8] Beechy, pp. 371 and 432. [9] Hewit, *lib. cit.*, p. 135.

its absence in Tierra del Fuego, where all these conditions are found in the highest intensity, the climate being one in which, even in summer, sleet and snow are common, while clothing can hardly be said to be worn, and the habitations, mere temporary erections, are not worthy of the name of huts, the state of filth in which the people live is horrible to think of, and the most delicious *bonne bouche* they ever obtain is a mass of putrid whale, their ordinary food being shell-fish.

Race.—Some have supposed that certain races have less tendency to the disease than others; thus the Arabs of the Malay Archipelago being free from it is supposed to be partly so accounted for,[1] but as the settled Arabs of Algeria are stated to be more affected by it than the Kabyles,[2] *race* can have nothing to do with their exemption in Java and adjoining countries. The reason already mentioned—their separation from other infected races—is more satisfactory. Europeans, again, are supposed to be more exempt than black races;[3] but Carter himself, the chief author depended on in the College of Physicians' Report, in support of this idea, has now changed his former opinion as to its unfrequency, as he ends his last report (1876) with a short chapter on "Leprosy in Europeans in India," in which he speaks emphatically of the liability of Europeans to leprosy, and mentions six cases known to him in Bombay "without in any way pushing inquiry," while "it is known that there are others." But, as the total number of Europeans in the Presidency of Bombay is only 28,900,[4] including 10,121 soldiers[5] (among whom the disease certainly appears to be almost unknown, *not one instance having occurred that I have been able to hear of* although they may be sometimes affected);[6] and the six known cases appear all to have been civilians, and these six are only a few in the *town* of Bombay, representing an *unknown number* scattered throughout the country; and as even these six would give 1 in 3100 of the 18,779 civilians in the Presidency, while the proportion in the whole population is 1 in 1982, it is evident that there is really no immunity of Europeans, though such might appear at first sight to be the case from their relatively small numbers in the country.

[1] Liveing, p. 66.
[2] Dr Ch. Claude Bernard, private letter.
[3] Coll. Phys. Rep. pp. 110, 116 (Carter and Stenhauser), and 158.
[4] Census, p. 260, the Christian population of Aden (Tab. 4, p. 367) being deducted.
[5] Army Medical Rep., 1873.
[6] If this were made a matter for special statistical inquiry, which could be easily done, it would be another strong argument in favour of the contagiousness of the disease, soldiers in India, though living in the country, being *more thoroughly isolated* from the disease than any other class, the native servants in attendance on them being at once dismissed on showing any signs of disease, and the Contagious Diseases Act being fully carried out in regard to the native women with whom they come into contact, while they themselves avoid cases with perfect horror. Some years ago the natives of a leper village near Jansi caused a perfect stampede of all the troops into their barracks by walking through the square. On the other hand, instances are known of *officers* being affected (Coll Phys. Rep., 104, 238, and 241), they having more freedom, and cohabiting with any one they choose. One case was so caused.

On a wider scale, again, the whole British population of India, soldiers excepted (of whom there were 55,425), as shown in the census returns, was 66,155. Now, from various sources I have easily collected twenty-seven cases, arising among that population during the last quarter of a century,[1] representing, of course, only a part (possibly small) of those actually affected, which may be looked on as more than a counterpoise to the fact that all these twenty-seven were not living at one time; yet this number alone gives 1 in 2459, which, considering that the data are so very incomplete, although positive so far as they go, speaks little for any immunity existing among Europeans. The same remarks apply to Europeans in the Dutch East Indies, from which Landré (p. 5) says he has seen about ten cases, and to the West Indies.[2] In St Kitts, I knew of one case in a planter, and have heard of one in a judge, a Scotchman, and I saw three cases (brother, sister, and cousin) in *poor whites*, children of parents who came from Sussex; and I heard of the *recent* death of two Englishmen (brothers), labouring men. This, out of a total white population of about 800, shows no immunity; the numbers are too small to argue from, but, so far as they go, show even a higher proportion than the 1 in 389 of the whole population. The same want of comparative statistics makes the statements as to the smaller number of whites than blacks (relatively) affected in the Cape and the Mauritius of no value.

The Jews in Bombay were stated by Carter to have been exempt,[3] or nearly so, he having *only* known of four cases, but as there are only 2500 Jews in Bombay, the proportion is after all quite high enough—higher, indeed, than in the other populations. T. Fox mentions [4] that it is said "there is no known case of leprosy in a Hebrew at the present time in Syria." That this is not a question of race, however, is shown by his own views as to modern being included in the Mosaic leprosy as one of its forms, by the history of the disease, by its being more prevalent (as far as known) among the Jews of Cairo [5] than among the other races, as is also the case in Jamaica.[6] It is known also in Asia Minor as the Judamata or Jews' disease.[7] The most probable explanation of the extinction or rarity of the disease among the Jews of Syria is, that the segregation enforced by the Mosaic law, combined with the persecutions the people have undergone, and their scattering abroad, during which those actually diseased

[1] Coll. Phys. Rep., pp. 81, 104, 158, 235 to 243 (15 cases); Hutchison, *op. cit*, p. 96.; Carter, 1876, Report, p. 36 (10 cases); and one case seen at the Edinburgh Med. Chirurg. Soc., in 1874, from Rungpore.

[2] See Milroy, Rep, p. 2; Coll. Phys. Rep., pp. 45, 46, and 85; also Brassac's Rep. (several cases); Guy's Hosp. Reports, 1859, p. 141.

[3] Coll. Phys. Rep., p. 110. [4] *Ed. Med. Jour.*, vol. i., 1866, p. 802.

[5] Coll. Phys. Rep. p. 53.

[6] *Ibid.*, p. 12. (One of the cases mentioned in Guy's Hosp. Reports, 1859, is that of a Jamaica Jew.) [7] *Ibid.*, p. 69.

and outcast would necessarily be neglected and starved, have stamped out the disease from among them. Under the head of history I might have observed, but do so here, that on a comparison of the writings of the Jewish traveller, Benjamin of Tudela (1160–73), in Western Asia and Europe, with notices of the places in which leprosy was most prevalent in the Middle Ages in Europe, and still remains so in Persia, that the very places where he mentions the greatest number of Jews, as in Samarkhand[1] (50,000 Jews), are or were the most affected by leprosy. From these, therefore, as well as from other examples mentioned in the history, it is clear that no race as such is ever exempt or likely to remain so from leprosy so long as, under favourable conditions for communication of the disease, an already affected race is brought into contact with it. At the same time, it is possible that there may be a greater tendency for the disease to *originate* among the negro races than others, and races most in contact with them are most infected, as in Morocco, a country in which the negro race dies out, but where an enormous importation of them has continued for centuries,[2] and where all races are consequently infected to a great degree.

Having thus considered the chief causes which have been supposed to have some connexion with the mode of origin of leprosy, I may be permitted to mention what, from my study of the history of the disease, I consider to be a most likely *causa vera*, possibly capable under favourable circumstances of causing the disease to arise *de novo*, after which it is propagated by contagion.

We have seen that the disease had, so far as history can teach us, two great centres of origin, Northern Central Africa and India. Now, it appears to me *that it is in the conditions obtaining in those centres of origin*, and specially in such conditions as differ from those obtaining in all other parts of the world, and more particularly in such conditions as those centres agree in, that we ought to look for the causative influences of leprosy, and *not* in the conditions of life in countries into which it has been imported and spread by contagion. Now, in India as in Africa, *want of salt, combined with the use of vegetable food*, are the prevailing characteristics of the diet of the mass of the people, and, as I have already said, what is the state of the population, has been so in India[3] and Africa for ages.

As to Africa, this great want is increased by the want of convenience for carriage, so that to some parts it has to be carried 700 miles chiefly on men's heads. Du Chaillu[4] mentions that among the Apingis, an inland equatorial tribe, it is so scarce that ten pounds will buy a boy slave. Parkes[1] mentions that a cake

[1] Kerr's Collection, vol. i. p. 102, etc.
[2] Sultan Muley Ismail (1727) imported 100,000 in a few years.
[3] Census of N.W. Provinces, p. lxxiii.
[4] *Lib. cit.*, pp. 456 and 289. [5] *Lib. cit.*, p. 305.

of salt, 14 × 2 × 2½ inches (containing about 70 cubic inches), which had been brought from the salt mines in the Sahara to the Niger country, costs usually two pounds or more. While Caillé[1] speaks of such cakes at Jenné, brought 650 miles inland by the Foulahs, as costing equal to four pounds; and there, Jenné being a large town, it is considered "common enough, obtainable by every one;" elsewhere, throughout the country, only the *very rich* were ever able to taste it. Bonnat, lately a prisoner in Ashantee, speaks, in a paper read before the *Societé de Geographie Commerciale*, of its being very dear at Selza, in Ashantee, where it costs five pounds (125 francs) a ton. But Selza is only about 130 miles from the coast.

In India the same want is felt by millions. In the Census of the North-West Provinces[2] it is stated that the "small farmer eats salt every day or two days, the labourer once in eight days, or in small quantities occasionally;" and it must be remembered that in one part of these very provinces, at Jansi, there are salt mines, but *from the great difficulty of carriage* it is too dear for labourers to obtain; as Dodd says,[3] "it may be confidently said that the Government which would place within reach of the poor cultivators (of Hindostan) an ample supply of salt would be sure to receive the blessings of millions." It is nothing uncommon for cargoes of salt to be sent from London to Calcutta. The want of good roads has been the curse of India.

Since my attention has been turned to this point some years ago, I have made it a special object of study, and can unhesitatingly assert that in no part of the world, as at present known, is the same scarcity, combined with a vegetable diet, observed, with one remarkable exception, New Zealand, where the natives do not use salt,[4] and where, *in the interior*, in former times, when every tribe was at constant war with its neighbour, it was impossible for the tribes of the interior, *the very place where Thomson saw Ngerengere*, to obtain salt in any form.[5]

In South Africa there is salt in the Kalahari Desert; and among the Bakwains, though it is scarce, meat is plentiful.[6]

In Southern Central Africa, as may be gathered from Livingstone's Zambesi and Last Journals, while it is abundant in some places, as in the Nyanza Country and to the south of Tanganyika, it is less obtainable in others, but at no spot has it to be carried more than 200 miles.[7] It is extracted by the Manyuema from

[1] "Travels through Central Africa," p. 465. Leared also (p. 193) speaks of its being sent from Morocco to Timbuctoo.

[2] P. lxvi. [3] *Lib. cit.*, p. 104. [4] Savage, *lib. cit.*, p. 60.

[5] I have already spoken of leprosy existing among the Fijians; it is worthy of note that they also *will not* use salt.—*Two Years in Fiji*, by Litton Forbes, M.D., p. 189.

[6] Livingstone, "Travels in South Africa," pp. 77 and 133.

[7] See "Zambesi," pp. 132, 216, 225, 586, and "Last Journals," vol. i. pp. 34, 98, 106, 176, 212; and vol. ii. 19, 56, 104, 116, 217, 256, 335.

grass roots. I observe also that Livingstone chiefly remarks on a want of it when at a distance from the great centres of population, as, on reaching Lake Nyassa after a journey from the Rovumma, and when two-thirds of the way between Nyassa and Tanganyika.[2] Besides, in Southern Central Africa *animal* food is much more abundant than in the north.

In regard to the rest of the eastern world, unlike Africa (which, as Livingstone says, is the oldest continent in the world, not having been repeatedly the basin of a great ocean, as Europe and Asia have been), it is, at least north of the Himalayas, abundantly supplied with salt The great plain of Europe and Asia, from the shores of Holland to the Yellow Sea (of which, indeed, the Sahara Desert itself is only a branch extending to the south-west), is one vast upheaved ocean bed, over which salt is necessarily scattered everywhere, as at Berchtesgaden in Bavaria, Wrielizka and Bochnia in Poland, Louvra in Hungary, Hallem in Upper Austria, and also in Catalonia in Spain, and Altemonte in Calabria, on the Steppes, in Transcaucasia, in the salt marshes to the south of the Caspian in Persia, as salt plains and wells in Thibet,[3] in the Tartar Desert,[4] and in the interior provinces of China, as Yunan,[5] as well as in Siberia at Okhotsk.[6] Thus, apart from the supply from the sea, there is no lack of salt in the interior of the old continents, and the carriage of it has always been easy in Europe, while in Central Asia it is so thickly scattered that carriage could hardly be said to be required, and horses are plentiful.

In the New World, in North America, west of the Rocky Mountains, most of the tribes have been animal feeders, and there has been no want of salt, it being found on the Alleghanies, in numerous salt lakes over the prairies, and in the Great Salt Lake, the Dead Sea of America.

The Mexicans depended on their supply from the sea-coast, although there are some saline plains in Mexico; and here I wish to point an example showing that simple want of salt itself as a condiment would not produce leprosy. The Tlascalans, enemies of the Mexicans, and surrounded by them, were thus, as Prescott mentions, deprived of salt, so that they did not care to use it, having been so for about half a century; but they had food of all kinds in abundance.

In South America and Peru there are extensive saline basins, 7000 feet above the sea level; near the Rio Negro in Patagonia,

[2] As at *ibid.*, p. 56. One remarkable exception to the, on the whole, sufficient supply of salt in Southern Central Africa, is in the great Barotse Valley, where "a kind of leprosy peculiar to the Barotse Valley" is mentioned. ("Travels in South Africa," pp. 503 and 600.)

[3] Marco Polo (in Kerr), p. 345; and Huc, vol. ii. and Prejavalsky, Mongolia, pp. xxv., 24, 52, 108, 116, and 118; also Humboldt, p. 409.

[4] *Ibid.*, vol. i. p. 70.

[5] Davis, p. 140; and Yunnan, R. K. D., in *Cornhill Magazine*, 1866.

[6] "All Round the World," p. 308.

not far from the sea-coast[1] (about forty miles) are large salinas, where the salt is crystallized in cubes; and before horses were introduced into the country, the inhabitants seem all to have lived near the coast so as to obtain salt, as all their tombs are found there.[2] On the upper waters of the Amazon the Indians extracted salt from the ashes of a palm-tree by lixiviation.[3] Thus, apart from the modern use of salt-fish, which is so universal, the natives of South America have always been able to obtain salt.

Thus, in no country in which salt is abundant, or where it is not so, but animal food is plentiful, has leprosy ever originated, while the distinguishing characteristics of the two great centres of origin of the disease, are want of salt, combined with a vegetable diet, and that in very insufficient quantity.

Carrying the inquiry even further, it is worthy of remark, that the millet of all kinds (both the Sorghum and Pannicum), which forms the staple food of Northern Central Africa, and is used to a very great extent in India, as well as the maize and rice, and the pease and barley, which mixed form the "gram"[4] so much used in the latter country, are all deficient in chlorides, so that those using them are not partially supplied even in their food with salt, as is the case with even the poorest diets having other grains, as oats and wheat, as their bases or as adjuvants, these grains containing an appreciable quantity of either sodium or potassium. In the grains used in Africa and India also, the quantity of *soda* and *potash* is comparatively small.[5] Even a poor diet, such as that of the "Scotch Local Prisons, Lowest Diet, 1851,"[6] contains about twelve grains of salt, of which about one-half would be in the one ounce of meat, while a man using one pound of fresh meat daily, with other generous diet, obtains about 100 grains, besides what he uses as a condiment,[7] in which form he would use nearly three times as much,[8] showing a great contrast to the absolute deprivation of it suffered by the people of Africa and India.

As to the necessity for salt to the healthy carrying on of the functions of the animal body there cannot be a doubt, when we consider the intense craving for it, and its visible effects on the condition of animals obtaining a proper supply of it. As every breeder of cattle knows, they at once fall out of condition if fed even on rich pasturage where there is no salt, unless it is freely

[1] Darwin, *op. cit.*, pp. 65 and 78.
[2] *Ibid.*, p. 169.
[3] Humboldt, Travels, p. 262.
[4] Census of N.W. Provinces, p. lxv.
[5] See Blyth's "Dic. of Hygiene," etc., pp. 68, 310, 494, and Parkes, p. 208.
[6] See Playfair's "Tables of Nutriment in Various Dietaries, 1860," table xi. (For short terms of imprisonment.)
[7] For bases of these calculations see Blyth, p. 362, table and footnote.
[8] In six months' observation in my own family (counting two children of five and six years as one adult), I found that not quite 300 grains were purchased daily for each adult.

supplied to them at the farm, when they lick it eagerly. In Australia (where there are large salt lakes) apparently poor pasture ground is preferred to rich, if there is plenty of salt on it. The same applies to camels, who keep in condition with, but lose flesh without, salt.[1]

I have seen it stated that human flesh is salt-tasted, and this is probably one cause of cannibalism in those deprived of flesh food.[2] Salt has been venerated from the earliest times, and was the chief thing used by the Romans, with bread and cheese.[3] All human beings, and especially vegetable feeders, have an instinctive craving for it. The Indians of Brazil ride long distances to obtain it, and their children eat it greedily.[4] Werne,[5] speaking of the Keks, and Baker[6] of the Obbos, attribute their disgusting habit of mixing milk with cow's urine to a desire to retain the salt, of which they cannot get a supply otherwise. In China, the wild hill tribes make raids on the Chinese villages in Yunan on purpose to obtain it.[7] Such examples show that no vegetable-eating tribe will do without salt if it is within their power to obtain it.

Having thus shown that it appears to be a necessary article of food, we will now consider shortly what are the probable consequences of a total deprivation of it. The chief action of salt in the body is, as is well known, to dissolve albumenoids. If the system is deprived of it, would we not expect that the albumenoids kept in solution by it should become deposited? Now, this is exactly what takes place in leprosy, in which, in the tubercular form, there is a deposit of albumenoids under the skin—in the non-tubercular, between the tubules of the affected nerves. All other changes follow from this, which is the primary one.[8]

[1] Prejavalsky, *lib. cit.*, p. 122.

[2] As, for instance, among the Fijians (Boddam Witham, *lib. cit.*, p. 345, "cannibalism caused by hunger"). He elsewhere mentions, that "for generations the chiefs of the mountains were the hereditary enemies of those lower down" (as in New Zealand); thus, these were deprived of salt, and "the common people were forced to be vegetarians," and droughts caused great distress. Such distress seemed to be only temporary, however, as Williams ("Fiji and the Fijians," 1860, p. 100) states that they had "abundant" food.

[3] Adam's "Roman Antiquities," p. 411.

[4] Darwin, *lib. cit.*, p. 110. He remarks that the Spanish Guachos, who are *flesh-fed*, "and lead the same kind of life," use hardly any salt.

[5] "Expedition to Discover the Sources of the White Nile," 1840, p. 278, etc

[6] "Albert Nyanza," p. 240.

[7] *Cornhill Magazine*, August 1876, "Yunnan," by R. K. D.

[8] Carter, *Trans. of Med. Soc. of Bombay*, 1861, pp. 60–68.—*Lancet*, 5th April 1873; Report on Leprosy and Elephantiasis, 1874, p. 3; *Trans. of Path. Soc.*, 1877, p. 2. I have already mentioned that Carter speaks of the brown "leprous elements," described by him in 1876 (*Path. Soc. Trans.*), as occurring in other skin diseases. They can hardly, therefore, have the importance in causing neuritis and other symptoms that he seems inclined to ascribe to them. See also Daniellssen and Bœck, *lib. cit.*, pp. 234–260. E. Wilson, *Lancet*, 15th February 1873. Coll. Phys. Rep. lxxiii.

The increase in albumen and fibrine in the blood, described by Daniellssen and Boeck[1] as existing previous to deposits or exudations taking place (the value of the analysis proving which is, however, denied by Kjerulf[2]), is probably due to the deranged state of the liver interfering with the proper excretion of urea.

And as chloride of sodium is, like chloride of ammonium, a liver stimulant, although not perhaps so active as the latter, which is probably the most active liver stimulant we possess,[3] and is, in fact, *the* liver stimulant supplied by nature so abundantly, the entire deprivation of it would tend to produce functional derangement, and with that derangement the feeling of intense langour, which is so marked a symptom in the earlier stages of leprosy, probably caused by retention of albuminoids in the blood. Thus, the deprivation of salt would in another manner assist in producing the primary phenomena of leprosy.

As showing where the disease seems to begin, I may mention that these exudations or infiltrations are first noticed in tuberculated leprosy around the bloodvessels of the corium,[4] where are seen elongated spaces occupied by round, nucleated (sometimes fusiform)[5] cells. In non-tuberculated leprosy these changes take place in the interfibrillar spaces of the nerves. The cell proliferation is looked upon as a neoplastic, not an exudative, formation by Virchow; but the later researches of Carter (1876) do not agree with this, the cells being, according to him, formed, at least primarily, from the exudation round the bloodvessels, and in the line of the lymphatics. Possibly both views may be reconciled, there being firstly exudation, and neoplastic formation of cells in it afterwards by cell division. The brown pigment granules already mentioned Carter looks on as new formations; but they are, as I have already said, found in non-leprous skin diseases.

In thus attempting to show that want of salt may be the primary cause of leprosy, I must not be understood, in the least degree, to indicate that all cases are so caused—what I have already said as to the communicability of the disease precludes this. If the theory I have tried to sketch out is correct, it would only account for primary cases in India and Africa, such cases becoming centres of contagion for others. I may here venture to say, that I do not see that any sufficient proof of the existence of any specific leprous poison has ever been advanced, but that all proofs at present available would rather show that all the diseased tissues of a leper are of themselves poisonous, and contain within themselves the power of causing similar diseased changes in the healthy tissues of others.

[1] Pp. 234 and 260.
[2] Virchow, Arch., *op. cit.*, p. 508; and Hebra, vol. iv. p. 172.
[3] As was ably pointed out by Murchison, in his classical Lectures on Functional Diseases of the Liver, *Br. Med. Jour.*, vol. i. 1874.
[4] Carter, in *Path. Trans.*, 1876, p. 297. [5] Virchow, *op. cit.*, p. 514.

Since forming, from my study of the subject, the ideas as to the causation of leprosy I have ventured to express, I observe that Dr Dickenson,[1] from his extensive observations on kidney disease, looks on deficiency in potash as a cause of lardaceous disease, in which, as in leprosy appears to be the case, there is firstly effusion and then growth of nucleated tissue. This has a close analogy to the deficiency of sodium salts, which I am led to believe is a cause of leprosy, though it may be that the want of the chlorine of common salt is of as much importance as that of the sodium.

I have thus endeavoured to put forward my theory of the primary production of leprosy, not, I hope it will be understood, in any spirit of dogmatism, but as one which appears to be best supported by the history of the disease in its early homes,[2] the conditions of life in which alone are of value in attempting to ascertain its true etiology, and by the consideration of the chemical action of the particular article of diet so deficient in those countries in the food of the people, this being taken in connexion with the pathology of the disease. Whatever value the theory may have, researches yet to be made, I hope, by such workers as Vandyke Carter, will show. If it is a correct one, then it is possible that at the present day cases are arising daily in India from the causes pointed out,—cases which might be prevented were a network of railways, roads, and canals spread over the country, putting salt or animal food within the reach of all. Such a time *may* be far distant, but it is one to be sincerely hoped for.

We will next consider the question of the heredity of leprosy.

Heredity. — Perhaps no opinion in regard to the etiology of leprosy has been more constantly or more confidently put forward than the one that it is certainly or even chiefly hereditary,[3] so many, to a superficial glance, appear to be the proofs that such is the case, while, on a careful examination of these proofs, they will be found to be almost, if not quite, as Landré thinks they are,

[1] *Br. Med. Jour.*, 3d June 1876, p. 684.

[2] As another work proving the communicability of a disease by tracing its history, as I have tried to do in regard to leprosy, I may here be allowed to mention the classical history of cholera by Dr C. Macnamara.

[3] See Schillingius, *op. cit.*, pp. 31 33. (He, be it remarked, states distinctly that children of lepers, while most obnoxious to the disease, and taking it early when they remain with their parents, *escape if they are removed from them;* thus, of course, being removed from the *contagion.*) Pruner, *lib. cit.*, p. 172. Macnamara, *op. cit.*, p. 17—"80 per cent. of 69 medical officers reporting think it hereditary." Dan. and Boeck, *op. cit.*, p. 82 *et seq.* Wise, quoting Hindoo writers, *lib. cit.*, p. 259. Wortabet, *lib. cit.*, p. 189. Hebra, vol. iv. p. 186. Planck in *Br. Med. Jour.*, vol. i. p. 434 (1877). Simpson, *op. cit.*, 1842, p. 406. Coll. Phys. Rep., p. 231 (Wilson), and elsewhere.

worthless, and at the most only point to a possibly hereditarily received predisposition as a cause in a few cases, which cases, however, might be as well, if not better, explained by the theory of contagion.

The proof chiefly relied on has been the occurrence of the disease in more than one member of a family, using the word as inclusive of uncles, aunts, and cousins. An example of this kind of reasoning is seen in the description by Liveing of a case of leprosy coming from Guernsey,[1] in a man whose *father* became a leper in India, and he (the son) was attacked at the age of fifteen. He had, Dr Liveing states, several brothers and sisters older than himself, *all* healthy, yet, in the face of this, Dr Liveing at once jumps to the conclusion that "it is really a hereditary case of the disease, though I admit there is some doubt on this point." But the father *cohabited with a coloured woman in India;*[2] if, as seems reasonable to believe, *he* took it from that woman, surely it is more likely that his son, living with him, took it from him than that heredity is the cause of the son's illness. Besides, Dr Liveing does not appear even to have inquired whether the father became a leper before or after the son's birth; if *before*, there might be some colouring given to the idea of heredity; if after, nothing but contagion could account for the son's attack. It is a great pity that the inquiries in such an important case should have been so very insufficiently made.

I have spoken at length of the case, because it gives a good example of the style of reasoning which has been used in regard to the heredity of the disease, Danielssen and Boeck themselves not having distinguished the periods of birth of children with respect to the times of life at which the parents became affected, and having always accepted the fact that two members of a family were affected as sufficient proof of heredity. This has also been done by Planck, Carter (up to 1874, but not in his last report, in which he admits the probable contagiousness of the disease), and many others. The practical outcome of this error has been that, reasoning in a circle, those authors have denied that the disease is contagious, because, being hereditary, cases occurring in the same family are so accounted for, while at the same time its non-contagious nature is stated as a proof of its hereditariness. It would just be as reasonable to say that scarlet fever is hereditary, because, as is constantly the case, children take it whose parents

[1] *Med. Times*, vol. ii., 1877, p. 644.

[2] This is the first case I have heard of, of a *private soldier* (which the father was) in British India contracting the disease, and, as an exception to the rule, bears out in a remarkable manner what I have already said as to the reasons of the immunity of soldiers, viz., the want of opportunity for the action of contagion, this man having obtained these opportunities, and suffered the consequences. The case is like that of the officer mentioned in the Coll. Phys. Rep., p. 241. Landré speaks of *Dutch* private soldiers being affected, while ladies never are, they never being exposed to contagion (p. 45).

have had it, and two or three members of a family have it at one time.

Let us for a moment ask, how is a hereditary disease known to be so, and then see whether leprosy shows such characteristics as warrant us in classing it among such diseases.

A hereditary disease is, then, one transmitted from parents to offspring, from *generation to generation*, or grandparent to grandchild, and under ordinarily favourable circumstances for its development, and often, in strongly hereditary [1] diseases, as phthisis, scrofula, etc., without such favourable circumstances; it is so transmitted whether the children live with their parents or not. The tendency to attack in the children is greatly increased by intermarriage of affected persons, and when there are a number of children in one family, generally about one-half, and often all or nearly all, are affected, especially when the parents suffer from the disease at the time of, or previous to, the birth of the children; finally, the children are *always affected after the parents*. I have no hesitation in saying, that no proofs can be brought forward to show that leprosy has the above-mentioned characters.

Firstly, let us see the value of the argument founded on more than one person being affected in a family, the word being used to include all relations to the fourth generation, as Danielssen and Boeck have done,[2] and from which they have concluded, that in 213 cases 189 were hereditary, such cases being most frequent on the *maternal* side and in the second and fourth generations. Now, when we remember that, if all relatives within the fourth degree are included, as many as 50 to 100 or more persons would be taken in, there is nothing wonderful that in Bergen, where every four-hundredth person is a leper,[3] or in St Kitts with nearly the same, even apart from contagion, some families should have two or more members affected. Again, as a man has twice as many grandparents as parents, this would account for the greater frequency in the second generation.

But is the argument founded on collaterals being affected of any value? I think not. In the cases I inquired into in St Kitts, out of 72 cases, in 8 the family history was uncertain, but in 2 of these 8 the uncertainty was only in regard to the grandparents, all others were healthy. Among the remaining 64 the most careful inquiry from the patient's friends and residents on the estates in regard to relations could elicit no history of leprosy in the family in 34 cases.[4] The other 30 had leprosy in both lines in 4 cases,

[1] Nouveau Dictionnaire de Med., tom. xvii. p. 451. [2] *Op. cit.*, p. 335.

[3] 1 in 508 in south and 1 in 272 in northern department.

[4] Landré (pp. 32 and 34) quotes Beiro, of Portugal, who only found 5 in 43 with even one other member of the family affected. Van Someren in Madras found only 2 in 31 cases descended from lepers. Porteus (Coll. Phys. Rep., p. 103) found among 31 lepers the mothers of only 2 had been affected, and 2 fathers; and Dr Day knew of many instances where only one member

in the direct line only in 5, and in the indirect line only in 21 cases; of these 21, 3 were in brothers or sisters *living together*, one case only becoming affected four years after her brother, who was attacked in Antigua, but returned and lived in the same house with her. There were 5 in brothers and sisters, and also in uncles and cousins besides, and 13 in uncles, aunts, or cousins (third cousins included). Now, of these 13 I found that 8 were in more or less continuous communication with the affected relatives; in one illustrative case, the boy having lived with his aunt while she was sick, apart from his mother. In 2 cases there was uncertainty as to contact, but in one of those who had been twenty years sick, his leprous aunt died of cholera three years after his attack, and he had lived all his life in one village beside his family and relations. In 3 instances only was it stated that there had been little or no communication, but of the truth of this statement I am more than doubtful in one case; and in another case whose half-niece was affected twelve years *before* him, he attributed his illness to *sleeping with a leper*—an example of the danger of concluding that such a case is necessarily hereditary, because there has been no contact with the affected relatives.[1]

From these figures it is clear, that unless the disease were proved to be non-contagious, the fact of its existing in the collateral line is no proof of heredity. The following series of cases will more forcibly illustrate this:[2]—William Mully, a white labourer, was attacked in 1857, having with his brother come to St Kitts in 1835; he died in 1862. Meanwhile, George Mully, his brother, took ill about 1861–62; he died in 1868. Thomas Naylor, a son of a second cousin of those brothers, born in St Kitts (the father came out in 1835, having been at school in Suffolk with the Mullys), was attacked about 1862–63; then a cousin of Thomas Naylor, William Hart, who, in consequence of his mother's death, lived four years with his uncle and cousins *while T. Naylor was sick*, and also lived on the same estate as George Mully and beside him, took sick about 1868. Lastly, the sister of Thomas Naylor was attacked in 1869. Now, had these been blacks, a more apparently conclusive suite of "hereditary" cases in the collateral line could hardly have been imagined; but as the Mullys were English and the three others of direct English descent, this idea is utterly untenable; while as they were naturally, being *poor whites*, from the same village, and living near one another, in constant communication, contagion would at once account for all the cases.

was affected. Porteus mentions that the 31 lepers had 111 healthy brothers and sisters.

[1] Dan. and Boeck, p. 514, mentions a case in Provence, whose sister was leprous, but who blamed sleeping with a leper as the cause of his disease.

[2] The necessarily total absence of heredity in the Sandwich Islands, already mentioned, is worth keeping in view when considering these cases.

I think I have said enough to show that the existence of leprosy in the indirect line is no proof of heredity.

But in 4 of the 72 cases there was leprosy in both lines, and the question arises, would this prove heredity? In the *first* of these four, Rebecca Fleming, age 30 years, the mother's mother and mother's half-brother were affected. But the latter was attacked twelve years *after* Rebecca, who was attacked when 15 years of age (his is the case mentioned already who slept with a leper), and it is more than probable that she was in contact with her grandmother, living as she did on the same estate. I may say here, that her mother's husband's daughter by a first wife, Catharine M., who *slept with Rebecca while she was sick*, became affected, although there was no leprosy in *Catharine's* family.

In the second case, Drusilla Elliot, age 24, attacked at 21 years (about 1868), her mother and niece were lepers, but the niece Ann Peats (the third case) was sick twelve years *before* the aunt. Ann lived about three miles from Drusilla in 1872, and there was no constant communication between them then, both being ill; but I believe that such communication took place previously. Drusilla's mother died in 1856, having been many years a leper, being one, so far as I could ascertain, when Drusilla was born.

Thus, Drusilla may have been infected by her mother previous to the latter's death, the twelve intervening years (if it really was so much, for it is possible and even probable that slight symptoms existed unnoticed for some years before 1868) being a perfectly possible period of incubation, or from Ann Peats, or, as in the case of Rebecca Fleming's uncle, from some other leper. All these possibilities would have to be disproved before the case could be looked on as proved to be hereditary. The third case, Ann Peats, born about 1854, became a leper about 1856, the year her grandmother (Drusilla's mother) died. From the confused statements made, it is uncertain whether her grandmother nursed her, it being stated that the latter was dead before Ann was born; but this, if the dates were correct, is an evident error, being an instance of the difficulty of obtaining such information among negroes. Even if she never did come in contact with her grandmother, however, the idea of heredity is negatived by the fact, that Ann was the *only child attacked in a family of thirteen brothers and sisters.*

The fourth case, Mary Jackson, whose father and second cousin were lepers, was the only *member of her family who lived with her father when he was sick*, he being separated from her mother. Thus, these cases also can be most readily accounted for by contagion.

Lastly, there were *five* cases with the disease in the direct line only. Of these, two were in a mother, R. P., and son, the latter 6 years of age. The mother was attacked, and some time afterwards the child, when he was about four years old. The mother was apparently healthy when he was born, but her father was a leper; the latter died a long time before. The boy's father was

healthy. I could not ascertain satisfactorily whether R. P. had lived with her father when he was sick or not, but the child was never in contact with him.

In the third case (in which I had great difficulty in getting any information), J. W., age 25, attacked at 19 years of age, stated that her mother was a leper, but not, so far as I could ascertain, till some time after J. W. was born. The fourth case, A. Y., lived with her mother, from whom she supposed she had got it, and was attacked at 26 years of age; she was 40 years old when I saw her. The fifth and last case, Alicia W., lived with and attended to her father when he was sick. She was attacked at 12 years of age.

Thus there is nothing in this series of cases to prove heredity, but rather the reverse, so long as the possibility of contagion by close contact is at all admitted.

I would say here also, that even in such cases as the first two of those five, were the mother even affected before the birth of the child, the fact that the child is a leper is no proof of heredity, in the true sense of the term, as there can be no closer communication than that between mother and child, so that the latter may possibly become affected with the disease as an infant or *in utero*; not, I believe, by hereditary transmission, but by infection.[1]

As to heredity from the father's side, one great difficulty in accepting this doctrine is, that the disease most decidedly prevents men having families.[2] In St Kitts I found that among 18 male lepers over 21 years, 9 were married and 6 had families, all of whom, however, with the exception of two children born to one man, were born *before* the patients became lepers (except in one case in which the last child was born one month after the father's attack).

[1] See Coll. Phys Rep., pp 70 and 102; and case 20, in Danielssen and Boeck, p. 436. I think light may be thrown on this by a consideration of what takes place in so called hereditary syphilis, transmitted from the father to the child through the mother, but which, in spite of the opinions of even such as Trousseau to the contrary, never seems to take place, *unless the mother is previously infected* (*Dict. de Médecine*, tom. xvii. p. 468), being, in fact, syphilis transmitted by infection through the mother. A somewhat similar error is made in concluding that cases of children attacked at three and four years of age with leprosy (whose mothers are lepers before their birth), are so from heredity, such cases being more probably really cases of contagion from the mother, the disease being possibly transmitted during intra-fœtal life or during the nursing period, and incubating for three years or so. It is observed that when the mother is affected at or about the time of the child's birth, the disease is apt to appear earlier in the children than when the father is affected (see Coll. Phys. Rep., p. 102). In only one case did I see children born to a man *after* he became a confirmed leper—these were three and eight years old, and perfectly healthy (evidence, so far as one case is of any value, against the heredity of the disease), so that the full analogy, with the transmission of syphilis, can hardly be carried out; but I have thought it worth while to draw attention to an apparent misuse of the word "hereditary" in regard to syphilis, so that the same error may not be repeated, as it often has been, in connexion with leprosy.

[2] Holmnsen only found 1 case among 12 in which parent and child were affected, in which the parent was affected *before* the birth of the child.

One man, attacked at 49 years of age, had eight children. Of the 9 unmarried, 6, attacked at various periods from 7 to 17 years of age, were simply incapable of being married, having the appearance at 21 and 22 years of age of boys of 15 or 16, shrivelled up by the disease. The other three, aged 30, 34, and 46 in 1872, attacked at 24, 20, and 25 years, had no chance of being married on account of the disease. Thus, if the disease is only to be looked on as possibly hereditary when occurring in children born *after* their fathers are attacked (and this is, I think, the most correct view), not one of the men could transmit it, so far as my observations go, the only two children born after their father's attack being healthy. Such cases show the absurdity of arguing that heredity is the chief mode of transmission of the disease, as it is plain enough that it rather stamps itself out, for when it attacks the male before puberty, it prevents the reproductive powers being developed, when about puberty it prevents marriage, and after marriage puts an end, in the majority of cases, to the reproductive power.[1]

As to females, I found in St Kitts, among 26 women (over 21 years), 9 had had children, 7 having had them *before* the disease appeared. I saw only one woman who had had *two* children *after* she was attacked—two, one 11 days, the other 3½ years old, both healthy; and in one other case, attacked at 34 years of age, who had children and grandchildren all healthy.[2] I am not quite certain whether any children were born after her attack, but believe not. Wortabet appears to be right in saying, that males lose the power of reproduction earliest.[3]

Again, of these 15 men and women, 6 had also grandchildren, yet not one of them were affected. All the families were perfectly healthy, with the exception of the *one* child of R. P. already mentioned, born *before* the mother's attack. In India, Lewis and Cunningham (p. 62) found the following results among 52 lepers:—25 males had had 27 children, 0 leprous, 4 dead; 27 females had had 76 children, 5 leprous, 30 dead, or a total of 5 leprous children among 109, or only taking the *living*, 69, far too small a proportion to show heredity. It is possible and even likely that, had those five been removed at once after birth from their mothers, to avoid contagion, they might have escaped. The proportion given above is even less than the three in 32 reported on in the "Coll. Phys. Rep." (p. 161).

[1] Milroy (p. 5), "In a large proportion of leprous unions (in Berbice) there was no progeny at all."

[2] I have lately heard that one of the seven has had a child since 1872, which died *not* a leper at 18 months.

[3] This nullifies to a great extent the argument that the disease is not contagious, because wives seldom take it from husbands. See also on this, Lewis and Cunningham, pp. 63 and 64, a Table showing clearly that *the earlier the age of attack the fewer children lepers have.* Wortabet also says, that the disease comes chiefly from the mother, owing to loss of reproductive power in males (*op. cit.*, p. 189).

Another proof against heredity is the great number of cases in which there is only one leper in a family, including relatives. I have already mentioned the number in St Kitts, which was 34 in 66, and Danielssen and Boeck,[1] Brassac, Milroy (p. 3), and others, mention cases to the same effect. But even *among the other* 32 in St Kitts, on careful inquiry, I found cases such as the following:—An uncle had been, and his niece and nephew were, affected, but *that uncle himself* was one of a family of *thirteen* children, *all of whom grew to middle age perfectly healthy*, as Dr Boon was able to assure me, he having known them all. And this is only one instance of families of twelve, six, and four brothers and sisters, inquired into by me, in which only one brother or sister was affected, showing of itself that the disease was not hereditary. In no family, including relations, have I ever seen among the blacks more than three members affected, and that of the uncle, nephew, and niece just mentioned was one of these

Reading, therefore, Danielssen and Boeck's Tables, as well as Mr Planck's, and Lewis and Cunningham's statistics (made up, as Dr Carter says, of India in regard to a people who live in little communities), and also, were it necessary, Dr Carter's Tables, in his 1874 Report, which he seems to attach little weight to, as he so readily expresses his belief now in the possibly contagious nature of leprosy;[2] reading those in the light I have endeavoured to throw on them, I cannot think they are of the slightest value in proving heredity.

I have already quoted a case of an aunt affected *after* her niece, and an uncle *after* his niece, and I may also mention the case of a mother attacked *after* her son's death from leprosy. The case was related to me by Dr Boon, an old resident in St Kitts, who knew the lady A similar case is mentioned by Schillingius,[3] and two cases of mothers after daughters by Max of Durand Fardel (*Gazette Méd. de Paris*, 14th July 1877); and a case in which the mother, father, and another child were attacked after an European child, who herself had been infected from a boy (*Landré*, p. 51). These cases clearly point to contagion, the mothers having nursed the children, and are against its being hereditary, as hereditary diseases are handed *to* descendants, not from them.

Again, in hereditary diseases when both parents are affected, it is certain that most of the children will be so, under circumstances favourable for its development. Yet Pruner mentions that he has

[1] P. 376, *et seq.*, "Coll. Phys. Rep.," *passim*; *Gaz. Med. de Paris*, 14th July 1877, etc.

[2] I can pay Dr Carter no higher compliment than to express my admiration for his readiness to give up any received theory which he has given much labour to prove the correctness of, when formerly unknown circumstances show that another theory explains the facts better.

[3] He quotes from Pallas. She was attacked a year after the second son of two affected.

seen "not only children, but married people, quite sound whose parents both died leprous."[1] Lewis and Cunningham's Table (p. 65) shows that in four families with 24 children, both parents being affected, 11 were lepers; but even in two of these families only one out of three in one, and of four children in another, were so. In ten families in which the mother only was affected, 13 children out of 32 were lepers, a slightly lower proportion than when both parents were affected (readily accounted for by the increased chances of contagion), and five of these families, with four to six children each, had only one child affected. In these families, again, in which the father only was a leper, only three children were so—one in each family. Two families contained five each, and one, two children. This would show the less tendency for the disease to follow the father having it.

Taking also the absolute number affected in any family and its relations, as compared with the number of individuals contained in such a circle, the idea of heredity, as I have already shown, is not supported[2]

Finally, it is, to say the least, strange that in China, where lepers intermarry, and are only allowed to marry lepers, that the descendants should be looked on as clean in the fourth generation,[3] an idea which was, still more strange to say, mentioned in regard to the lepers in Martigues,[4] in the south of France, fully a century ago, in one of the very localities in which Danielssen and Boeck, a century later, reported that the disease was propagated by heredity alone.[5]

Now, how could a really hereditary disease ever die out in families in which all the parents were tainted?

In regard to Norway itself, where Danielssen and Boeck chiefly formed their opinions as to the heredity of leprosy, believing that this is the chief cause of its increase in that country (as between 1850 and 1862),[6] as already stated, all the people of the west coast are exposed to the same pernicious influences. Yet, according to

[1] P. 173.

[2] I am aware that the value of such statistics as I have put forward may be questioned, owing to the wish in many persons to conceal the fact that there had been leprosy in the family; but as I made my inquiries with my eyes fully open to this, and never trusted to the statements made to me by the patient alone when other sources of information were available, I have every reason to believe in their correctness. In one case I obtained the family history for five generations back from the boy's grandmother, a most respectable woman well known to me. All were healthy except him.

[3] Landré, p. 37.

[4] "Medical Observations and Inquiries by a Society of Physicians in London," 1757, p. 204. "An account of the leprosy in Martigues, extracted from a French letter to Dr Clephane from Dr Joannis, a physician at Aire, dated 15th October 1755."

[5] P. 514.

[6] In 1850, 1500; in 1862, 2119 lepers, in nearly two million inhabitants.

the observations of Kierulf,[1] cases arising *de novo, i.e.*, according to him, not from heredity, *always arise in places where the disease is endemic, and never where it is unknown*, while, according to Holmsen,[2] when it arises in a new district *it is always caused by a leprous individual*. These facts are entirely against heredity.

Summing up, therefore, leprosy is not always, but only very rarely, transmitted from generation to generation, has never been proved to be transmitted without contact, is not constantly transmitted even when both parents are diseased, seldom affects more than one child in a family, and those only successively, independently of age, sometimes the youngest first, after contact, and goes back from child to parent when in contact. From all I have learned of the disease, I can find no proof of even the hereditary predisposition allowed to exist by Virchow, but feel much inclined to believe with Landré, that contagion is the only cause of its propagation. Even one well-authenticated case of a son or daughter of a leper removed in infancy to a country where the disease is unknown, as from the West Indies to England, and becoming a leper twenty or thirty years afterwards, would do more to establish the possibility of hereditary transmission than hundreds of cases of persons who have been exposed to the possibility of contagion either from their parents themselves or others. Such proof might also be sought in localities where it is dying out, where the *last* person affected was the grandchild of a leper, who had never been in contact with his ancestor; but until such proof is adduced, the disease cannot be looked on as hereditary.[3]

[1] Virchow's *Archiv*, 1853, b. v. p. 13; and Landré, p. 28.

[2] Landré, p. 29.

[3] Such proof is *not* afforded by the case of Berns, the last Shetland leper (Simpson), as it is only stated that the disease was in his family, not if in parents or grandparents, nor if he was ever in contact with such relatives.

Equally unsatisfactory is Boeck's proof (!), which he considers so strong that "natural science surely requires nothing further" (Carter, 1874, Rep. p. 48), from nine Norwegians in America. Of eighteen cases seen, nine left Norway lepers, the *other nine* were attacked two to fourteen years after emigrating; of the first nine, four had leper relatives, five had not; all the second nine had. Now this proof is utterly defective, firstly, because whatever Boeck may believe, the incubative period in cold climates, especially with a good diet, is indefinitely prolonged, possibly beyond eight years, as in Dan. and Boeck's case (p. 339); so that, while those attacked two years after arrival almost *must* have, the one or two attacked fourteen years after, *might* have brought it from Norway; secondly, because only relatives, not leper parents, are mentioned in connexion with the second nine, and we have seen that cousins, etc., having the disease proves nothing; thirdly, because nothing is stated as to the last cases attacked being related to or in contact with the others—a communication almost, if not quite certain to happen among foreigners in a strange land whose community of language would drive them together. It is a pity these points were not inquired into, as without them we must reject the so-called proof, and believe that some of the nine brought the disease with them from Norway; the others either did so or were infected in America.

I do not deny that leprosy may be occasionally hereditary, but only say that it has never been proved to be so.

Contagion.—As the whole of the foregoing part of this work has been a series of proofs of the contagiousness of the disease, I have little more to say in this section than to try to meet a number of objections that have been raised against this theory, and quote cases. I must premise, however, that by using the word "contagion" I do not pretend to express any distinct belief as to the probability of the disease being conveyed by simple contact, being more inclined to believe that it is carried by inoculation in most cases, though long-continued contact even of unbroken healthy with diseased skin may be sufficient.[1]

Authors opposing the contagious theory more or less may be divided into two classes: the first entirely denying that it is contagious or communicable, as Danielssen and Boeck, Virchow, the Committee of the Royal College of Physicians, Wortabet and Pruner; the others admitting that it may be contagious, but holding that contagion plays a very insignificant part in its propagation, as Planck. Carter, as I have already pointed out, formerly held that it was not contagious, but *perhaps* inoculable,[2] but now seems more inclined to admit that it is more communicable than he was then led to believe, in so far as it is less hereditary.[3]

The chief arguments against contagion have been, 1st, That many married couples live together for years, one being diseased, without the other becoming affected;[4] 2d, Hospital dressers, hospital physicians, and in former times queens, who sometimes washed the sores of lepers,[5] are said not to be, or have been, attacked. 3d, That even inoculation of the leprous matter has failed to reproduce it,[6] and that medical men engaged making post-mortems of lepers, and having their hands bathed with the fluids of such bodies, are not infected.[7] 4th, Many are exposed to contact with those suffering from the disease, while but few are attacked.[8] 5th, It has never spread in England, or other countries now clear of it, from imported cases. We will consider these objections *seriatim*.

In regard to married couples, what I have already stated in regard to the decrease in or loss of procreative power in the males should be kept in mind, as this reduces the risk to a wife from

[1] It may be worth raising the question, whether in hot countries where the pores of the skin are constantly open, there is not more liability to communication by simple contact. Such a series of cases as are mentioned by Landré (p. 51) of five persons in Surinam, infected one after another, suggest this idea.

[2] *Op. cit.* (1862), p. 29.

[3] Reports, 1876, pp. 20 and 21.

[4] Coll. Phys. Rep., lxix. Milroy, p. 5. Kaposi (Hebra), p. 185. Wortabet, *op. cit.*, p. 192. (His one quoted case, in which the husband, a Jew leper, had been four years married to his wife, proves nothing, the time being too short.)

[5] Simpson, *op. cit.*, p. 412.

[6] Coll. Phys. Rep., pp. 13, 14, and xliv.

[7] Bakewell, Vacc. Rep.

[8] Simpson, *op. cit.*, 400, and Virchow, *lib. cit.*, p. 505.

cohabitation to exactly the same as that of any other person continually in contact with a patient. Again, although at no age is there immunity from attacks, yet the tendency to the disease certainly seems to be greatest within the first thirty years, as in 47 cases of Wortabet's,[1] 36 were attacked before thirty years of age, and only 4 after forty. Similar results appear in Danielssen and Boeck's tables,[2] which show that in altogether (in Norway and Southern Europe) 272 cases, 134 were attacked before twenty, and 202 before thirty years of age. In 72 cases in St Kitts, I found that in 36 cases of joint evil, the mean age of attack was twenty-four, the earliest six, and the latest fifty. In 36 tuberculated cases the mean age was sixteen years, the earliest three years, and the latest fifty. The numbers attacked at or under the tenth, twentieth, etc., years were as follows :—

	Years 10	Years 20	Years 30	Years 40	Years 50
Tuberculated .	7	18	8	1	2
Non-Tuberculated .	9	15	4	2	6
Totals of both kinds .	16	33	12	3	8
		61		11	

Thus 49 of the 72 cases were attacked between birth and their twentieth year, and 61 before they had completed their thirtieth year, leaving only 11 attacked after that age. I compared those numbers with the numbers of the population under twenty and thirty years, and I found that 48 per cent. of the population were under twenty years, and 68 per cent. under thirty, so that practically one-half of the population (those under twenty years) furnished two-thirds of the cases, and two-thirds of the population (those under thirty years) furnished six-sevenths.

The greatest tendency to the disease appears to exist from the tenth to the twentieth year, which furnished 33 cases, or 47 per cent. of all the cases, from 22 per cent. of the population, while 26 per cent. of the population (the number living under ten years of age) did not furnish half that number, and 20 per cent. (the number living between twenty and thirty) only furnished 12 cases. After the thirtieth year is passed the tendency appears to become almost *nil*, as of the 3 cases in the table, one was attacked at

[1] *Op. cit.*, p. 188.

[2] *Lib. cit.*, p. 330. Tuberculated cases in Norway, 188, of which 136 were attacked before thirty (and other 32 before forty). Non-tuberculated 65, of whom 53 were attacked before thirty. In South of Europe 19 cases, all tuberculated, 13 being attacked before the thirtieth year. There is some appearance of a tendency to earlier attack in tropical climates.

thirty-one years, and the others at thirty-four and thirty-six years respectively, between which and forty-eight years only *one* was attacked (at forty-four years). There seems about the forty-eighth year to be a slight increase in the tendency, especially to joint evil, as all the 6 cases of that kind, and one of the tuberculated, that appear between the fortieth and fiftieth year, were attacked between the forty-eighth and fiftieth year.

The bearing of this on the escape of women married to lepers is obvious; the earliest age at which any married man was attacked in St Kitts was twenty-eight years; it was some time thereafter before he became a confirmed leper, and meantime his wife, who was about the same age with himself, was rapidly passing beyond the age of susceptibility. The wives of those attacked later in life would have still less chance of being affected.

Thus, leprosy attacking a male prevents marriage, and when it occurs after marriage, by the time the man is in a state to communicate the disease his wife has from her age become in most cases insusceptible.

In spite of these facts, which tend to keep down the numbers, however, the fact that wives have, in a number of instances, been attacked after husbands, and that where inquiries have not been carried far enough to decide which was attacked first, married couples have been noted as both being lepers, renders the argument against contagion worthless. Tilbury Fox,[1] Planck,[2] E. Wilson,[3] Van Holst[4] of Dutch Guiana, Manget of Demerara,[5] and Nicolson of Antigua,[6] each quote a case of an European infected, by, or at least after, cohabitation with a leper woman. Kaposi[7] mentions a case of an Italian affected at Cairo, whose wife was attacked two years later. Proto Medico[8] (Corfu) and Regnaud[9] (Mauritius) mention three cases of wives affected from husbands. Dr Carney[10] (Guiana) says—"A woman had connexion with an old leprous African; she afterwards became leprous. Carter[11] gives similar cases to Regnaud's, and mentions a case of a husband affected after a wife, and two of wives after their husbands. In one of the last cases *her* son was also attacked. Macnamara[12] gives four cases (from the Indian Report on Leprosy) of wives after husbands and one of a husband after a wife. Those so attacked belonged to healthy families. Besides

[1] *Edinburgh Medical Journal*, March 1866, p. 802.
[2] *British Medical Journal*, vol. i. 1877, p. 434.
[3] Coll. Phys. Rep., p. 431. (This *may* be the same case as Fox mentions.)
[4] *Ib.*, p. xliv.
[5] Milroy, Rep., p. 10, and Coll. Phys. Rep., p. 45. Her child by him afterwards became affected.
[6] Coll. Phys. Rep., p. 20.
[7] Hebra, vol. iv. p. 184. [8] Coll. Phys. Rep., p. 44. [9] *Ibid.*, p. 86.
[10] Quoted in *Lancet*, 1867, vol. i. p. 253.
[11] *Trans. Med. Soc. of Bombay*, 1862, p. 30.
[12] *Op. cit.*, pp. 22–24.

these, Pruner[1] and Shier[2] and Brunelli[3] (Crete) speak of leprous couples without giving particulars, and Schillingius[4] distinctly says—"I could point out many examples to the contrary by name both of husbands and wives who have contracted the disease during marriage, did shame permit." In St Kitts, Hannah Carty, æt. twenty-seven years, lived, slept with, and washed the clothes of, T. Wilson, when a young girl, he being covered with sores. She was attacked at seventeen years of age. Her family are all healthy. Whether it is possible for the disease to be transmitted by sexual intercourse without inoculation is still quite undecided. I am inclined to think not. It must be clearly remembered that inoculation may take place during connexion if there is the slightest ulceration of the cervix uteri. E. Wilson's case I have just quoted, in which syphilis was conveyed at the same time as leprosy, is one of the few cases tending to show that the latter can be conveyed by cohabitation.

In the face of the cases I have quoted I cannot but think that the statement that wives or husbands are never affected from one another, or, at least, after one another, is incorrect, and any argument founded on it falls to the ground.

The second argument, viz., that hospital-dressers, surgeons, and others attending lepers are never attacked, were it even true, is of no value, as the same might be said of such persons in Lock Hospitals, yet no one denies that syphilis is communicable. But the case of, at least, one medical attendant, Dr Robertson of the Ile Curieuse Asylum, Seychelles,[5] and those of several hospital-dressers,[6] some of whom, at least, were of clean families, are on record. Similar to these, though not occurring in leper asylums, were the cases of Drs Livingstone and Kirk, threatened with the disease after attending a leper,[7] and such cases as that of a Brahmin servant of healthy family, who was attacked after twelve years' attendance on a leper master,[8] having had to *wash and dress his sores*. Carter mentions two cases occurring in the children of a sepoy in charge of the Dhurumsala hospital, who with his wife was healthy.[9]

In the case of queens who washed the sores of lepers as an exercise of piety, the contact was too occasional for any conclusion to be drawn from these particular persons not becoming affected afterwards.[10]

The third argument, that even inoculation may be practised in

[1] *Op. cit.*, p. 173 [2] Milroy's Rep., p. 5. [3] Coll. Phys. Rep., p. 64.
[4] *Op. cit.*, xxxvi. p. 34.
[5] *Lancet*, 23d February 1867 (quoted from The Indian Report).
[6] Hillebrand in Macnamara. *Op. cit.*, p. 57. Three cases—two in Calcutta, one in Java. Two cases reported as having occurred in the Almorah Asylum (Coll. Phys. Rep., p. 141) are authoritatively contradicted in Lewis and Cunninghame's Rep., p. 58.
[7] Zambesi, p. 225.
[8] Coll. Phys. Rep., p. 141.
[9] *Op. cit.*, 1862, p. 30.
[10] The fact that *an ox* in a leper asylum in the Mauritius (Coll. Phys. Rep. p. 88) died of the disease also tends to refute this argument.

some cases with impunity,[1] and that those making post-mortem examinations of lepers, as Dr Bakewall remarks, may have their hands bathed in the secretions without being affected, is worthless, when it is considered, firstly, that in the very few cases in which inoculation was practised the systems of those undergoing the dangerous experiments were not in the least likely to have had any tendency to be affected, they being in good health and well fed; and in regard to post-mortems, the danger of dissection wounds is well known, yet were every student who cuts his finger while dissecting, or has his hands bathed in the undoubtedly poisonous secretions of dead bodies, to die, not one in ten would ever pass through their curriculum alive.[2] The case of Dr Livingstone, who was attacked *after suffering privation*, having scratches on his hands; one mentioned by Larrey,[3] in which the disease *began in the wound on a stump;* and that of Hillebrand,[4] in which a European child in Borneo was affected after thrusting a thorn into himself after a leper boy had in his presence done so,—all tend to show the erroneousness of such an argument, and that inoculation is the chief, if not the only, manner by which the disease is propagated, such propagation only taking place quickly when some special circumstance, as the person being wounded, makes inoculation easy and certain, while more prolonged intercourse is generally necessary to afford opportunities for inoculation in ordinary circumstances. It is possible at the same time, however, that in tropical climates, where the pores of the skin are constantly open, a kind of inoculation through the skin, so to speak, may by prolonged and repeated applications of the diseased discharges take place, even without any scratch or wound existing on the person of the person so infected.

Fourthly, No doubt, of many exposed to the disease only a certain number take it, but exactly the same may be said of every contagious disease; even the most violently *infectious* diseases, poisoning every fluid surrounding the patient, never attack *all* exposed to their influence — how much less can leprosy, which requires, it appears, either direct inoculation or very prolonged contact, to be repeated constantly, to attack all brought in any way into communication with the diseased person. That it does sometimes, however, attack a number of persons from one source is proved by the series of cases mentioned by Macnamara and Landré, and those already spoken of by myself.

Fifthly, As to its *never* spreading when imported, I have already shown that it is not likely to do so in a well-fed population; but Dr Owen Ree's case of the Irishwoman in Stepney, and Gaskoin's case from Guernsey, which I have already remarked upon, must be

[1] Coll. Phys. Rep., xliv.

[2] Paget, *Lancet*, 3d June 1871, in a lecture on "Dissection Poisons," points out that immunity may be obtained by custom, just as the system may get accustomed to strong drink or arsenic. The question is worthy of consideration, whether such immunity may not apply to the effects of the inoculation of leprous matter. [3] *Op. cit.*, p. 225. [4] Macnamara, *op. cit*, p. 57.

accounted for otherwise than by contagion brought from abroad before the assertion can be accepted as correct, or any argument founded on it be accepted as of value.

Finally, Can it be truly said that the instances of supposed contagion are so few and imperfectly related as not to assist in proving that the disease is contagious?[1] I think not, keeping in mind, as I have already said, the long period of incubation, the ignorance of the greater mass of the populations of the countries where the disease is prevalent, causing great difficulty in tracing long-forgotten or wilfully-hidden chances of receiving the contagion; looking, too, at the extreme difficulty in satisfactorily proving the communicability of some diseases with even a very short incubative stage, I think many of the cases on record are, especially when the proof they afford is taken in conjunction with the history of the disease, quite conclusive as to its contagious nature, it being always to be kept in mind that one *positive* case overweighs fifty negative ones.

Larrey[2] gives instances, one of which has been already mentioned, among French soldiers, who, suffering from privation and being *wounded*, were specially liable to inoculation. I have already spoken of wives taking it from husbands, and in treating of heredity given series of cases of my own, with those mentioned by Macnamara and Landré and Max, which could best be explained by the theory of contagion. Max[3] mentions one case of a widow, aged 58 years, with seven children, who went to live with a daughter, a leper, and was attacked five years later when 63 years of age; in another case,[4] a slave woman attended *her master's father, a leper*, and was attacked after his death. Besides these already quoted, Landré gives ten cases of contagion among well-to-do Europeans or their children, all of whom are stated anterior to their attack to have continuously or repeatedly been in contact with lepers.[5] Heredity could have nothing to do with such cases. He also mentions (p. 58) the case of a mulatto woman of clean family, who being in constant contact with two leper relatives of her husband, became affected. Macnamara,[6] quotes seventeen cases from the Indian Report. The following remarkable series is among them,—1st, A woman; 2d, In five years, her daughter living with her; 3d, The woman's husband, in four years more; 4th, Her husband's brother's wife *living in the same house*, not a blood relation; 5th and last, in about two years after, the husband's brother. Quoting Dr Rose, he mentions the case of M.

[1] *Vide* Coll. Phys. Rep., p. lxix.
[2] *Op. cit.*, p. 225.
[3] *Gazette Med. de Paris*, 14 Juilliet, 1877, cas. xiii.
[4] Cas. viii.
[5] I am astonished at the reckless injustice of Liveing's criticism on these cases (p. 92), viz., that they would show that the disease is "highly infectious," and therefore "prove too much if they prove anything," in the face of Landré's distinct assertion that they were "*continuellement* en contact," and it requires "contact très intime," p. 79. No such conclusion can be drawn from his cases, nor does he desire that it should be.
[6] *Op. cit.*, pp. 21 to 24.

Sneider, who lived with his uncle, M. De Souza, while the latter was a leper, and was attacked with the disease before ulceration and profuse discharge set in in his uncle's case. M. Sneider was Dr Rose's apothecary, and had a hospital orderly under him, who "contracted leprosy from him, and died in less than twelve months from the time the disease first became manifest." All the other cases, which I have no space to quote more fully here, show that prolonged contact with a leper, especially after ulceration has set in, is fraught with danger to the healthy.

Manget [1] mentions a case in a white man, contracted by sleeping with a Maltese leper and smoking the same pipe. Danielssen and Boeck mention four similar cases in Europe,[2] and I have already spoken of two I saw in St Kitts, so caused, where there was no relationship between the person affected and the one who took it. Manget also mentions the cases of three black children who were all affected after playing constantly with a coloured child not related to them, who was in their mother's charge. In the College of Physicians' Report, also, there are a number of other cases given besides those already quoted, the details of some of which are no doubt too meagre to afford individually conclusive proof of contagion, as, for instance, that of Dr Duffey's (p. 45), viz., "a healthy girl, æt. 7, slept in the same bed with a boy, æt. 9, who was diseased; she became affected with leprosy." To those holding that heredity is an important factor in the etiology of leprosy this girl *might* have been one of a leprous family, or she *might* have got the disease without sleeping with the boy, but looking on heredity as of no value as a factor, seeing that Europeans sleeping with lepers became affected, thus excluding heredity, I must say that I consider that such cases were too hastily put aside by the framers of the Report, and too lightly valued, when the number of them given is taken into consideration. Taken together with the other proofs I have given in this work, they afford indubitable proof that leprosy is a communicable disease. Dr Pollard [3] mentions that the *whole* of the children of a distinguished family in Guiana were attacked after playing with a leprous negro boy. The late F. Wigley, Esq., President of St Kitts, related a case to me of a white gentleman who was attacked after a leprous servant had surreptitiously made use of some of his master's clothes to dances, at which of course he would sweat very much. In regard to the remainder of the case in the College of Physicians' Report, I need only notice them shortly. At p. 202 is a case of a master affected after a servant "who was constantly about his person," similar to the one I have

[1] Milroy's Rep., p. 10; and Coll. Phys. Rep, p 45.

[2] *Op. cit.*, p. 440, Case 25; and 481, Case 13 (raised by a leper), Norwegian; and Case 1 at Provence, and Case 7 at Rhodes (p. 520, *et. seq.*) Cases 13 and 7 were the only ones in the family. They all considered that they got the disease by sleeping with lepers.

[3] *Lancet*, 23d Feb. 1867.

just related; p. 36, "a young girl" slept with a young woman,[3] seven or eight years later she was a confirmed leper, subsequently her mother took it; p. 32, two cases of young men by proximity, or direct contact (details not given); p. 86, a stepson of healthy family from a leprous stepfather (Regnaud); p. 198, W. E., European boy in Sarawak, after playing constantly four years previously with a Chinese leper boy, all W. E.'s family were healthy; and lastly, at p. 239, a somewhat unsatisfactory case of an English colonel who believed that he got the disease "from sleeping in an unclean bed in a negro's hut." I only mention this case because the circumstances are similar to those mentioned by Larrey in regard to one of his cases in a French officer.

Thus it will be seen cases are not wanting to add to the other proofs that the disease is communicable. I must say, also, that I do not think that the universally received opinion which has obtained for ages[1] in countries where the disease has existed so long, and been held by all medical authorities up to the time of Schillingius and Hillary, such authors being close observers of natural phenomena, though they were not, perhaps, so much given to collecting cases and giving details as those of the present day—I think an opinion so supported should have been treated with more respect, and a contrary one expressed with more caution, than was done in the College of Physicians' Report.

To sum up the whole of the proofs of communicability I have given in this work:—

1. It has always spread from race to race wherever an infected race was brought into contact, under favourable conditions, with a non-infected one.

2. It has been and is most prevalent amongst those races and nations among whom the freest communication with lepers is allowed by public opinion and law.[2]

3. The so-called proofs of heredity commonly advanced being utterly defective, most, if not all of the cases accepted by some authors as hereditary are best accounted for by communicability.

4. The cases on record of probably communicated leprosy strongly support this view, and, taken with the other proofs, show that the disease is undoubtedly communicable, probably only by long continual contact or inoculation,[3] but possibly through drinking water.

[1] See *Wise*, p. 159.

[2] Daniellssen and Boeck's words in regard to Norway are worthy of quotation on this point:—"A la même époque où la Spedalskhed par les mesures énergiques opposées à sa marche est devenue plus rare dans toute l'Europe, dans notre pays elle n'a pas été combattue avec tant de fermeté que dans les autres pays, et par cette raison elle y apparait encore à une degré inquiétant."

[3] The fact lately observed, that mosquitoes can imbibe the *filaria sanguinolenta* with the blood, suggests the possibility of some cases of leprosy being communicated by means of these insects.

Leprosy.[1]

Having thus concluded the treatise on the etiology of leprosy (so fulfilling one chief purpose of this work), I now propose to treat very shortly of the following, viz.:—Symptoms, diagnosis, age of attack, duration and sex, prognosis and treatment. Under the latter I will endeavour, under the heading of treatment of infected populations, to point out the practical means of preventing the disease as indicated by my ideas of its etiology, this being the chief object of the work.

Symptoms.—Tuberculated leprosy. This is sometimes preceded by an eruption of dark blotches on the skin of the body, face, and extensor surfaces of the limbs, and sometimes by maculæ similar to that which precedes non-tuberculated leprosy. These are followed by tubercles which appear on the face, on the cheeks, eyebrows, and lobes of the ears. These are formed by infiltrations of the subcutaneous tissue, and are hard, raised lumps. Meanwhile the hands and feet swell, the skin of the whole body becomes bronzed in colour in the white, and anæsthetic, the eyebrows drop off, the cornea becomes inflamed, and the voice raucous; later on ulceration of the tubercles takes place, and dysentery often ends a life of misery.

Acute Leprosy.—I have seen one case of this, the patient being attacked suddenly with sharp fever, and the lumps on the face and swelling of the hands appearing in a fortnight or so. She had been two months ill when I saw her, but had all the appearance of having suffered from the disease for several years.

Non-Tuberculated Leprosy.—Maculæ,[2] at first red, tending to spread, becoming pale, glistening, without scales, and depressed

[1] As Dr Bristowe has now supplied the want I formerly referred to by describing leprosy in his "Text-Book of Medicine," and a description of the disease is given in the New Sydenham Society's Translation of Hebra, vol. iv. (in which, however, I think, erroneously, morphœa is described as related to leprosy), I beg to refer for fuller details of symptoms to those easily accessible works, though I think I said enough to enable any one to diagnose the disease even if, as might be the case, he could not obtain these references. Carter and Liveing's works, and the Coll. Phys. Rep. (p. xvi., etc.) may also be referred to. I may say that I have given the ordinary division into tuberculated and non-tuberculated, but others may be adopted, as that of Macrae (*Med. Times*, vol. ii. 1875, p. 103), into tubercular, anæsthetic, mixed, and atrophic. The first and last are the common forms in the West, the anæsthetic commonest in the East Indies. Labonté (*Ed. Med. Jour.*, Nov. 1878) gives an excellent description of atrophic leprosy. He, I may say, thinks the disease hereditary, but to my mind the cases he publishes do not in the least support this view.

[2] The macular leprosy (lepra leprosa, Carter) may, I think, be looked on as simply lepra vulgaris, having the leprous poison as its exciting cause, while the analgesia of the centre is not, so to speak, properly to be looked on as a symptom of the local skin disease, but rather of the leprous disease itself. The absence of scales in no way affects the opinion, as Cazenave and Schedel describe lepra vulgaris without scales. In a country where leprosy is common,

and anæsthetic in the centre, sometimes appearing and disappearing several times, with months intervening, then accompanied by pemphigus on the palmar aspect of the fingers and toes, then contraction of the fingers and toes, and wasting of the palmar muscles, followed by a dry caries of the bones, and gradual disappearance of the whole digit, leaving the nails in some instances on the knuckles, are all seen as symptoms of this kind of leprosy. At the same time the skin of the body becomes dry and harsh, and sometimes analgesic and anæsthetic. Paralysis of the orbicularis is a marked symptom, allowing the lower eyelid to become everted, giving the face a hideous appearance. The skin in the white race becomes of a dirty pale colour. The nose in a few cases falls in.

In both varieties the lymphatics are affected, being swollen, and often painfully.

Diagnosis.—The non-tuberculated variety can hardly be confused with anything else, but I have seen a case of syphilis very like at first sight to tuberculated leprosy, but the tubercles on the face were rather rounder, and though he had been ill many years, there was no analgesia. It has been confused even lately with elephantiasis Arabum, but the latter is a strictly local disease (being simply tropical erysipelas, with a tendency to recur, and leaving a deposit in the subcutaneous tissue after each attack). It only attacks the lower limbs or scrotum, and could never be mistaken for elephantiasis Græcorum by any one really acquainted with the two diseases. I have seen one instance of non-tuberculated leprosy, in which elephantiasis Arabum (Barbadoes leg) coexisted.[1]

Leucoderma is distinguished by the paper-like whiteness of the affected skin, and absence of analgesia.

Age, Duration, and Sex.—I have already mentioned the ordinary age of attack. The age at death I found in St Kitts to be, taking the average of the ages of 62 lepers who died between 1859 and 1872, 32·2 years.[2] This includes both kinds. As the average

lepra will generally indicate the commencement of leprosy, especially in the East, where non-tuberculated is the most common variety. As Carter points out, hence probably has arisen some of the confusion between the two diseases; but this does not in the least support the idea that there is any necessary connexion between them, or that the lepra vulgaris of Europe is a remnant of real leprosy. Other skin diseases may precede or accompany leprosy. The eruption of the maculæ is sometimes accompanied by sharp fever.

[1] Anomalous cases sometimes occur in Europe, which have been looked on even by men of the highest attainments as leprous, but which, from their want of symmetry, of analgesia, and of constitutional symptoms, cannot, I think, be properly considered as such. They are liker elephantiasis Arabum, or some obscure lymphatic disease, or of tropic nerve disease, with consequent gangrene. (See, besides cases already quoted, Bell, in *Lancet*, vol. ii. 1875, p. 420, and compare them with a case of elephantiasis in France in the *Abeille Médicale*, Oct. 1878, and one of spontaneous hemianæsthesia and gangrene in *Le Courrier Médical*, 10th Aug. 1878.)

[2] The average age of forty-two who died between 1817 and 1825 was 29·7 years, being, like the ages of the population, generally shorter than at present. The average age of the whole 105 would be thirty years.

of attack was, I found, twenty years (sixteen in tuberculated, and twenty-four in non-tuberculated), the average duration would be fully twelve years. The oldest age at death was seventy years (doubtless joint evil), the youngest six and a half.

It has been stated by Planck that lepers live as long as the rest of the population. Against this I may mention that, comparing the number of deaths and of lepers in the slave registers in 1817–26, and deaths in the registration books in 1859 to 1870, with the number of lepers living in 1854 and 1872, and correcting the results obtained by comparing the total number of deaths among lepers receiving hospital relief from 1867 to 1872 with the number of these lepers, I found that the average rate of mortality among lepers was 7½ per cent. yearly, being two and a half times that of the population generally (3 per cent.), and four times that of the population over five years of age.[1] Thus leprosy undoubtedly, as might be expected, shortens life.

I had no means of distinguishing in these registers the kind of leprosy, but the age of the living lepers gives an idea of the different mortality of the two kinds, viz. :—

Tuberculated, 34 cases.	Non-tuberculated, 37 cases.
Average age, 22¾ years,	39 years.
Oldest, 50 years,	65 years.
Youngest, 6 years,	14 years.

Those figures agree, as far as can be expected, with those of Daniellssen and Boeck,[2] who give the average duration at death of tuberculated cases as 9½ years, and of non-tuberculated as 18½ years, and with Carter's,[3] who gives 9 to 12, and 16 to 20 years, as the ordinary time. Wortabet[4] found only 2 in 47 who had been affected more than fifteen years.

Sex.—In St Kitts, from my inquiries already referred to, I found that in 1817, of 94 lepers, 60 were females, 34 males. From 1817 to 1827 the slave register shows the deaths of 26 females and 16 males. From March 1858 to September 1870, 32 males and 24 females died;[5] and from March 1868 to March 1872, 12 males and 10 females receiving relief died.[6] Together these give 44 males and 34 females. In April 1872 the 72 lepers I found in the island were 33 males and 39 females. Thus, in slave times more females than males were affected; at the later date the numbers are more equalized, possibly because, as slaves, lepers were

[1] The expectation of life at five years in St Kitts, as I calculated from the registrar's returns for six years, 1864–70, was 52·2 years, giving a mortality in the population over that age (before which very few are attacked with leprosy) of under 2 per cent. The other figures are from calculations made by me after going over every entry in the books during the periods stated.

[2] *Op. cit.*, p. 352. [3] Rep. 1874, p. 8. [4] *Op. cit.*, p. 188.

[5] Registrar's book: 4 were white, 11 coloured, 40 black. A large proportion of white, 1 in 11, three times the proportion of white to coloured and black in the population. [6] Hospital book.

put apart by their owners to the sea-shore or mountains, and only the *women* of their family would be allowed to attend to them, rendering the latter more obnoxious to the disease.

Of Wortabet's cases,[1] 19 were males and 16 females; and of Daniellssen and Boeck's in St George's Hospital, 74 males and 77 females. Thus, the evidence from Spain, Norway, and St Kitts, though the numbers are comparatively small, show that the great overplus of males in the census of India, if it even shows (as I do not think it does) the *real* proportion of the two sexes affected, is not caused by any special liability of the male sex as such to leprosy, but depends on the circumstances of life under which the population lives. As is seen from the figures I have given in regard to St Kitts, the proportion may vary at different times, as the conditions of life change.

Pathology.[2]—This subject I have mentioned incidentally in speaking of etiology. I have only to add here, that the exudation and deposit of albuminoid matter in the subcutaneous tissue in tuberculated, and in the interfibrillar spaces in non-tuberculated leprosy, consist of small, nucleated, round cells, crowded together (Carter), and of spindle-shaped elements (Virchow). The pressure of these destroys the surrounding tissues. Daniellssen and Boeck describe a change in the albuminous materials of the blood, which precedes, according to them, this deposit. The thickening of the skin and ulceration in the one kind, and the pemphigus and caries and destruction of the extremities in the other, are all caused by the primary changes.[3]

The mucous membrane of the larynx is generally the seat of deposit in tuberculated cases. The lungs are seldom attacked.[4] Changes in the other viscera are too inconsistent to be looked on as pathognomonic.

Prognosis.—This is, in all cases, bad. I have, however, seen one case of joint evil in St Kitts, who, as far as the mere arrest of the disease, after it had deprived the patient of all her fingers, was concerned, might be called cured.[5] She was a leper in 1817, and was nearly 70 years old when I saw her in 1872. She was then in fair health.

I also saw in Edinburgh, in 1874, at the Medico-Chirurgical Society, a case of tubercular leprosy,[6] which might be looked on as cured, the tubercules having disappeared and left the face dusky (he was a white man), scarred, and wrinkled, but very probably the disease would reappear. Daniellssen and Boeck figure in their atlas one case of spontaneous cure of tuberculated leprosy. Carter

[1] *Op cit.*, p. 187. [2] P. 334.

[3] For full pathological details, I beg to refer to the works, already quoted, of Carter and Hebra, also Dan. and Boeck, p. 216 *et seq.*

[4] This is in Norway, but, as Sweeting (*Medical Times*, vol. ii. 1860, p. 208) says, phthisis is a common cause of death in the West Indies.

[5] Landré (p. 12) gives a similar case, and Macrae (*Medical Times*, vol. ii. 1875, p 118) another. [6] The case is described by Liveing, p. 126.

(Rep. 1876) speaks of the tendency in mild cases to spontaneous cure.

Treatment.—This must be spoken of under two heads,—1st, that of the individual; 2d, that of the population. Since the kings of Egypt are said to have bathed in the blood of slaves, and the Hindoos used cow's urine as a medicine;[1] and eight centuries back, when Psellos recommended the emerald mixed with water, the certain cures, simple and compound, recommended for the disease, would, if merely named, fill a volume. Passing over all others, and simply mentioning arsenic as having been sometimes useful, I will merely consider very shortly Beauperthuy's treatment, and those by chaulmoogra and gurjon oil. Beauperthuy's treatment was almost entirely local, and consisted chiefly in the destruction of the tubercules by castor-nut oil and other irritants. The treatment was severe, and a number of cases relapsed soon after, so that it may now be said to be out of date.

Chaulmoogra Oil is said by Carter[2] to be of decided utility; under its influence a retrograde metamorphosis of the tuberculous matter is encouraged to take place, "the nodules in the skin do subside, and the sensory nerves more or less regain their function." But it does not prevent the exacerbation of symptoms which comes on suddenly at intervals. Thus, it appears to act by interfering with the effects of the leprous poisoning of the system, rather than by any specific action on the poison or poisonous matter itself.

I may say that in Carter's cases good diet and hygiene were combined with the use of chaulmoogra oil.

Gurjon Oil.—This, in the hands of Dr Dougall, of the Andaman Islands convict establishment, seems, although it may, as Carter says, not be a specific, to have produced better results than any drug yet known, restoring to comparative health those who have suffered for a long time from the disease, even becoming fit for active employment, though they had long been useless. It is best used externally as a mixture of three of lime-water to one of the oil; internally, half an ounce of the same mixture twice a day is given. It should be rubbed in twice a day for about two hours, after the body has been thoroughly washed. The oil acts as a laxative and diuretic. Ordinary diet is given. The oil is not a caustic.[3] Thus both chaulmoogra and gurjon oil may be looked on as on their

[1] Wise, pp. 117 and 263, and asses' urine (Ætius), see C. Wilson, 1st March 1876. This, though disgusting, would supply the *want of salt* in the food, which I have pointed out as a probable primary cause of the disease.

[2] Rep. 1876, p. 33. For fuller information, see Macnamara, *op. cit.*, p. 45, and *Med. Record*, vol. ii. 1867; Mouat, *Med. Rec.*, vol. i. 1856, p. 239; and Hobson, *Med. Times and Gaz.*, vol. i. 1860, p. 559.

[3] Should any reader, wherever situated, wish to carry out this treatment, he may find the following full references useful:—Dougall, "Gurjon Oil," *Edin. Med. Jour.*, vol. i. 1877, p. 845; *Indian Med. Gaz.*, 1874; *The Doctor*, vol. ii. 1874, p. 157; *Brit. Med. Jour.*, vol. i. 1875, p. 178; E. Wilson, in *Lancet*, 16th May 1874; Carter, Reports, 1876, second series, p. 36; and also, Dougall, in *Med. Times*, vol. i. 1874, p. 683, and vol. ii. p. 586; and Macrae, in vol. ii. 1875, pp. 103 and 118.

trial, which will take some years to give reliable results. With any medicine or without it, good diet and cleanliness have always been found useful.

Change of climate even from one infected country to another, as in one case of Landré's from Portugal to Algiers, is always beneficial, and may arrest the disease indefinitely.

Treatment of the Population.—This, the last division of this work, and, so far as the objects for which it was written are concerned, the most important, as being that to which all I have said in the part on Etiology leads up, I will treat of as shortly as possible, though more fully than the mere sketch which circumstances indicated I should give of the symptoms, diagnosis, etc.

The treatment of any population among whom leprosy is common should be carried out with two objects, the first being to prevent new cases arising from any known or suspected causes of origin; the second, to prevent its spread from already existing cases by contagion or like means.

If I am correct in my idea, that want of salt, combined with a vegetable diet in insufficient quantity, is a primary cause, or the primary cause of leprosy, then everything that can remove such conditions of life should be encouraged. In India this might be earnestly urged on the Government as one out of the many reasons for the remission of the salt tax. Although two millions might be a heavy loss to the revenue (that being the amount raised by it), its remission would be a great gain in the end, as tending to the good of the people; the consequent cheapening of salt would be a blessing to millions, who might then be able to procure a sufficient supply to keep them in health. How heavy this tax is may be roughly estimated, when we consider that one ounce is about the yearly supply for many millions of the poor cultivators, and that the population of British India is roughly 200 millions; so that each 100 persons pay 20s. of salt-tax yearly, or about 2½d. each person; thus, each ounce (so far as *those* consumers are concerned) is taxed to something like fifty times its value. Surely a free breakfast table is yet far off in India when an absolute necessary of life is thus taxed.

To further cheapen food and improve the state of the population, railroads and good roads are the greatest want, the difficulty of carriage making many articles dear in some localities and cheap in others. More perfect irrigation of the country, and great care to prevent the destruction of forests by the Bygás and such other aboriginal tribes,[1] which renders the climate of the surrounding districts too dry, would all be beneficial.

The spread of Christianity, and consequent doing away with the Brahminical prohibition of the use of flesh food, will assist. The use of flesh meat in moderation ought to be encouraged for many other reasons, as leaving the people less liable to death by famine

[1] See Forsyth, "The Highlands of Central India," p. 364.

in times of bad crops, and making them more able to bear such misfortunes by giving them more stamina.

To prevent the spread of the disease when it has arisen, there can be no question that segregation is the best and surest means, whatever be the theory of its spread that we accept, but especially if it is really communicable, and whether it is actually contagious, in the ordinary sense of the term, or simply inoculable, or may be conveyed through water or food.

In St Kitts the great decrease in the number of lepers, both absolute and relative to population, from 95 (in 20,149) to over 53 (in 20,700), that took place between 1817 and 1854 (a period commencing only ten years after the abolition of the slave trade, a traffic which constantly imported new lepers), and during the first two decades of which segregation was strictly enforced by the slave-owners,[1] this great decrease, as compared with the slight relative decrease (if any, but certainly not an increase) from over 53 (in20,700) to 72 (in 28,000) in 1872, speaks strongly for the value of segregation.

Landré[2] points out forcibly that under Dutch rule Surinam had far fewer lepers than after it was taken by the English in 1799, and restored to the Dutch in 1816, the Dutch laws having been exceedingly strict in preventing the importation of diseased Africans, while the English had no such laws.

I have already tried to show the effects of segregation in Europe. In Norway, during the last twenty years, the disease has decidedly diminished most in those districts in which the most perfect segregation in hospitals has been carried out,[3] and this although only about one-fourth of the leper population are so segregated.

Whether there is any necessity for segregation in the earlier stages of the disease may possibly be questioned, but, in the later, the ulcerative stages, it is the undoubted duty of every government, with the well-being of the population at heart, to insist on such a measure just as strictly as they would against smallpox.

It is sad to think that in any colony of England a leper should be allowed *to keep a school*, as I have seen to my horror in St Kitts. In misgoverned Crete[4] such things might be, but done in an English colony, with the tacit sanction of the Government, acting under the instructions of the Home Government, themselves instructed by the Royal College of Physicians of London as to the non-contagious nature of the disease, the latter acting on utterly worthless *negative* evidence, so done, such an affair is a disgrace to humanity.[5]

[1] Emancipation took place in 1834, but the slaves remained apprentices till 1838, and it would be some years later before the effects of the freedom of lepers to mingle with others could show themselves.

[2] *Op. cit.*, p. 6. [3] Carter, 1876, Reports. [4] "Col. Phys. Report," p. 65.

[5] This appears the more forcibly when we see the Spanish Government—one

Such segregation as I advocate should include the entire separation of the sexes, except of those already married, and those only being allowed to live together if arrangements could be made for *the immediate* removal of any children born to them, whose married rights, so far as the circumstances allow, ought to be considered.[1]

It has been objected by the Government of India that the expense of segregating over 100,000 lepers in asylums would be too great, but I hardly think this is a proper view of the case. No such asylums need be built, but segregation could still be enforced by the compulsory confinement of lepers to certain spaces of land on which proper villages could be built for them, while they would when able be encouraged to work on the surrounding land. Properly managed, such communities might be partly self-supporting. Of course, after a leper was once put into such a village, a severe penalty should be enacted from any one aiding or abetting him in leaving it; at the same time there could be no harm, I believe, in allowing the lepers, under proper supervision, sometimes to see and converse with their friends at some place near the village, *so long as no contact was allowed.*

Probably over two hundred such villages would be required for the whole of India. Of course medical officers would be required to live near each of them, but the work could be nearly, if not quite, all done by lepers. Such segregation would, I believe, with the other means already mentioned, succeed in stamping out the disease.

In bringing this work to a close, I would beg to say that I have throughout tried to be strictly accurate in all statements made or references quoted; errors may have, and from the great number of references, possibly have been made in the latter as to pages or the like; but if I have in any way thrown new light on the subject, or brought it more within the reach of some to whom information may have been wanting, or, above all, if I have at all assisted in proving that leprosy is a communicable disease, I will feel that my time and labour have been well spent, and, so far as I could expect, my object in spending them gained.

Note.—To any one to whom many of the references given by me may not be available, Neale's *Medical Digest* may be very useful; there are also a few in the *New Syd. Soc. Retrosp.*, 1873–4, p. 88.

supposed to be much behind the British in many ways—ready at once to establish a lazerat in Alicant on the disease threatening to spread in that province (*Le Mouvement Médical*, 12th October 1878).

[1] Through the kindness of Dr Semper of St Kitts, I have lately been informed by letter that the two children of Hannah Carty, now 6½ and 10 years old, are still perfectly healthy, though born when she was a confirmed leper. Such examples show that the children of lepers are not always doomed to be attacked, though safer when removed from their parents.

EDINBURGH : PRINTED BY OLIVER AND BOYD.