

British India and the “Beriberi Problem”, 1798–1942

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In 1898 Patrick Manson gave a graphic account of beriberi. He described how medical visitors to “native hospitals in many parts of the tropical world” were likely to have “their attention arrested by the large proportion of cases of partial paraplegia, of cases of oedema of the legs, and of cases of general dropsy. These, for the most part, are cases of beriberi”.¹ The visitor, Manson continued, “may be struck with the thinness of the patients’ calves, the flabby state of the gastrocnemii, and by the fact that if, whilst making the examination, he should handle these and the neighbouring muscles somewhat roughly ... the patient will call out in pain and try to drag the limb away.”² Similar cases were to be found throughout the hospital wards: “Some are so trifling that they are up and moving about with more or less freedom; others are so severely smitten that they lie like logs in their beds, unable to move a limb or perhaps even a finger. Some are atrophied to skeletons; others are swollen out with dropsy; and some show just sufficient dropsy to conceal the atrophy the muscles have undergone.”³

Manson’s description, written on the cusp of discoveries that transformed the scientific understanding of beriberi, gave the disease, despite its apparently diverse manifestations, an embodied visibility.⁴ The clear corporeal presence of beriberi stood, however, in contrast with Manson’s uncertainty (and that of many of his contemporaries) about the exact nature and cause of a disease, despite its widespread distribution in the tropics among “such conglomerations of humanity as are found in Oriental jails, schools, mining camps, plantation lines, armies, ships”.⁵ For Manson, beriberi typified those diseases that remained part of the “tropical pathological puzzle”. Despite personally believing it to be “a disease of locality”, and in this respect “resembling malaria”, Manson felt obliged for want of evidence to include

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¹ Patrick Manson, *Tropical diseases: a manual of the diseases of warm climates*, London, Cassell, 1898, p. 223.

² *Ibid.*, p. 223.

³ *Ibid.*, pp. 228–9.

⁴ McCarrison cited this passage in his own attempt, thirty years later, to describe the defining physical characteristics of beriberi: Robert McCarrison, *Beri-beri columbarum*, Calcutta, Thacker, Spink, 1928, p. 1.

⁵ Manson, *op. cit.*, note 1 above, p. 233.

it among “diseases of undetermined nature”.⁶ His account captures a defining moment in the historical understanding of a disease, illuminating the contrast between what could readily be observed and experienced by the physician and what yet remained aetiologically indeterminate. Beriberi, sometimes seen in the nineteenth and early twentieth centuries as little more than a name (one, moreover, with an uncertain, even “fanciful”, provenance⁷), a convenient label for a diverse set of symptoms rather than a single, specific disease, sharpens our sense of that epistemological irresolution and contests medical histories that are predicated, from the security of retrospection, on the certainty of a disease’s nature and existence.

The “discovery” and “conquest” of beriberi is a story that has been extensively told. Medical historians have recounted the pioneering identification of the disease in maritime Asia, the growing incidence observed during the nineteenth and early twentieth centuries and the alarm to which this gave rise in Japan and among western powers in Asia, the investigations into the analogous condition of polyneuritis in fowls and the part thus played in the discovery of vitamins and their properties, the supplanting of earlier environmental or germ-theory explanations by a new understanding of the disease as caused by nutritional deficiency, and the general recognition by about 1910 of the connection between beriberi and diets of milled “white” rice. To this narrative of progress from ignorance and uncertainty to enlightened understanding is added the subsequent quest to establish the chemical and physiological processes by which the lack of vitamin B1 (thiamine) causes beriberi. This is a history that has been written in terms of the emergence of nutritional science, as a seminal episode in the history of tropical medicine, and in relation to national and international measures to raise dietary standards and to combat a disease responsible for impeding health and development across the tropical world.⁸

This article does not seek to re-excavate old ground but, using Manson’s irresolution as its point of departure, to present a different perspective on the history of the disease. It does so, firstly, by looking at India, a country largely ignored in most recent accounts of beriberi, and yet one that had a long history of engagement with the disease, extending across the colonial *longue durée*. Secondly, although some medical historians may elect to pass rapidly over the “false theories” that preceded the vitamin-deficiency explanation,⁹ the conflicting claims made about the disease in nineteenth- and early twentieth-century India were integral to its interpretive history and influenced the ways in which the “beriberi problem” was addressed

⁶ *Ibid.*, pp. 233, 235, 238.

⁷ *Report on the medical topography and statistics of the Northern, Hyderabad and Nagpore Divisions, the Tenasserim Provinces, and the Eastern Settlements*, Madras, Vepery Mission Press, 1844, p. 89. Morehead cautioned against the “unnecessary introduction” of the word “beriberi” into Indian nosology as tending to “retard and obscure” the knowledge of what he regarded as a form of “general dropsy”: Charles Morehead, *Clinical researches on disease in India*, 2 vols, London, Longman, Brown, Green and Longmans, 1856, vol. 2, p. 685.

⁸ Robert R Williams, *Towards the conquest of beriberi*, Cambridge, MA, Harvard University Press, 1961; K Codell Carter, ‘The germ theory, beriberi, and

the deficiency theory of disease’, *Med. Hist.*, 1977, **21**: 119–36; Kenneth J Carpenter, *Beriberi, white rice, and vitamin B: a disease, a cause, and a cure*, Berkeley, University of California Press, 2000; Ken De Bevoise, *Agents of the apocalypse: epidemic disease in the colonial Philippines*, Princeton University Press, ch. 5; Lenore Manderson, *Sickness and the state: health and illness in colonial Malaya, 1870–1940*, Cambridge University Press, 1996, pp. 90–2; Julyan G Peard, *Race, place, and medicine: the idea of the tropics in nineteenth-century Brazilian medicine*, Durham, NC, Duke University Press, 1999, pp. 51–63; Anne Hardy, ‘Beriberi, vitamin B1 and world food policy, 1925–1970’, *Med. Hist.*, 1995, **39**: 61–77.

⁹ Williams, *op. cit.*, note 8 above, p. 13.

or ignored. And thirdly, the disease highlights the ways in which colonial medicine in British India evolved its own ways of observing, conceptualizing and responding to disease, ways that were often isolated from, or at variance with, dominant international trends. While beriberi can be understood as illustrating the idiosyncratic nature of India’s disease ecology, it can no less significantly be seen to illuminate the tensions and traditions operating within its colonial medical establishment. Beriberi became emblematic of the discomfort felt by many in positions of medical authority in British India with innovations in tropical governance across monsoon Asia,¹⁰ which required a more interventionist strategy, and with international trends in the investigation and containment of tropical diseases, which placed increased importance on both laboratory science and public health activism. What emerges is less a history of “discovery” and “conquest” than one of continuing contradictions and constraints on action in which the “beriberi problem” was lost sight of rather than being resolved.

India and the Early Discourse on Beriberi

The early history of beriberi has been traced to various locations across Asia including China, Japan and Indonesia. Already identified in Japan as *kakké* (“leg disease”), descriptions of the disease were also given by early western physicians in the region, notably Jacobus Bontius in Java in 1629.¹¹ Although the origin of the name “beriberi” was (and is) obscure, and was said to be a term used by none of the indigenous populations afflicted by the disease,¹² as the designation for a condition hitherto unknown to Europe, it drifted around maritime Asia, carried on the tides of western commerce and conquest, before making landfall in South Asia in the late eighteenth century.

The first substantial English account of beriberi was made in Ceylon in the late 1790s and early 1800s by an army physician, Thomas Christie, during the initial phase of British occupation of the island.¹³ Christie had had contact with Dutch physicians (from whom he adopted the name “beriberi”),¹⁴ but his observation of the disease was principally among soldiers of the English East India Company recruited from the south-eastern (Coromandel) coast of India, where it was reputedly widespread. Although he believed “beriberi” to be a Sinhalese name, Christie did not think that the disease greatly affected the local population, and, though he claimed that it afflicted both Indians and Europeans, most of his cases in fact concerned white soldiers. He gave a detailed description of the disease, noting in particular paralysis of the lower limbs, oedema and dropsy.¹⁵ Christie forwarded his observations to William Hunter in London, who was engaged in an enquiry into mortality among Indian lascars, but, while identifying many similarities between beriberi and

¹⁰ On “monsoon Asia”, see Robert Orr Whyte, *Rural nutrition in monsoon Asia*, Kuala Lumpur, Oxford University Press, 1974; V D Wickizer and M K Bennett, *The rice economy of monsoon Asia*, Stanford University Press, 1941, pp. 1–4.

¹¹ Edward B Vedder, *Beriberi*, London, John Bale, Sons and Danielsson, 1913, pp. 1–9; Harold J Cook, *Matters of exchange: commerce, medicine, and science in the Dutch golden age*, New Haven, Yale University Press, 2007, pp. 195–6.

¹² George M Giles, *A report of an investigation into the causes of the diseases known in Assam as kála-azár and beri-beri*, Shillong, Assam Secretariat Press, 1890, p. 1.

¹³ William Hunter, *An essay on the diseases incident to Indian seamen, or lascars, on long voyages*, Calcutta, The Honourable Company’s Press, 1804, pp. 77–141.

¹⁴ *Ibid.*, pp. 86, 128.

¹⁵ *Ibid.*, pp. 77–80.

shipboard scurvy, Christie also stressed significant differences. Not only were the swollen gums, foetid breath and spotted limbs found in scurvy absent, but the disease was also unresponsive to anti-scorbutic lime juice. He cited “moisture, impure air, despondency [and] dirtiness” as contributing factors, but primarily attributed the disease to “a want of stimulating and nourishing diet” among *sepoys* (soldiers) whose food consisted almost entirely of rice. He considered a more plentiful and varied diet the best treatment.¹⁶ There was much in Christie’s account that anticipated later discussion, including the particular vulnerability of south Indians, the emphasis on diet as both cause and cure, and the characterization of beriberi as a disease similar to, but not identical with, scurvy.¹⁷ However, perhaps because it was buried within Hunter’s account of lascar diseases, Christie’s observations failed to attract much comment.¹⁸

In the 1830s the locus of enquiry shifted from Ceylon to south India, from where several descriptions of the disease were published.¹⁹ One stimulus for this was the decision by the Madras Medical Board in 1832 to offer a prize for an essay on beriberi as a way of encouraging scientific investigation into a disease known to be “insidious in its attack, rapid in its progress, and fatal in its termination”, and apparently widespread among soldiers from the northern districts of the Madras Presidency.²⁰ Of the four essays received, all written by army medical officers, the prize was awarded to John Grant Malcolmson, an assistant surgeon in a Madras European regiment. In his *Practical essay*, Malcolmson suggested that the name “beriberi” was derived from the Hindustani word for a sheep and referred to the awkward, sheep-like gait that was characteristic of the disease, though he noted that the local population failed to distinguish this from many other complaints: its Telugu name, “timmerē waivo”, was said to cover palsy, tingling sensations, and even rheumatism. Although, as the Medical Board required, Malcolmson investigated “the practice of the more intelligent native doctors” and the medicinal substance known as “treeak farook”, beriberi was not a disease in which indigenous testimony and therapeutic practice played much part.²¹

The primary symptoms of beriberi, as Malcolmson understood them, included numbness, paralysis of the lower limbs, oedema and dropsy. Drawing on his own experience and that of other army officers, Malcolmson described the disease in similar terms to Christie but, at a time when medical topography was coming into vogue in India,²² he was anxious to

¹⁶ *Ibid.*, pp. 79, 128.

¹⁷ *Ibid.*, pp. 82, 86. The idea of beriberi as a scurvy-like complaint was a recurrent one: Morehead, *op. cit.*, note 7 above, vol. 2, pp. 685–99; E D W Greig, *Epidemic dropsy in Calcutta*, Calcutta, Superintendent of Government Printing, India, 1911, pp. 6–8. That scurvy resulted from “a defect of nourishment”, and might be found on land as well as sea, was well established in early nineteenth-century medical literature in India: B Burt, ‘On land scurvy among the natives’, *Transactions of the Medical and Physical Society of Calcutta*, 1829, 4: 14–20.

¹⁸ But see James Johnson and James Ranald Martin, *The influence of tropical climates on European constitutions*, New York, Samuel and William Wood, 1846, p. 385, where Christie’s views on “debauchery” as predisposing to beriberi are cited.

¹⁹ Including an anonymous essay cited in *Report on the medical topography*, *op. cit.*, note 7 above, pp. 89–96; P W Wright, ‘Remarks on beriberi’, *Edin. Med. Surg. J.*, 1834, 41: 323–9. Wright’s essay was one of the four submitted to the Madras Medical Board in 1833.

²⁰ Madras Consultations, 11 May 1832, Board’s Collections F/4/1454: 57130, India Office Records (hereafter IOR), British Library, London.

²¹ John Grant Malcolmson, *A practical essay on the history and treatment of beriberi*, Madras, Vepery Mission Press, 1835, pp. i, 2–6, 296–311.

²² Mark Harrison, ‘Differences of degree: representations of India in British medical topography, 1820–c.1870’, in Nicolaas A Rupke (ed.), *Medical geography in historical perspective*, London, Wellcome Trust Centre for the History of Medicine at UCL, 2000, pp. 51–69.

establish the likely geographical and climatic influences on beriberi. He noted that it mainly prevailed among troops stationed in the coastal tract of the Northern Circars, an area about 60 miles wide and 400 miles long running from Ganjam in the north to Nellore in the south. Malcolmson thus helped establish the idea of beriberi in India as a highly localized disease, confined to only a small portion of the Madras Presidency and absent even from neighbouring districts.²³ By his account, European as well as Indian troops suffered from beriberi (though he mostly described the latter). He noted a marked seasonality to the disease, its highest incidence occurring in the months from August to October, following the monsoon rains. This climatic aspect of the disease was, in his view, “of the highest importance to the health of the troops”, for their movements could be adjusted so as to avoid the most “unfavourable” months.²⁴ Beriberi appeared, too, to target newcomers, proving most fatal after ten to eighteen months’ residence in the area, but (like malaria and other diseases observed in India at the time) declining with “seasoning” or acclimatization.²⁵ Although unable to document the extent of beriberi among civilians, Malcolmson believed the lower classes suffered most. However, beyond proposing environmental influences,²⁶ he could offer no conclusive explanation for its cause. He thought it unlikely that it was due to a “deficient and unhealthy diet”, for the area appeared blessed with abundant grain, though he conceded that “deficient nutriment” might “predispose” the poor to beriberi.²⁷

Many characteristics of Malcolmson’s essay situate it in its place and time—the military site of observation, the dearth of information about the mass of the population, the recourse to medical topography and environmental reasoning. He made no attempt to situate beriberi more widely as a “tropical disease”, and yet, as the investigation of beriberi burgeoned in the late nineteenth century, his essay was often cited, especially by medical authors in British India, as an authoritative text.²⁸ It is important to bear in mind, though, that other descriptions of beriberi appeared around the same time as Malcolmson’s and gave a different account of the disease. For instance in the same year, 1835, a naval surgeon, James Bankier, suggested a significant, if ill-defined, link with poor vegetarian diets, and, while noting the existence of the disease in India, emphasized that “the Ceylon palsy” was also present on that island. In a gesture towards the idea of beriberi as a contagious disease, and not one simply confined to a small part of south India, Bankier described it as “a very troublesome ... complaint”, analogous to cholera, a disease then rampant in India and making deadly inroads into Europe.²⁹

Thus, by the late 1830s beriberi had attracted a broad spectrum of medical opinion in colonial South Asia without a consensus as to its nature and causes having been arrived at. In the

²³ Malcolmson, op. cit., note 21 above, p. 27.

²⁴ Ibid., p. 16. For the seasonality of acute thiamine deficiency, see Whyte, op. cit., note 10 above, pp. 33–6.

²⁵ Malcolmson, op. cit., note 21 above, pp. 21–5. On “seasoning”, see Mark Harrison, *Climates and constitutions: health, race, environment and British imperialism in India, 1600–1850*, New Delhi, Oxford University Press, 1999, pp. 88–92.

²⁶ Other writers blamed the “low, damp and more or less swampy” conditions of the Northern Circars: Wright, op. cit., note 19 above, p. 323; *Report on the medical topography*, op. cit., note 7 above, p. 89.

²⁷ Malcolmson, op. cit., note 21 above, pp. 42, 46.

²⁸ William Campbell Maclean, *Diseases of tropical climates*, London, Macmillan, 1886, p. 311; Giles, op. cit., note 12 above, p. 1; C M E Dubruel, *Le bérubéri*, Paris, J-B Baillière, 1906, p. 7.

²⁹ James Bankier, *Essay on the origin, progress and treatment of cholera, with remarks on beriberi and diet*, Madras, J B Pharoah, 1835, pp. 409–10. The idea of beriberi as a contagious disease had a long history, and not just in South Asia: e.g., B Scheube, *The diseases of warm countries: a handbook for medical men*, 2nd ed., London, John Bale, Sons and Danielsson, 1903, p. 191.

main, though, beriberi appeared to be a disease of relatively minor significance, outweighed by diseases—such as smallpox, cholera and malaria—that caused far greater mortality. Despite references in Christie and Malcolmson to the incidence of the disease among soldiers, accumulating evidence showed it to be a minor threat to both European and Indian troops.³⁰ Malcolmson suggested that the incidence of beriberi might be under-reported due to official insistence on the use of William Cullen’s nosology to record diseases: it might be subsumed under other headings including “dropsies”—which was precisely where Charles Morehead, writing from Bombay in the 1850s, believed it belonged.³¹ In practice, though, it was usually returned as a “specific disease”, alongside leprosy, elephantiasis and scurvy. The number of cases reported from military hospitals and civil jails in the Madras Presidency in the 1820s and 1830s was relatively low even in the districts where beriberi was considered endemic and that for a disease seen primarily to afflict middle-aged men and rarely women and children.³² Significantly, Malcolmson’s essay, written in late 1832 or early 1833, made no reference to famine, and yet a few months later, Guntur, one of the districts where beriberi appeared to be endemic, was devastated by drought and dearth. It is unclear how far beriberi was implicated in the resulting upsurge of sickness and mortality, though reports from some interior districts listed beriberi among the main causes of prison deaths in 1833–4.³³ Medical accounts of the Madras famines of 1866 and 1876–8 concentrated on cholera, smallpox, dysentery and malaria, but were silent on beriberi.³⁴ Despite a growing literature in India linking diet and disease, much of it concerning prisoners and labourers,³⁵ beriberi garnered hardly a mention.

Beriberi as Anomaly

The occasional essays on beriberi that appeared in Indian medical journals in the mid- and late nineteenth century emphasized its almost exclusive concentration in the Northern Circars, its apparently non-contagious nature and likely climatic (or, less frequently, dietary) influences.³⁶ When medical officers with south Indian experience discussed beriberi in a wider context, it was generally, as in Christie’s observations, in terms of rice-eating Madras soldiers overseas and carrying the disease with them.³⁷ Despite being listed among “tropical diseases”, beriberi appeared, from a South Asia perspective, to be an extremely localized affliction and, as with cholera,³⁸ often served as a vehicle for ideas of Indian exceptionalism, with a continuing prioritization of place and climate as determinants

³⁰ In India beriberi occurred almost entirely among Madras troops, who formed a declining proportion of the Indian Army. In 1905 there were 15 hospital admissions for beriberi among Indian troops with two deaths: *Annual report of the sanitary commissioner with the Government of India, 1905*, p. 126.

³¹ Malcolmson, op. cit., note 21 above, pp. 9, 13–14; Morehead, op. cit., note 7 above, 2: 684–90.

³² *Report on the medical topography*, op. cit., note 7 above.

³³ *Report on the medical topography and statistics of the Ceded Districts*, Madras, Vepery Mission Press, 1844, pp. 24, 27, 58.

³⁴ Alexander Porter, *The diseases of the Madras famine of 1877–78*, Madras, Superintendent, Government Press, 1889.

³⁵ David Arnold, ‘The “discovery” of malnutrition and diet in colonial India’, *Indian Econ. Soc. Hist. Rev.*, 1994, 31: 1–26.

³⁶ W F Thomas, ‘Beri-beri’, *Indian Med. Gaz.*, 1889, 24: 107–10.

³⁷ J Smyth, ‘Beri-beri’, *Br. Med. J.*, 1889, i: 193.

³⁸ Mark Harrison, ‘A question of locality: the identity of cholera in British India, 1860–1890’, in David Arnold (ed.), *Warm climates and western medicine: the emergence of tropical medicine, 1500–1900*, Amsterdam, Rodopi, 1996, pp. 133–59.

of disease. When W C Maclean, a former Surgeon-General of the Indian Army, lectured on the “diseases of tropical climates” at the Army Medical School at Netley in the 1880s, his account of beriberi dealt entirely with the disease as he had encountered it in south India or among south Indian troops in Burma: only a brief postscript noted recent reports of beriberi among soldiers of the Dutch East Indies. While the causes of this “extremely fatal” disease remained “obscure”, Maclean had little doubt that the water, soil and climate of the Northern Circars were largely responsible, accentuated by poverty, insufficient food and clothing. The “half-starved” Madrasis sent to Burma returned “more or less scorbutic” and, on exposure to the climate of their home districts, succumbed to the disease.³⁹ Reports of beriberi among south Indian coolies in Rangoon or lascars arriving in British ports reinforced the perception that this was a disease peculiar to south India and its extensions overseas.⁴⁰

The elusive and anomalous nature of the disease, whose “barbarous” designation seemed to strengthen its obscurity,⁴¹ was compounded by uncertainty over its possible epidemicity, especially when discussion shifted, late in the century, from Madras to the north-eastern provinces. Since to many medical commentators “beriberi” appeared little more than a name attached to a set of symptoms, rather than a discreet disease entity, it could be applied to a variety of conditions. Although paralysis of the lower limbs and oedema were considered primary manifestations of the disease, until the late 1880s doctors in Assam followed their counterparts in Ceylon in calling anaemia among tea-estate labourers “beriberi”. Only after W R Kynsey’s report on “the anaemia, or *beriberi*, of Ceylon” appeared in 1877 was this identified as hookworm disease and differentiated from the beriberi described by Malcolmson.⁴² Even so, it took more than a decade for the “lamentable confusion” between ancylostomiasis and “true” beriberi to be resolved.⁴³

In a further shift in the understanding of the disease and its geographical locus, in 1886 Norman Chevers, a long-serving medical officer who rose to be Surgeon-General of the Bengal Army and Principal of the Calcutta Medical College, claimed that beriberi had for too long been placed in an “anomalous position in our nomenclature of diseases, as the last and least defined in the roll of general diseases”. It should be re-designated “beriberi fever” and placed next to scarlatina, “with which fever it had certain marked features in common”.⁴⁴ In making this assertion Chevers drew on recent reports of a disease termed “epidemic dropsy” which had appeared in Madras in 1877, then spread to Bengal and northeastern India before following Indian emigrants to Mauritius. Unfazed by “one of the most obscure problems in aetiology”, Chevers argued that what earlier authors described as “beriberi” was actually the sequel to an earlier attack of fever. Malcolmson and those who followed him “threw over this disease a darkness which has never yet been dispelled, by commencing their description of its symptoms at the beginning of the end”: they started with the onset of numbness rather than the fever that occasioned it. It was this contagious form of beriberi that had then spread to Mauritius, Singapore, Japan and Brazil.⁴⁵

³⁹ Maclean, *op. cit.*, note 28 above, pp. 308–20.

⁴⁰ C Barry, ‘Notes on beri-beri in Rangoon’, *Indian Med. Gaz.*, 1900, **35**: 343–5; T K Munro, ‘A case of ship beri-beri’, *Lancet*, 1909, **i**: 529–30.

⁴¹ Maclean, *op. cit.*, note 28 above, p. 308.

⁴² Giles, *op. cit.*, note 12 above, pp. 1–3; C P Costello, ‘Remarks on the anaemia of coolies, or the

beriberi of Ceylon as found in Assam’, *Annual sanitary report of the province of Assam*, 1888, p. 20.

⁴³ W J Buchanan, ‘Beri-beri and rice’, *Lancet*, 1898, **ii**: 577–8.

⁴⁴ Norman Chevers, *A commentary on the diseases of India*, London, J and A Churchill, 1886, p. 60.

⁴⁵ *Ibid.*, pp. 61–2.

Chevers' interpretation of beriberi as an epidemic fever was an ingenious attempt to collapse the increasingly widespread reporting of beriberi into what was known of the disease in South Asia. Despite attempts to distinguish between the two,⁴⁶ the result was an interpretive entanglement of "true" beriberi with epidemic dropsy which dominated a large part of the Indian medical literature until the 1930s, and which, having received a fresh fillip from outbreaks of epidemic dropsy in Calcutta, Bengal and Assam in 1909–10 and 1926, distracted from research on beriberi itself.⁴⁷ This eventually proved a false trail: epidemic dropsy was ultimately shown to be distinct from beriberi, probably caused by contaminated cooking oil.⁴⁸ Significantly, the invocation of epidemic dropsy in India had no parallel in debates elsewhere, though its supposed relationship with beriberi was sometimes accepted on the authority of the British medical establishment in India.⁴⁹ But, for many in the Indian Medical Service (IMS), its identification as the epidemic form of beriberi offered an apparent explanation for a disease exhibiting many similar symptoms, including breathlessness, foot-swelling and paralysis. It gave beriberi the epidemic status India's colonial physicians had intermittently sought for the disease, and seemed to give their investigations into beriberi a unique insight and an international mission that, in the competitive world of tropical medicine, might put British India on a par with, even ahead of, investigations elsewhere.

India and the International "Beriberi Problem"

By the early twentieth century, there existed a vast scientific literature on beriberi, including articles and books in Dutch, French, German, Portuguese, Norwegian, Japanese and English.⁵⁰ The geographical and linguistic range of these works expressed growing international concern at the increasing incidence of a disease that, although primarily observed in monsoon Asia, seemed to extend across the tropical world and beyond. Moreover, while medical observers in India had, until Chevers, regarded beriberi as a local disease of secondary importance, research in Japan, the Dutch East Indies, British Malaya and Brazil showed it to be a disease of great economic, social and political significance, resulting in particular in high levels of incapacity and mortality among soldiers, sailors, plantation labourers, prisoners and asylum inmates. Such was the level of concern that in Japan, the Dutch East Indies, Singapore, Malaya, and American-occupied Philippines, solutions were anxiously sought to the "beriberi problem", both in the sense of establishing what caused the disease and of finding out how to prevent it. While contrary views continued to circulate, following the pioneering work of Kanehiro Takaki in Japan and Christiaan Eijkman in Java a dominant view emerged that rice diets were primarily to blame, just as their elimination or modification offered the most plausible solution.⁵¹ Although by 1910 researchers in British India had made major contributions to the investigation of malaria, plague, kala-azar and

⁴⁶ Kenneth Macleod, 'Beri-beri and epidemic dropsy', in William Ernest Jennings (ed.), *Transactions of the Bombay Medical Congress, 1909*, Bombay, Bennett, Coleman, 1909, pp. 273–5.

⁴⁷ Greig, op. cit., note 17 above; Hugh W Acton and R N Chopra, 'The problem of epidemic dropsy and beriberi', *Indian Med. Gaz.*, 1925, **60**: 1–18.

⁴⁸ R B Lal and A C Gupta, 'Investigations into epidemic dropsy', *Indian J. Med. Res.*, 1941, **29**: 157–65.

⁴⁹ For example, Vedder, op. cit., note 11 above, p. 296.

⁵⁰ In 1894 Scheube listed 363 works on beriberi: B Scheube, *Die Beriberi-krankheit: eine geographisch-medicinische Studie*, Jena, Gustav Fischer, 1894, pp. 207–18. In 1903 he revised this list to include many recent publications: Scheube, op. cit., note 29 above, pp. 222–6.

⁵¹ Carpenter, op. cit., note 8 above, chs 3–6.

other tropical diseases, the position regarding beriberi and nutrition research was very different.⁵² Here India’s medical services appeared aloof, anachronistic, intent on pursuing issues, like epidemic dropsy, outside observers believed tangential to the debate.⁵³

Across much of monsoon Asia beriberi appeared to be a very modern disease. For this there were several explanations. Since the 1870s there had been the rapid growth of mechanized rice-milling. Instead of de-husking by hand, rice was now processed in power-driven mills, which, along with the husk, removed the inner skin or pericarp, the grain then being further buffeted to produce a polished, white appearance. In removing the thiamine contained in the pericarp, milling exposed consumers whose diet consisted almost entirely of white rice to the risk of beriberi. Rice polishings containing the vital nutrients removed by milling were thrown away or fed to animals. Milling fuelled a revolution in taste. The demand for hand-pounded rice fell: white rice was sought after as more palatable, more prestigious and might even be cheaper. Milling also reduced the bulk of raw paddy by a third, making rice more economical to transport and less prone to deterioration when stored and shipped. With the growth of massive exports from Burma, Thailand and Vietnam into the Philippines, Indonesia, and increasingly by the 1920s into India, the regional trade in milled rice expanded enormously—and with it beriberi. The immense value of rice exports and their central importance to Asian economies partly explains why beriberi was so difficult to tackle locally and internationally: there were too many commercial and political interests at stake.⁵⁴ Because of its cheapness, availability and acceptability, milled rice became the principal—even exclusive—diet of social groups associated with modern occupations and institutions. Beriberi spread not through contagion, as some commentators imagined, but by the replication of similar institutions and workforces across the region and through the shared dietary practices that accompanied them. The Japanese navy was one of the first to be hit in the 1870s, then soldiers in the Dutch East Indies, followed by tin-miners in Malaya, prisoners, policemen, coolies and asylum inmates in Singapore, Hong Kong, Manila and Saigon.

However, the extent of beriberi among the population at large was difficult to determine. Unlike many tropical diseases, beriberi was seldom mapped.⁵⁵ It was inscribed not across broad swathes of colonial territory, like cholera and plague, but on local sites and institutionalized bodies. Data from urban hospitals, jails and asylums gave little indication as to how widespread beriberi might be especially in rural areas. Some epidemiologists ranked beriberi among the main causes of morbidity and mortality in Asia. W L Braddon argued in 1907 that in Malaya there were currently more than 150,000 cases and 30,000 deaths a year from beriberi, and that among the Chinese population, who were most affected, there

⁵² W R Aykroyd, ‘The Nutrition Research Laboratories, Coonoor’, in H M Sinclair (ed.), *The work of Sir Robert McCarrison*, London, Faber and Faber, 1953, pp. xi–xxix, on p. xiii.

⁵³ On the development of the Indian medical services and their outlook, see David Arnold, *Colonizing the body: state medicine and epidemic disease in nineteenth-century India*, Berkeley, University of California Press, 1993; Mark Harrison, *Public health in British India: Anglo-Indian preventive medicine, 1859–1914*, Cambridge University Press, 1994; Anil Kumar, *Medicine and*

the Raj: British medical policy in India, 1835–1911, New Delhi, Sage, 1998.

⁵⁴ In the 1930s Vietnam exported more than a billion francs’ worth of rice, equivalent to nearly half its export trade: Charles Robequain, *The economic development of French Indo-China*, London, Oxford University Press, 1944, pp. 276, 310.

⁵⁵ But see *Annual report of the Public Health Commissioner with the Government of India, 1930*, vol. 1, map facing p. 134; J W D Megaw, S P Bhattacharji and B K Paul, ‘Further observations on the epidemic dropsy form of beri-beri’, *Indian Med. Gaz.*, 1928, **63**: 417–39, p. 419.

might have been 100,000 deaths over the previous twenty years.⁵⁶ In the Philippines, where American researchers established infantile beriberi as a leading cause of death, a mortality of between 12,000 and 20,000 a year was reported during the 1920s and 1930s.⁵⁷

City-based calculations fuelled claims that millions of people across Asia suffered and died from the disease. Edward Vedder of the US Army Medical Corps reckoned in 1913 that, since Hong Kong had 10,000 cases in a population of 350,000, “it may well be imagined that millions of cases must occur among the remainder of China’s teeming population”.⁵⁸ In the same year Victor Heiser, soon to become the leading figure in the Asian operations of the Rockefeller Foundation’s International Health Board, claimed that beriberi cost “the Orient” 100,000 lives a year.⁵⁹ A decade later his deputy called beriberi “a huge public health problem” and declared that the “effective control of beriberi in the Orient promises to be of great benefit to vast populations”.⁶⁰ Expressions of alarm were not confined to Euro-Americans. A Japanese researcher remarked in 1925 that there was “no other disease so important socially as beri-beri. It attacks young people during the most productive period of life ... It is time to establish the etiology of the disease and completely prevent it.”⁶¹

It is unnecessary to retrace the history of beriberi research in this period. Following pioneering investigations in Japan and the Dutch East Indies, the critical stage of enquiry occurred in a remarkably short period between 1908 and 1912. Building on Braddon’s work, which had identified an unknown rice ergot or toxi-infection as the likely culprit, Henry Fraser and A T Stanton in Kuala Lumpur sought to establish a nutritional rather than a bacteriological link with milled rice. Beriberi, they reported in 1910, “is a disorder of nutrition ... associated with a diet in which white rice is the principal constituent”. Since white rice “makes default in respect of some substance or substances essential for the maintenance of the normal nutrition of nervous tissues”, beriberi could be prevented “by substituting ... a rice in which the polishing process has been omitted or carried out to a minimal extent, or by the addition to a white rice of articles rich in those substances [such as rice polishings] in which such white rice now makes default”.⁶² Fraser told the inaugural meeting of the Far Eastern Association of Tropical Medicine (FEATM) at Manila in 1910: “our researches have conclusively shown that beriberi can be prevented by the use of unpolished rice and as surely produced by the use of highly polished rice.”⁶³ Although it took many years before the role of vitamin B1 was fully established,⁶⁴ by 1914 the longstanding problem of beriberi appeared largely to have been solved. To the minds of many in the international medical community, the cause of the disease was now clear, as was

⁵⁶ W Leonard Braddon, *The cause and prevention of beri-beri*, London, Rebman, 1907, p. 1.

⁵⁷ *Report of the Philippines*, Geneva, League of Nations, 1937, pp. 20–1.

⁵⁸ Vedder, op. cit., note 11 above, p. 15.

⁵⁹ Victor G Heiser, ‘Beri-beri’, *Far Eastern Association of Tropical Medicine: comptes rendus des travaux du troisième congrès biennal*, Saigon, A Portail, 1914, pp. 369–73, on p. 371.

⁶⁰ W A Sawyer, ‘Advantages of nation-wide and inter-national organization for disease control, with special reference to hookworm disease and beriberi’, in A L Hoops and J W Scharff (eds), *Far Eastern Association of Tropical Medicine: transactions of the*

fifth biennial congress, London, John Bale, Sons and Danielsson, 1924, pp. 183–92, on p. 192.

⁶¹ Kenta Omori, ‘Studies on the cause and treatment of beri-beri in Japan’, *Far Eastern Association of Tropical Medicine: transactions of the sixth biennial congress*, Tokyo, Kyorinsha, 2 vols, 1925, vol. 1, pp. 183–204, on p. 203.

⁶² H Fraser and A T Stanton, ‘The etiology of beri-beri’, *Transactions of the Society of Tropical Medicine and Hygiene*, 1910, 3: 257–69, pp. 266–7.

⁶³ Henry Fraser, in ‘Discussions on the papers’, *Philippine J. Sci.*, 1910, 5: 137–44, p. 141.

⁶⁴ Carpenter, op. cit., note 8 above, chs 7, 8.

the means by which it could effectively be countered. In actuality, though, the controversy was far from over.

In the presentation and evaluation of evidence for the white-rice, vitamin-deficiency theory of beriberi and in the search for positive measures to eradicate the disease, one international organization—the Far Eastern Association of Tropical Medicine—played a particularly prominent role. From its foundation in 1908 to its dissolution in 1938, the FEATM provided a unique platform for research on tropical diseases and, despite political and cultural divisions, for the exchange of professional expertise across the tropical and sub-tropical regions of South, Southeast and East Asia.⁶⁵ Over its thirty-year existence the FEATM held ten international congresses, which, at their height, attracted as many as 500 delegates from more than twenty countries and colonial dependencies. Most delegates (including those from British India) were high-ranking government medical officers, and the congresses were treated by the hosts as prestigious state occasions. Initiated by the Americans in the Philippines and in part overlapping with the operations of the Rockefeller Foundation, the organization became a vehicle for a new trans-national spirit of collaboration and information-exchange in the governance of the Asian tropics. However, the effectiveness of the FEATM was inhibited by the lack of permanent headquarters, the insecurity of its funding and the absence of an ongoing executive to implement its recommendations. Since no one imperial power or international body was willing to bankroll its activities, the FEATM lacked the authority that later accrued to the League of Nations’ Health Organization or, after the war, to the World Health Organization.

Although many different health issues were taken up by the FEATM, beriberi occupied a conspicuous place from the outset. It was a disease that occurred virtually throughout the region covered by the FEATM, the “Far East” of its title being almost coterminous with the world’s main paddy-producing and rice-consuming countries. A further reason why beriberi figured so prominently was due to timing, for it was precisely in 1908–10, at the very time the association came into being, that the breakthrough was made in the scientific understanding of beriberi. Although many research papers on the disease were published in Europe, the initial findings of Braddon, Fraser and Stanton in Malaya, as later the views of British India’s Robert McCarrison and John Megaw, were widely circulated and discussed in the region through presentations made to the FEATM congresses. Some participants further believed that the FEATM was an appropriate agency to combat the disease as well as to debate its nature and cause. Heiser remarked in 1911 that the advances made in the past year, by Fraser and Stanton in particular, “in placing the etiology of beriberi upon a scientific basis” had “proceeded sufficiently to warrant the inference that prophylactic medicine has the knowledge at its command to place this scourge among the preventable diseases”.⁶⁶ At Manila in 1910, Francis Clark, Hong Kong’s medical officer, moved a

⁶⁵ For the FEATM’s aims and composition, see J W Scharff (ed.), *A report of the fifth congress of the Far Eastern Association of Tropical Medicine*, Singapore, Government Printing Office, 1923, p. 24. The FEATM and its engagement with the beriberi issue are discussed more fully in David Arnold, *Tropical governance: managing health in monsoon Asia, 1908–1938*, Asia Research Institute, Singapore, Working Papers Series 116,

<http://www.ari.nus.edu.sg/publications/ariwps.htm>.

⁶⁶ Victor G Heiser, ‘Practical experiences with beriberi and unpolished rice in the Philippines’, *Philippine J. Sci.*, 1911, 6: 229–33, p. 229. Vedder echoed this view: “We are now in a position to prevent the disease in any country that can and will follow our advice just as surely as we can prevent smallpox and yellow fever”: Vedder, op. cit., note 11 above, p. iii.

resolution which stated that there was now “sufficient evidence” to show that “beriberi is associated with the continuous consumption of white (polished) rice as a staple article of diet” and urged governments to act. He believed the resolution would “enable us to take early steps to protect the natives under our care from a disease which is responsible for much suffering and many deaths”.⁶⁷ Although the resolution provoked some dissent even at the time (and was passed after many delegates had left),⁶⁸ the claim that beriberi was a “disorder of nutrition” principally caused by “a diet of which over-milled rice forms the staple”,⁶⁹ became a doctrine repeated at subsequent congresses held at Hong Kong, Saigon, Batavia and Singapore. Inspired by the apparent success of interventionist public health measures in the Philippines,⁷⁰ Heiser sought to go further, calling for an international agreement to tax white rice so as to drive it from the market-place or to confine its use to only those rich enough to afford it and likely, anyway, to enjoy more varied diets. In support of this strategy, he claimed that “the solution of the beri-beri problem would probably save more human lives and at the same time be of greater economical advantage than any one health measure proposed in modern times”.⁷¹

The British India Response

The white-rice theory of beriberi and the stance taken by the FEATM resolutions on the subject placed the Government of India (and many other colonial and national governments across monsoon Asia) in a quandary. From the outset British India had been an enthusiastic participant in the FEATM, sending delegates to Manila in 1910 and some of its most senior medical officers attended subsequent meetings.⁷² Until the late 1930s, when war loomed in the East, the government viewed the association as an important vehicle for the presentation of innovative research and a way of publicizing India’s “outstanding position where health problems are concerned”.⁷³ It regarded the holding of the seventh FEATM congress at Calcutta in December 1927 as a highly prestigious occasion for which it was anxious to attract wide international participation, and as a high profile event that would demonstrate its continuing commitment to medical research.⁷⁴ However, India’s growing dependence on rice imports, principally from Burma, the increasing demand for milled rice (which by the 1930s fed an estimated 70 per cent of rice-eaters in the Madras Presidency alone) and political fears of unrest if attempts were made to restrict the sale of white rice, along with its longstanding commitment to free trade and extreme reluctance to meddle with the lucrative rice trade, made the government unsympathetic to the radical interventionism proposed

⁶⁷ Francis Clark, ‘Discussions on the papers’, *Philippine J. Sci.*, 1910, 5: 144.

⁶⁸ Richard P Strong and B C Cowell, ‘The aetiology of beriberi’, *Philippine J. Sci.*, 1912, 7: 271–414, pp. 289–90.

⁶⁹ Scharff (ed.), op. cit., note 65 above, p. 13.

⁷⁰ Warwick Anderson, *Colonial pathologies: American tropical medicine, race, and hygiene in the Philippines*, Durham, Duke University Press, 2006; Victor Heiser, ‘Unsolved health problems peculiar to the Philippines’, *Philippine J. Sci.*, 1910, 5: 171–8.

⁷¹ Heiser, op. cit., note 59 above, p. 372.

⁷² A Hooton (ed.), *Report on the first biennial meeting of the Far Eastern Association of Tropical Medicine*, Bombay, Government Central Press, 1910.

⁷³ M Yeatts, Education, Health and Lands, 23 June 1938, Education Health and Lands (Health), 34–3, 1938, National Archives of India, New Delhi.

⁷⁴ L/E/7/1504, IOR; RG 1.1, series 600A, box 3, file 15, Rockefeller Archives, Tarrytown, New York; *Seventh congress of the Far Eastern Association of Tropical Medicine souvenir: the Indian Empire*, Calcutta, Thacker, 1927.

by Heiser and the FEATM resolutions.⁷⁵ In British India’s decentralized health system, it was left to the provinces to issue their own statements about the possible linkages between milled rice and beriberi. In 1923 the Government of Madras, one of the provinces most critically involved, offered the advice that the “best way to avoid beri-beri” was “to use a varied diet ... and not to restrict the diet too rigidly to machine-milled rice”.⁷⁶ But not until the Second World War did India’s governments move to regulate the rice trade and, for reasons of war-time shortages rather than public-health priorities, restrict rice-milling.⁷⁷

It was not state policy alone that prevented a more active campaign against rice-milling. The medical profession in India also, in the main, regarded interventionism as unwarranted. But there was a broad spectrum of opinion, even within the elite IMS. One response was incredulity, disbelief that what was claimed as the effects of white rice elsewhere corresponded with what India knew of beriberi. In Madras, Bengal and Burma (until 1935 a province of British India) the number of rice mills had grown rapidly since 1910 without any concurrent upsurge in beriberi.⁷⁸ The disease still appeared to be a relatively minor cause of mortality in British India, averaging 257 deaths a year between 1924 and 1933, and, Burma apart, mainly concentrated, as before mechanized milling began, in the Northern Circars (where deaths averaged 66 a year over the same period).⁷⁹ In most jails in southern and eastern India prisoners were fed a diet consisting largely of rice and yet, unlike in Southeast Asia, they seldom developed beriberi. Was it possible, asked the superintendent of Dacca Central Jail, that “the organism of beri-beri only attacks rice from the further East?”⁸⁰

Two further factors militated against ready acceptance of the white-rice theory in India. One was that, whereas in the early nineteenth century beriberi was thought to be mostly confined to south India or to south Indians abroad, by 1900 the beriberi literature principally focused on Southeast Asia where the Chinese figured as its main victims. The institutions in Singapore in which beriberi occurred were populated almost entirely by Chinese; Chinese labourers, prisoners and asylum inmates were those most commonly discussed (and photographed) in the medical literature.⁸¹ While it was denied that beriberi was race specific (and might affect Europeans),⁸² in the racialized discourse of late

⁷⁵ N Gangulee, *Health and nutrition in India*, London, Faber and Faber, 1939, p. 136.

⁷⁶ Madras Government Order 639, Local Self-Government (Public Health), 14 Apr. 1923, Tamil Nadu Archives, Chennai. For local discussion of the FEATM resolutions, see GO 1034, LSG (PH), 26 June 1923, TNA.

⁷⁷ Henry Knight, *Food administration in India, 1939–47*, Stanford University Press, 1954, chs 12, 14; Williams, op. cit., note 8 above, pp. 247–8.

⁷⁸ In 1912 there were 68 registered rice-mills in Madras and 245 in Burma, rising by 1938 to 342 in Madras, 411 in Bengal and 683 in Burma. Rice-milling was one of India’s leading industries, employing 72,000 workers by the late 1930s. *Reports on the working of the Indian Factories Act* for the relevant years and provinces.

⁷⁹ *Annual Report*, op. cit., note 55 above, p. 135; *Annual report of the Public Health Commissioner with the Government of India, 1933*, vol. 1, p. 83. The

incidence of beriberi in Burma was in fact rather different from that in British India: see Judith L Richell, *Disease and demography in colonial Burma*, Singapore, NUS Press, 2006, ch. 6. Mortality from beriberi in the Philippines was far higher than in the Northern Circars despite similar population numbers: Williams, op. cit., note 8 above, p. 250.

⁸⁰ Buchanan, op. cit., note 43 above, p. 578.

⁸¹ “There is hardly a place or district where Chinese are found in which beri-beri does not occur”: C W Daniels, *Observations in the Federated Malay States on beri-beri*, London, E G Berryman, 1906, p. 46. See also William Fletcher, ‘Rice and beri-beri’, *J. Trop. Med. Hygiene*, 1909, **12**: 127–35, photographs following p. 134.

⁸² Aldo Castellani and Albert J Chambers, *Manual of tropical medicine*, 3rd ed., New York, William Ward, 1920, p. 1680; Dubruel, op. cit., note 28 above, p. 59; Scheube, op. cit., note 29 above, pp. 196–7.

nineteenth- and early twentieth-century tropical medicine it was typically on the Chinese body that beriberi was inscribed. Medical writers in India often accepted this racialization of the disease by remarking that “true” beriberi was only to be found there (as in Calcutta) among the Chinese community, a radical departure from earlier, south Indian studies.⁸³

A second factor, which seemed further to distance India from Southeast Asia, was allied to this. Braddon showed in his research in Malaya that while the Chinese suffered heavily from beriberi Tamil labourers were almost entirely immune. His explanation was that that the Chinese consumed “uncured” milled rice, cooked after de-husking, while Indians ate “cured” (parboiled) rice, soaked and steamed before milling.⁸⁴ This explanation, amplified by later research which showed that parboiling rice retained sufficient thiamine to prevent beriberi,⁸⁵ helped account for the fact that “true” beriberi was rare in India, for among most castes and communities in Madras and Bengal rice was cooked and eaten only after parboiling, whether it was milled locally or imported, already de-husked, from Burma. This further underscored Indian exceptionalism, for almost nowhere else in Asia was rice eaten in this way: indeed, most other rice-consumers regarded the smell and taste of parboiled rice as repugnant.⁸⁶ The main reason for the concentration of beriberi in the Northern Circars was, as McCarrison suggested in 1924 and as later research confirmed, that here, exceptionally, was a locality where “uncured” rice was eaten for most of the year and by poor people with little access to nutritive foods—an anomaly within an India that was itself at variance with other rice-eating societies.⁸⁷

But the principal reason for scepticism about the white-rice/vitamin-deficiency thesis in India was that many influential figures in the medical establishment clung to the belief that beriberi was better explained by “toxi-infection” of the kind that Braddon had mooted in 1907, before the role of vitamins had been identified. According to this view, beriberi was spread by a “poison” on the surface of the rice grain and was not due to any deficiency within it. Rice was the agent, not the cause. Despite the dominance of the nutrition-deficiency thesis in the early FEATM congresses, this bacterial explanation, influenced by germ theory and the Pasteurian tradition, was widely held not only in India but also by other opponents of the milled rice theory or by those countries, like Japan and Thailand, which were alarmed by the cultural as well as economic implications of seeking to prohibit so popular and prestigious a commodity as white rice.⁸⁸ Although historians normally note only the FEATM resolutions in favour of the white-rice theory,⁸⁹ the organization was also, throughout the 1920s, one of the main platforms for the “intoxication” view. The French were goaded by the 1910 Manila resolution into holding their own beriberi commission, which asserted that beriberi could not be unequivocally identified with white rice, nor could it be conclusively proved that the polyneuritis in chickens (used as the experimental basis for beriberi research) was identical with human beriberi. The disease, the commission claimed, occurred among people who

⁸³ Buchanan, op. cit., note 43 above, p. 577; Greig, op. cit., note 17 above, p. 5.

⁸⁴ Braddon, op. cit., note 56 above, pp. 150–98.

⁸⁵ W R Aykroyd and B G Krishnan, ‘Rice diets and beriberi’, *Indian J. Med. Res.*, 1941, **29**: 551–5, p. 551.

⁸⁶ Williams, op. cit., note 8 above, pp. 113–14.

⁸⁷ Robert McCarrison and Roland V Norris, ‘The relationship of rice to beri-beri in India’, *Indian Medical Research Memoirs*, no. 2, 1924, pp. 3–4; Aykroyd and Krishnan, op. cit., note 85 above, pp. 553–5.

⁸⁸ Scharff (ed.), op. cit., note 65 above, pp. 33–43.

⁸⁹ For example, Williams, op. cit., note 8, pp. 48–9.

did not eat milled rice (or even rice at all) and might result from a toxin absorbed by rice after milling.⁹⁰

Although American, Dutch and British researchers generally endorsed what had by 1912 become established as the vitamin-deficiency explanation, scientific opinion did not simply divide along national lines: the British in India as well as the French in Indochina appeared divided, and it was a Japanese delegate who asked of the Tokyo congress in 1925, “Who can say that the theories of intoxication and infection have entirely failed?”⁹¹ To American chagrin, the FEATM decided that an international convention to control beriberi through restrictions on milling and the rice trade was for the present unattainable. Instead the organization urged governments to pursue their own research in the belief that a consensus would eventually emerge.⁹² A Dutch delegate summed up the prevailing mood when he observed in 1925 that there were “great difficulties” in trying to impose a policy: “Very little can be done internationally—perhaps nothing at all.”⁹³ By the time the FEATM met at Calcutta in 1927 “interest in this problem” was already “slacking”.⁹⁴

In India the principal opponent of the milled-rice hypothesis was J W D Megaw of the Indian Medical Service. Successively Director of the Calcutta School of Tropical Medicine (1921–28), Inspector-General of Civil Hospitals in the Punjab (1928), and Surgeon-General in Madras (1928–30), Megaw was Director-General of the IMS from 1930 to 1933.⁹⁵ Almost throughout his Indian career, from 1910 to 1930, Megaw argued that epidemic dropsy was “closely related to, if not identical with, beri-beri” and hence that—in India—the problem of beriberi was equally one of epidemic dropsy.⁹⁶ Since dropsy had caused considerable sickness and mortality in eastern India in 1877–8, 1909–10 and 1926,⁹⁷ this elevated the idea of epidemic beriberi to a position of unprecedented importance in India. It also held out the prospect of research in India revealing the disease in an entirely new light and to the benefit of the international medical community.⁹⁸ Claiming in 1921 that no one had yet “brought forward convincing evidence of the distinction between the two diseases” (beriberi and epidemic dropsy), Megaw argued that scientists had accepted the vitamin-deficiency

⁹⁰ MM. Bréaudat, Le Dauteuc, Jeanselme, Kermorgant, Marchoux and Potterin, ‘Rapport sur le bérubéri’, *Bulletin de la Société de Pathologie Exotique*, 1911, 4: 575–604. For the French investigation of both the “toxi-infection” and “alimentary” theories, see P-Noël Bernard, *Les Instituts Pasteur d’Indochine*, Saigon, A Portail, 1922, pp. 154–8.

⁹¹ Akira Fujinami, ‘Historical review of research on kakke in Japan’, FEATM, op. cit., note 61 above, vol. 1, pp. 11–17, on p. 17.

⁹² Scharff (ed.), op. cit, note 65 above, p. 43.

⁹³ C D de Langen, ‘The international control of beri-beri’, FEATM, op. cit., note 61 above, vol. 1, pp. 61–85, on p. 85.

⁹⁴ O Deggeller, 18 Nov. 1927, L/E/7/1504, IOR.

⁹⁵ Megaw had strongly held views on other matters too, including population control: David Arnold, ‘Official attitudes to population, birth control and reproductive health in India, 1921–1946’, in Sarah

Hodges (ed.), *Reproductive health in India: history, politics, controversies*, Hyderabad, Orient Longman, 2006, pp. 22–50.

⁹⁶ J W D Megaw, ‘Notes on cases of the “epidemic dropsy” type of beri-beri at the Presidency General Hospital, Calcutta’, *Indian Med. Gaz.*, 1910, 45, offprint, p. 8, Megaw Collected Papers, Wellcome Library, London; see also Megaw, ‘The beriberi and epidemic dropsy problem’, *Indian Med. Gaz.*, 1923, Megaw Collected Papers.

⁹⁷ In Calcutta alone in 1909–10 there were 1,581 reported cases of epidemic dropsy with 368 deaths: E D W Greig, *Epidemic dropsy in Calcutta (final report)*, Calcutta, Superintendent of Government Printing, India, 1912, p. 9.

⁹⁸ J W D Megaw, ‘Epidemic dropsy: its bearing on the beri-beri problem’, in J Cunningham (ed.), *Far Eastern Association of Tropical Medicine: transactions of the seventh congress*, 3 vols, Calcutta, Thacker, 1928, vol. 3, pp. 349–60, on p. 349.

theory “with wonderful”—in his view ill-founded—“unanimity”.⁹⁹ The medical profession, he later added, “appears to have accepted a view of beriberi which has not been established on a sound basis and which is inconsistent with many observed facts”.¹⁰⁰ There had been no change in the diet of the people of Calcutta—no sudden switch from hand-pounded to milled rice—to account for the 1909 epidemic, nor were many of its victims poor and malnourished. The evidence was “much more suggestive of an intoxication than of a food deficiency”.¹⁰¹

Megaw questioned (as did many of his French contemporaries) the link between polyneuritis in fowls and human beriberi, and with a side-swipe not only at researchers in Malaya but also at McCarrison’s work at Coonoor, Megaw asserted: “we have been relying too much on laboratory findings and not paying enough attention to the disease as it occurs among human beings”. For him the real evidence of the disease was to be found among its victims, and, encouraged by dissent in the FEATM,¹⁰² he called for new research that would put aside “all preconceived ideas on the subject”.¹⁰³ Although the idea of epidemic beriberi went back to Chevers, Megaw was more obviously inspired by Braddon, whose early research he continued to defend long after others deemed it outdated.¹⁰⁴ As Director of the Calcutta School of Tropical Medicine in the 1920s Megaw gave fresh impetus to the “intoxication” theory, promoting research designed to show that beriberi erupted epidemically when rice became damp or had been badly stored and so able to absorb and transmit the “toxi-infection”. In contradiction of the white-rice theory, Megaw asserted that milling was beneficial since it meant that newly husked rice was available for consumption rather than being stored for long periods in a vulnerable state.¹⁰⁵

Heiser and others might ridicule such claims,¹⁰⁶ but Megaw was a senior figure in the IMS, who used the FEATM congresses and other public platforms to defend his views and to argue that it was still “an open question” whether beriberi was one disease with a single cause or “a group including several disease entities which we are unable to differentiate from each other”.¹⁰⁷ Since his stance on beriberi proved no bar to his becoming Director-General of the IMS, the highest position to which a member of the service could aspire, one can only assume that the Government of India was not unwilling to

⁹⁹ Megaw, ‘Presidential Address’, Medical Research Section, Eighth Indian Science Congress, *Transactions of the Asiatic Society of Bengal*, 1921, Megaw Collected Papers, p. ccviii.

¹⁰⁰ J W D Megaw, ‘The beriberi problem’, in FEATM, *Transactions of the fifth biennial congress*, Megaw Collected Papers, p. 14.

¹⁰¹ Megaw, op. cit., note 99 above, pp. ccviii–ccix. This was a longstanding view in Calcutta, where the city’s health officer claimed that the “arguments against the food theory are very strong”: T Frederick Pearse, ‘Report of the Health Officer on the outbreak of beri-beri or epidemic dropsy’, *Report of the municipal administration of Calcutta, 1909–10*, p. 101.

¹⁰² At Tokyo in 1925, where he attacked the “dogma that beriberi could and would be controlled by the prohibition of over-milled rice”, Megaw claimed

that there “seemed to be just as many who were dissatisfied with the orthodox vitamine [sic] deficiency view as those who declared their adherence to that view”: J W D Megaw, ‘The sixth congress of the Far Eastern Association of Tropical Medicine’, *Indian Med. Gaz.*, 1926, **41**: 349–56, p. 349.

¹⁰³ Megaw, op. cit., note 99, above, p. ccix.

¹⁰⁴ Megaw, op. cit., note 100 above, p. 11.

¹⁰⁵ *Ibid.*, p. 11; Megaw in *Annual report of the Calcutta School of Tropical Medicine, 1926*, p. 31.

¹⁰⁶ Victor Heiser’s Diary, Tokyo, 16 Oct. 1925, RG 12.1, RAC.

¹⁰⁷ Megaw, op. cit., note 100 above, p. 3. As late as 1930 Megaw held that beriberi was the name given to “a disease group”, not a single disease: Leonard Rogers and John W D Megaw, *Tropical Medicine*, Philadelphia, P Blakiston’s Son, 1930, pp. 438–9.

have as its chief adviser a man who was openly sceptical about the detrimental effects of rice-milling. As late as 1930 its Public Health Commissioner published a report on beriberi in Madras (prepared while Megaw was provincial surgeon-general) which made no mention of vitamins but dwelt instead on human susceptibility to a toxin possibly located in the soil and the “highly significant” correlation between rainfall and peak incidence of the disease.¹⁰⁸

Beriberi as Exemplar

The most celebrated response to the “beriberi problem” in India came not from Megaw but from his IMS colleague Robert McCarrison and his work at the Nutritional Research Laboratories at Coonoor in south India from 1914 until his eventual retirement in 1935. McCarrison’s contribution to the investigation of beriberi and nutrition deficiency disease in India has been extensively noted, and it is unnecessary to repeat here what has already been said about his work.¹⁰⁹ However, in the context of the history of beriberi research in India several points need to be made.

Although McCarrison would appear to represent a new departure in British Indian discussions of beriberi by identifying the disease specifically with problems of deficient nutrition, his line of enquiry and the manner in which he presented his findings echoed earlier research in India. This was not so much the nineteenth-century debates over beriberi, with which McCarrison had little engagement, as the work of an earlier IMS officer, David McCay, Professor of Physiology at Calcutta Medical College, on prison diets in eastern and northern India shortly before the First World War.¹¹⁰ Even though McCay’s research predated the knowledge of vitamins and referred instead to “protein deficiency” and the “mal-assimilation of nitrogenous foodstuffs”, McCarrison saw in it an important precedent for his own work.¹¹¹ Not least significant was McCay’s demonstration that poor nutrition was reflected in physiological differences between different Indian “races” and their diets, especially between the wheat and dairy diets of the Sikhs, Pathans and other “martial races” of the northwest and the “poor rice” diets of the “non-martial” Bengalis, Biharis and Madrasis.¹¹² This articulation not only made use of the language and concepts of the “martial races theory”, one of the dominant ideas of imperial governance in India; it also drew upon an extensive European medical discourse in India, going back to the early nineteenth century, in which rice diets were condemned as insufficient to support physical and mental well-being. Chevers, for instance, had believed, with evangelical zeal, that the only way to rescue Bengalis from their physical incapacity was to stop them from growing rice and make them take up a “higher” food staple.¹¹³

¹⁰⁸ *Annual Report*, op. cit., note 55 above, pp. 136–7. There are clear echoes here of the anti-contagionist opposition by IMS officers to a bacteriological explanation for cholera and insistence upon the effects of soil, climate and other “local conditions”: Harrison, op. cit., note 53 above, pp. 112–15.

¹⁰⁹ Sinclair (ed.), op. cit., note 52 above.

¹¹⁰ D McCay, *Investigations into the jail dietaries of the United Provinces*, Calcutta, Superintendent of Government Printing, 1912.

¹¹¹ R McCarrison, ‘The nutritive value of wheat, paddy, and certain other food-grains’, *Indian J. Med. Res.*, 1927, **14**: 631–9, pp. 631–2.

¹¹² McCay, op. cit., note 110 above, ch. 9.

¹¹³ Chevers, op. cit., note 44 above, pp. 568–9. Although Chevers’ account of deficient rice diets was separate from his account of epidemic beriberi, later authors tended to conflate the two: e.g., Greig, op. cit., note 17 above, pp. 6–8.

The attack on rice, and its association with negative racial stereotypes, made it easier for McCarrison to popularize his findings on beriberi well beyond medical circles and among Indians as well as Europeans. Just as Manson tried thirty years earlier to make the disease visible through his first-hand account of beriberi patients, so McCarrison, to far greater public effect, used the results of feeding laboratory rats with a healthy “Sikh” or poor “Madras” diet to give clear ocular testimony to what was actually a complex scientific and sociological issue. Those who visited his laboratories in the 1920s, or studied the photographs used to illustrate his experiments, were deeply impressed.¹¹⁴ His claim, made to India’s Royal Commission on Agriculture in 1926 that of all the disabilities from which the Indian masses suffered “malnutrition is perhaps the chief”, resonated with a powerful sense among many middle-class Indians of their physical weakness and the need to build a healthier Indian nation.¹¹⁵

Although McCarrison’s work at Coonoor was originally dubbed the “beriberi inquiry”, it approached the disease less from an international, pan-tropical angle than from a specifically Indian standpoint. In the wake of the FEATM resolutions condemning milled rice, McCarrison reiterated the point, clearly not unwelcome to the government,¹¹⁶ that beriberi in India long predated mechanized milling and remained largely confined to the Northern Circars, where peculiar local circumstances accounted for its persistence. While eschewing the idea of epidemic dropsy as a form of beriberi, McCarrison shared the scepticism of many medical officers and researchers in India about the way in which “the beriberi problem” had been presented internationally as a disease directly, even exclusively, related to white rice, arguing that before any action was taken against milled rice it was necessary to “be sure that its application would be attended with the desired results”. As the Northern Circars showed, the statement that “beri-beri appears when decorticated rice is used for any length of time” did not “always apply in India”.¹¹⁷

It was also apparent from McCarrison’s research that, while beriberi was not a major health hazard outside the Northern Circars, it served to exemplify the far wider impact of nutritional deficiency diseases in India and the urgent need to rectify this not just through improved diets but also, more holistically, through reformed agricultural practices. The implicit strategy of conducting research on beriberi to exemplify the problem of nutritional poverty while simultaneously demonstrating the localization of that disease within

¹¹⁴ N Gangulee, *The Indian peasant and his environment*, London, Oxford University Press, 1935, p. 116. See also R McCarrison, ‘A good diet and a bad one: an experimental contrast’, *Indian J. Med. Res.*, 1927, **14**: 649–54; *idem*, ‘Problems of nutrition in India’, in Sinclair (ed.), *op. cit.*, note 52 above, pp. 265–78.

¹¹⁵ For the national concern for nutrition, see, Gangulee, *op. cit.*, note 75 above; U N Brahmachari, ‘The role of science in the recent progress of medicine’, Presidential Address, *Twenty-third Indian Science Congress, Indore, 1936* (n.p.d.), p. 3; editorial, ‘Nutritional research in India’, *Calcutta Med. J.*, 1942, **39**: 67–9. For all its local referents, McCarrison’s research formed part of the wider recognition in the 1920s and 1930s of the inadequacy

of diets across the British empire: Michael Worboys, ‘The discovery of colonial malnutrition between the wars’, in David Arnold (ed.), *Imperial medicine and indigenous societies*, Manchester University Press, 1988, pp. 208–25.

¹¹⁶ Speaking at the FEATM congress at Singapore in 1923, S R Christophers, on behalf of the Government of India, cited McCarrison’s research as indicating that, since a correlation between rice milling and beriberi was still unproven, “it is not thought that legislation to prevent the over-milling of rice would effect the purpose for which it is intended”: Scharff (ed.), *op. cit.*, note 65 above, p. 37.

¹¹⁷ McCarrison and Norris, *op. cit.*, note 87 above, p. 33.

what McCarrison called its “endemic haunts”,¹¹⁸ was further developed by McCarrison’s successor at Coonoor, W R Aykroyd, and by B G Krishnan and other Indian co-workers,¹¹⁹ though without the resort to the “martial races” stereotypes McCay and McCarrison employed. It also became possible after both Megaw and McCarrison had left India to reassess the nature and scale of the beriberi problem. As late as 1930 it had been maintained that infantile beriberi of the kind described by the Americans in the Philippines twenty years earlier was “unknown” in India.¹²⁰ However, research in the Northern Circars showed this to be incorrect and that higher than expected levels of mortality among infants three to four months old indicated the likely effects of this form of beriberi.¹²¹

The exemplary use of beriberi as the emblem for India’s wider nutritional disorders was far-reaching. Before about 1910 beriberi, despite its supposedly vernacular name, was seldom discussed in Indian medical treatises or regarded as comparable to diseases like malaria and diabetes which appeared to constitute major threats to Indian well-being.¹²² But, spurred on by the 1909 outbreak of epidemic dropsy in Bengal and by the findings of McCarrison and Aykroyd, beriberi became, during the interwar period, a source of widespread, if principally middle-class, Indian concern. When the “beriberi problem” was debated at the Calcutta FEATM congress in 1927, newspaper headlines proclaimed that beriberi was now “curable by diet”.¹²³ Indian newspapers and medical journals, trading on a new-found obsession with vitamins and healthy diets, carried advertisements for medicines, diet supplements and tonics that claimed to cure or prevent beriberi, even though few middle-class households were likely to experience a disease whose incidence had always been concentrated among the poor.¹²⁴ Indian medical researchers developed an expertise in beriberi and related conditions, and, from having been more in the western mind than the Indian eye, beriberi and the new nutritional culture in which it took pride of place became the subject of frequent articles and editorials in the Indian medical press.

By 1942, when the wartime crisis brought nutritional research to a temporary halt, beriberi had acquired a public as well as professional prominence in India it had never previously enjoyed. The idea of beriberi had become a powerful exemplar of India’s poor diets, its nutrition-deficiency diseases, and even the poverty of the agriculture that fed and supported the great majority of its population. Beriberi epitomized a desire for national self-improvement as well as individual well-being. For the first time, the government began to face calls from Indian doctors for milled rice to be banned as a danger to the nation’s health.¹²⁵

However, the exemplary role, enjoyed by beriberi since the 1920s, proved short-lived. In common with many other parts of monsoon Asia, and despite increased awareness of

¹¹⁸ *Ibid.*, p. 4.

¹¹⁹ Aykroyd and Krishnan, *op. cit.*, note 85 above, p. 551.

¹²⁰ *Annual report*, *op. cit.*, note 55 above, p. 136.

¹²¹ W R Aykroyd and B G Krishnan, ‘Infantile mortality in the beriberi area of the Madras Presidency’, *Indian J. Med. Res.*, 1941, **29**: 703–8. On infantile beriberi, see Vedder, *op. cit.*, note 11 above, pp. 240–64; Williams, *op. cit.*, note 8 above, pp. 81–94.

¹²² For two early Indian works on the subject, see Sarat Chandra Ghose, *Beri-beri: its causation,*

prevention and homoeopathic treatment, Calcutta, A C Dutt, 1910; S M Varis, *A study of malaria and beri-beri*, Allahabad, Pioneer Press, 1912.

¹²³ *Pioneer Mail*, 16 Dec. 1927, p. 11.

¹²⁴ Alembic Chemical Works, Baroda, advertised the “Alembic Elixir Haemoglobin” for “deficiency diseases like sprue, rickets [and] beri-beri”: *Calcutta Med. J.*, 1942, **39**: xviii; Glaxo marketed “Berin” for the treatment of beriberi, anorexia and neuritis: *Calcutta Med. J.*, 1943, **40**: xii.

¹²⁵ For example, B C Guha, ‘Editorial: Better nutrition’, *Indian Med. J.*, 1938, **32**: 593–6, p. 593.

infantile beriberi, the disease was in apparent decline by the 1960s. Improved nutrition, health propaganda and heightened public awareness may all have contributed to this, but perhaps beriberi had simply become subsumed once more within the broad category of nutritional diseases. The characteristic symptoms of the disease, so evident to Manson and McCarrison, seemed decreasingly visible, one writer in the early 1980s remarking that the “classical form” of beriberi was now rarely seen and “almost non-existent”.¹²⁶

¹²⁶ P K Shukla, *Nutritional problems of India*, New Delhi, Prentice-Hall, 1982, pp. 133, 136.