History

# On the etiology and transmission of leprosy in nineteenth century Madras, India

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## INTRODUCTION

Thanks to the Norwegian physician Gerhard Hansen, we knew of *Mycobacterium leprae* (Actinobacteria: Actinomycetales: Mycobacteriaceae) and much of the etiology of leprosy (Hansen's disease) by 1873.[1] Although the subtle biological details of *M. leprae* were known only in the late 19th century, the Madras Presidency in British India was a particular focus of leprosy treatment and research in the early nineteenth century. The present paper focuses on the local British medical efforts to understand leprosy in early nineteenth-century Madras, particularly those detailed in the little known paper by the Madras surgeon William Judson van-Someren, published in the Madras Quarterly Journal of Medical Science in 1861.[2] The Madras Leper Hospital (MLH) was established as an institution separate from the Madras Native Infirmary (MNI) in 1814, because both medical authorities and patients at the MNI had become increasingly concerned that contact with the leprosy patients would spread the disease.[2,3] van-Someren, as superintendent of MNI, took the opportunity of a growing patient cluster at MLH to learn about a disease, which caused so much personal suffering and to critique current theories of disease transmission.

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## VAN-SOMEREN ON THE ETIOLOGY AND TRANSMISSION OF LEPROSY

The key debate over the etiology of leprosy at the time was the mechanism of disease transmission. van-Someren analyzed the patient population under his charge in terms of the tubercular—anesthetic model of 1861, in an attempt to find patterns of hereditary transmission.[2] His observations challenged the view of other Madras surgeons including James Lawder, who was in charge of MNI in 1839 and from 1840, the MLH. Lawder argued in favor of inheritance as a major cause of leprosy transmission,[2] van-Someren found evidence that of 13 married patients, no leprosy was found in the 46 children among them. Similarly, none of the parents of the 13 had the disease.[2] Consequently, van-Someren argued that "inheritance does not constitute a strong predisposition to the disease, even if do so at all."[2]

In the absence of observable evidence of leprosy transmission through inheritance, van-Someren sought and critiqued alternative theories including the observation of W. G. Davidson, Lawder's contemporary at MLH. Davidson, expressing a dominant international view at the time suggested[2]:

"This disease would appear to originate in the use of moldy grain and other unwholesome food as semi-putrid fish or animal food,—want of the use of salt and of vegetables."

van-Someren broadly supported Davidson's remarks, particularly as they accorded with M. M. Boeck and Danielssen's groundbreaking work *Traité de la Spédalskhed ou éléphantiasis des Grecs*, published in Paris in 1848. van-Someren's position seems to exist

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in transition between humoral theories of disease causation and the more familiar understanding of the links between diet and health.[2] His specific observations on disease typology closely anticipate facts of biology and etiology of leprosy we know today. Still seeking a clue to the cause of the disease, van-Someren describes leprosy as a "blood disease, which keeps a toxic element, such as the potential poison of any zymotic disease, or even such a materies morbi as lithic acid [uric acid] or urea." In current terminology, 'zymotic disease' means any epidemic, endemic, contagious, or sporadic affliction, induced by either a morbific principle or an organism, and its action on the host system is similar to fermentation.[4]

We know today that *M. leprae* is an obligate intracellular bacterium.[5] In the context of the physiology of mycobacteria in general and in the specific context of *Mycobacterium smegmatis*, we know that these bacteria utilize sugars (e.g., pentoses, hexoses) for their respiration following an anaerobic pathway (= fermentation). *Mycobacterium smegmatis* strain mc2155 has been demonstrated to be capable of fermenting glucose for its respiratory activity through an increased uptake of glucose; this has been proved by the occurrence of a special class of transport proteins, *viz*., glucose permease.[6] More evidence is available today to infer that the mycobacteria use a metabolic pathway, whereby cells grow slowly using oxidative phosphorylation to generate large quantities of ATP at a slow rate, similar to the physiology of diverse fermentative microbes.[7] van-Someren also uses the term 'toxic element,' deriving 'toxic' from *toxicum*, *toxicus* (Latin) to mean 'poison.' We know today that during infection, *M. leprae* confronts stress-indicating radicals (e.g., reactive nitrogen species [RNS] and superoxide [$O^{2-}$]) at the inflammation site.[8] We also know that the products of RNS (e.g., nitric oxide, peroxynitrite) produced in lepromatous tissue result in nerve damage.[9] van-Someren refers to the accumulation of either 'lithic acid' or 'urea.' Although the accumulation of these metabolic products has no direct bearing in the contemporary understanding of the etiology of leprosy, what cannot be denied is the metabolic relationship between the production of uric acid and peroxynitrite in human systems and the role of nitrogenous-waste materials in human diseases in general.[10]

In his later argument, van-Someren argues for dyscrasia in the blood as a contributory factor to leprosy. He[2] similarly offers insights into the inflammatory character of the disease. He notes that in leprosy, "The proportion of albuminous materials is largely increased; while that of the red-corpuscles is notably diminished, and as the latter are formed at the expense of the former, it is difficult to reconcile this diminution of the red discs with the super-abundance of well-elaborated albumen in the vital fluid. It is well known that in certain vessels of the body, this nitrogenous compound exists in the crude and imperfect form of albuminosae, and the idea has been broached by pathologists that when found in the renal excretion, its presence is due to the fact that, in this ill-elaborated form, it easily transudes the tissues of the kidneys. If this suggestion be true, the facile escape of albuminous matter into the cutaneous and mucous tissues, so as to form tubercular elevations characteristic of tubercular leprosy and its exudation into the serous tissue of the arachnoid membrane covering the spinal cord in the anesthetic form of the disease, are explicable in the same way, and our therapeutics should be directed towards the removal of the dyscrasia, which characterizes the blood."

Considering what we know of the etiology and prognosis of the illness, some of van-Someren's remarks appear fascinatingly true. Leprosy being an inflammatory disease – similar to tuberculosis—notably shows low total counts of RBC.[11] In general, we are aware today that a rise in albuminous materials also indicates the inflammatory nature of the illness. However, Bulakh *et al*.[12] point out that serum-cholinesterase levels decrease abruptly with exacerbation of leprosy, whereas serum-albumin levels drop gradually. Elevated levels in albuminous materials, *sensu* van-Someren, probably refer to the granulomatous condition of leprosy. Today, we know that in patients with a high level of T-cells (a type of lymphocyte) responsiveness against *M. leprae*, granulomas of outer skin develop rapidly, whereas in patients with a low level of T-cell responsiveness, *M. leprae* multiplies unrestrained, thus building in numbers in tissue, leading to a gradual manifestation of the disease.[13] In the late 1830s, Charles Cagniard-Latour[14] and Theodor Schwann[15] proposed that micro-organisms play a more vital role in the economy of nature than anyone could imagine. Their principle led to the conviction that micro-organisms exist in all putrefying matter, either of animal or of plant source, and that they induced putrefaction. This principle influenced surgeons of the late nineteenth century to see putrefaction as a key mechanism in the

prognosis of any disease of 'unknown' etiology; therefore, pathological examinations were targeted at assaying the end and by-products keeping putrefaction in view.[16] For instance, in the context of van-Someren remarks, excessive albuminous materials were considered to have arisen in consequence of putrefaction; however, what needs to be factored here is that leprosy in 1860s was a disease of unknown etiology.

## CONCLUSION

Poor-quality food, overcrowding, impure air, lack of hygiene, which rely on poverty, van-Someren identifies as the key reasons for the blood dyscrasia, which according to him leads to leprosy. In support of this argument, he highlights the prevalence of leprosy in the British Isles from the twelfth to the sixteenth centuries, which disappeared with economic development and wealth increase. In the absence of the fact that *M. leprae* was known 12 years later and clear mechanisms of disease transmission, van-Someren concludes that the best prophylactics against the development of leprosy were hygienic measures, a healthy diet, and encouraging patients in gardening to grow food for a healthy diet.[2]

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## REFERENCES

1. Hansen GH. Investigations on the etiology of leprosy. Norsk Mag Lægevidens 1874;4:1-88.
2. van-Someren WJ. A brief historical sketch of the Madras leper hospital with some notices of leprosy as it is seen in that institution. Madras Q J Med Sci 1861;3:271-94.
3. Buckingham J. Leprosy in colonial south India: Medicine and confinement. London: Palgrave Macmillan; 2002.
4. Bazin H. Vaccination a history: From Lady Montagu to genetic engineering. Eurotexted. Esher: John Libbey; 2011.
5. Britton WJ, Lockwood DN. Leprosy. Lancet 2004;363:1209-19.
6. Pimentel-Schmitt EF, Jahreis K, Eddy MP, Amon J, Burkovski A, Titgemeyer F. Identification of a glucose permease from *Mycobacterium smegmatis* mc2155. J Mol Microbiol Biotech 2008;16:169-71.
7. Cook GM, Berney M, Gebhard S, Heinemann M, Cox RA, Danilchanka O, *et al*. Physiology of mycobacteria. Adv Microb Physiol 2009;55:81-182.
8. Fabozzi G, Ascenzi P, di Renzi S, Visca P. Truncated hemoglobin Glb0 from *Mycobacterium leprae* alleviates nitric oxide toxicity. Microb Pathogen 2006;40:211-20.
9. Schön T, Hernández-Pando R, Baquera-Heredia J, Negesse Y, Becerril-Villanueva LE, Eon-Contreras JC, *et al*. Nitrotyrosine localization to dermal nerves in borderline leprosy. Br J Dermatol 2004;150:570-4.
10. Pacher P, Beckman JS, Liaudet L. Nitric oxide and peroxynitrite in health and disease. Physiol Rev 2007;87:315-427.
11. Skinsnes OK. Infectious granulomas: Exposit from the leprosy model. Annu Rev Med 1982;33:47-67.
12. Bulakh PM, Ranade SM, Chandorkar AG, Muley PR. Correlation between serum choline-esterase and serum albumin in leprosy. Indian J Lepr 1985;57:524-8.
13. Anton H. Short review of general pathological anatomy. Department of Pathological Anatomy. Grodno: Grodno Medical University; 2006.
14. Cagniard-Latour C. Mémoire sur la fermentation vineuse. Ann Chim Phys 1838;68:206-22.
15. Schwann T. Vorläufige Mitteilung, betreffend Versuche über die Weingährung und Fäulnis. Ann Phys Chem 1837;41:184-93.
16. Woodhead GS. Bacteria and their products. London: Walter Scott; 1891.