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Scurvy on sea and land: political economy and natural history, c. 1780–c. 1850

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From the late eighteenth century, the ways in which scurvy was understood changed in consequence of the abandonment of humoral pathology and the adoption of a new causal framework informed by nervous physiology. Although there was some narrowing of the etiological framework around dietary deficiency in the wake of the navy's success with the issue of citrus juices, this was rarely to the exclusion of predisposing causes such as fatigue, weather and flagging spirits. Within the navy, the persistence of a multi-factoral framework was relatively unproblematic, for the standard issue of citrus juices and improvements in victualling occurred at the same time as other important reforms in naval health. But outside the navy it was a different matter. In other institutional settings, the continuing belief in the importance of factors other than diet created tensions between medical officers and administrators who found such inclusive views politically inconvenient.

After a brief survey of the principal changes in the physiology of scurvy, this article examines how the problem of scurvy was understood, not only in the navy but also in the armies of the East India Company and in British prisons. These were not the only contexts in which scurvy caused concern, but they serve to illustrate the fact that it remained a complex and controversial disease. The article shows how different medical cultures and institutional imperatives took the natural history of scurvy in different directions.

Keywords: convicts; diet; East India Company; political economy; prisons; Royal Navy; scurvy

Introduction

During the seventeenth and eighteenth centuries, scurvy came to prominence as a disease of sailors and soldiers. There was general agreement that it was caused by putrefaction of the humours, the corruption of which was often attributed to a diet of noxious foods or prolonged exposure to cold and damp. By the late eighteenth century, however, the way in which scurvy was understood began to change, in common with many other diseases. The humoral theory was gradually abandoned and new insights were incorporated into medicine from nervous physiology and pneumatic chemistry. Utilising these theories, medical authors began to portray scurvy as a disease of dietary deficiency, even though few could agree on what precisely was missing. From the mid-1790s, the view that scurvy was due to defective diet was bolstered by practical experience. The universal provision of citrus juices for naval vessels brought a dramatic reduction in cases of scurvy and gave the British fleet an advantage over its enemies. For this reason, the 1790s are often regarded as a watershed in the health of the navy and in the history of scurvy

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itself. Thereafter, we are told, medical interest in the disease began to decline.¹ It was as if the problem of scurvy had been solved.

The reality was very different, for scurvy remained a complex and controversial disease through to the middle of the nineteenth century and beyond. This article offers an explanation of why such a diversity of views persisted beyond 1790 and why it was not until the mid-nineteenth century that a loose and rather fragile consensus began to emerge. The reasons, I suggest, owe more to prevailing doctrines of political economy and administrative expediency than to the force of scientific logic. After a survey of changing views on the physiology of scurvy during the eighteenth century, this article examines how the problem of scurvy was understood in the Royal Navy and in two other contexts in which it presented a problem: the territories of British India and the new penitentiaries constructed in Britain. These were not the only settings in which scurvy gave cause for concern, but they suffice to demonstrate that its etiology remained unstable and that attempts were made to order the disease in different ways.

A multifaceted disease: scurvy in the eighteenth century

Scurvy did not suddenly appear in the eighteenth century, but it became far more common at that time. Ships were able to remain at sea for longer because of the discovery of longitude and other aids to navigation. The global wars and trading interests of the eighteenth century also meant that many vessels were undertaking such voyages. Although scurvy was by no means the principal medical problem facing the Royal Navy – especially by comparison with typhus or ‘ship fever’ – it often combined with other diseases to reduce the morale and efficiency of crews.² Nor was scurvy solely a problem affecting mariners. It was widely recognised as a land disease, occurring among soldiers on active service and among all peoples subject to famine and deprivation. However, scurvy was not yet regarded as a disease of deficiency in any sense of the term. It was not simply that individuals differed in their understanding of why the disease occurred, but very few writers attributed it to any single factor. This diversity has been widely acknowledged in historical studies of the period and some writers – particularly Christopher Lawrence – have made much of the fact that even James Lind (who seemingly demonstrated the efficacy of citrus juices during a trial in 1747) saw scurvy as having many causes, including poor hygiene and discipline. Indiscipline resulted in dirty ships and feeble bodies, he claimed, both of which were conducive to scurvy.³

Lind did not therefore ‘discover’ that scurvy was a deficiency disease; nor was his work unjustly neglected by the Admiralty. His results were significant, but the methods he employed were not as persuasive as they are now. Medical men using experiments and trials had to struggle for many years to establish the superiority of quantitative methods over more traditional sources of knowledge.⁴ Like most other diseases at the time, scurvy continued to be understood within a familiar framework of causation which was derived from the natural histories of Aristotle and which had been the stock in trade of European medical practitioners from ancient times. It had, however, undergone numerous refinements, incorporating new theories about how the body worked. Renaissance anatomy and physiology, chemical and mechanical theories of disease, and theories of contagion all left their mark.⁵ Nevertheless, the essential attributes of etiology had changed little. Most diseases were understood in terms of the inter-relationship between so-called ‘proximate’ causes (deranged organs, fluids and manifestations of disease in the body); ‘predisposing causes’ such as fatigue and depression; and ‘exciting causes’ such as vitiated diet, contagion and atmospheric influences.

This complex but flexible framework allowed diseases to be understood in much the same way as other natural phenomena and they were classified like plants and animals into different genera and species. This exercise was generally known as nosology or nosography and many

physicians devised their own classifications. In most of these, scurvy was normally listed as one of the 'putrid diseases'; that is, as a disease characterised by putrefaction of the 'humours' or four substances from which the fluid parts of the body were said to be comprised (blood, phlegm, and black and yellow bile). Corruption of the humours seemed to be indicated by many common symptoms of the disease such as swellings and other outward signs of decay, as well as by loss of teeth and foetid breath. The majority of writers therefore attributed scurvy to factors which were conducive to decay. James Lind, for example, believed that it was caused by perspiration which had been thwarted by moisture in the air, combined with the effects of a diet composed chiefly of salted meat. These factors tended to an 'alkalescent' disposition of the body – a term derived from the famous Dutch physician Herman Boerhaave (1668–1738), whose writings were indebted to the new chemical philosophy. Boerhaave's work had a formative influence on teaching at the newly established medical faculty at the University of Edinburgh; Lind studied there before entering the navy and afterwards completed an MD at the same institution.⁶ However, in Lind's view the action of dietary factors could be aggravated by indolence and filth, both of which predisposed the body to corruption.⁷

The putrid theory of scurvy was also evident in accounts of the disease as it occurred in the British Army, most influentially John Pringle's theory that the disease arose from 'gradually accumulated putrefaction' caused by moist or vitiated air, lack of exercise, deficiency of fresh vegetables and rotten items of diet. Salted meats, such as those used by sailors and sometimes on military expeditions, were liable to become putrid, he believed. Pringle's 'medical annotations' indicate that scurvy was attributed to a great variety of causes and that there were many supposed preservatives and treatments. Some of these, such as malt extracts, are well known but there were many others, including tea and various types of acids. Pringle referred to such substances as 'antiseptics', meaning that they were able to reverse or retard the process of putrefaction.⁸

Sir John Pringle (1707–82) was not merely a respected military physician, having been physician to the continental army in the War of Austrian Succession (1740–8),⁹ but also President of the Royal Society of London between 1772 and 1778.¹⁰ As such, his opinions had a formative influence on medical theory throughout the English-speaking world.¹¹ However, the tenets of the putrid theory of scurvy are equally evident in continental writings on the disease. The Austrian court physician Baron Gehard van Swieten was typical in regarding scurvy as a disease brought about by humours that had become putrid, acrimonious and condensed.¹² In his view, it was to be cured by attenuating the 'viscosity of the humours and in preventing or correcting putrefaction'.¹³ This meant avoiding external causes such as 'noisome vapours, arising from marshy grounds and stagnating waters, inaction, scarcity of greens and vegetables, drinking of corrupted and stagnating waters, the use of salted and smoked flesh and fish . . . damp and low lodgings'.¹⁴ It also meant counteracting predisposing factors such as 'fear and sorrow' and avoiding food which had gone bad. It was for this latter reason, he insisted, that persons trapped in besieged places commonly fell prey to the disease.¹⁵

This etiological framework remained relatively unchanged until the last two decades of the eighteenth century. Authors placed different degrees of emphasis on factors such as diet, but most conceived of scurvy as a putrid disease and described it in the familiar vocabulary of humoral medicine.¹⁶ But from the 1780s, writings on scurvy began to be coloured by nervous theories of disease, the principal source of these in Britain being the distinguished Edinburgh professor, William Cullen (1710–90). Cullen believed that the proximate cause of most diseases was a disorder of the nervous system, which could be brought about by either the action of external agents such as weather or poisons, or by personal dispositions and habits.¹⁷ Nervous physiology had gained ground slowly through the middle decades of the century and by the 1780s it is clearly evident in works on scurvy, such as that of the physician Francis Milman.¹⁸ The Oxford-educated Milman moved in distinguished circles in London but he had little direct experience with the

disease. Nevertheless, his connections and his fellowship of the Royal College of Physicians ensured that his work was taken seriously. Milman concurred with earlier writers that scurvy was a putrid disease, but he did not regard it as having originated from putrefaction of the humours. Instead, he believed that scurvy arose from nervous 'debility' – a term derived from the writings of John Brown, a rebellious pupil of Cullen.

Brown was a controversial figure, but his influence can be detected beyond his immediate circle and contributed to a growing tendency to place the nervous system at the centre of medicine and intellectual life.¹⁹ In Milman's view, scurvy was the consequence of several influences acting jointly on the nervous system. These included 'exciting causes' such as indigestible diet and food lacking in nutrition, as well as 'predisposing causes' such as indolence, fatigue, lack of exercise/sleep, cold, sorrow and melancholia.²⁰ Prevention lay in counteracting as many of these as possible, for example, by protecting bodies against the elements, providing better food, and insisting on cleanliness and discipline.²¹ Thus, despite the fact that Milman had abandoned humoralism for nervous theory, his causal framework remained practically the same: diet was merely one element – albeit an important one – among many. The only real differences lay in some of Milman's recommendations for prevention and cure. Instead of the common tendency to treat scurvy with a variety of substances reckoned to counteract putrefaction, Milman, like other Brunonians, recommended 'stimulants' such as brandy and opium, which were supposed to act by raising the body's nervous tone. In addition to the usual prophylactics, he also recommended water impregnated with 'fixed air' (later known as carbon dioxide), betraying a debt to the pneumatic chemistry of Joseph Priestley and others.

Pneumatic medicine of the kind associated with Priestley tended to appeal to the same people who were drawn to Brunonianism: men outside the physician elite such as practitioners in the armed forces, some of them religious and political dissenters.²² But the majority of works on scurvy published in the decade after Milman's *Enquiry* were less radical in tone and of comparatively little consequence. They tended to combine the most acceptable and least controversial elements of older works with insights from newer ones, embracing both humoral and nervous theories of disease. Some were practical in nature,²³ but others were clearly designed to appeal to all tastes and to serve as advertisements for their authors' patent remedies.²⁴ There were, however, two important exceptions. Both these works were written by naval practitioners whose authority rested on claims to direct and extensive experience. One was written by Thomas Trotter (1760–1832) and the other by Gilbert Blane (1749–1834).

Like many practitioners entering the armed forces, Trotter joined the navy with an incomplete university education, in his case at Edinburgh, to which he later returned to take a medical degree. A man of modest means but untiring assiduity, Trotter worked his way up through the service to become second physician at Haslar Hospital in 1793 and physician to the Channel Fleet a year later.²⁵ Trotter shared Milman's disdain for humoral pathology but he was more indebted to his former professor, William Cullen, than to Brown and he dismissed 'fashionable' theories such as the Brunonian notion of direct debility.²⁶ Brown was regarded by many physicians as a mountebank who had spawned a host of imitators touting patent medicines which claimed to restore nervous vitality.²⁷ However, Trotter was also critical of some aspects of Cullen's work, particularly his theory of scurvy, in which he detected vestiges of humoralism (his notion that scurvy was characterised by a saline state of the blood).²⁸ Trotter attempted to distinguish his own work by appealing to his extensive experience at sea by comparison with landlubbers like Milman. In his view, scurvy was caused primarily by the lack of fresh vegetables and not, as Milman claimed, by a diet that was indigestible.²⁹ Trotter thought the juices of lemons and limes particularly effective, but at the time he wrote his first treatise on the subject, in 1786, the problem of preserving them on long voyages had still to be overcome.³⁰ Trotter also differed from most previous writers on scurvy in that he rejected the view that it could be caused by the

elements alone, although he admitted that dampness and cold could be contributing factors.³¹ Other predisposing factors included indolence and depression, and Trotter noted the tendency of scurvy to appear among those whom he termed 'skulkers'. Such feelings were often brought on by impressment, he argued, and this was one of the reasons why he condemned this arbitrary system of conscription.³²

In the second edition of his *Observations on the scurvy* (1792) Trotter continued to denounce Milman and other Brunonians, but he embraced another medical fashion in the form of pneumatic chemistry.³³ Citrus fruits and fresh vegetables, he argued, were rich in 'vital air' (oxygen) and this explained why ships well provisioned with such victuals had been able to protect their crews from scurvy. Trotter later admitted (in response to criticism from the radical physician Thomas Beddoes) that he had made certain mistakes in his rendering of chemical theories.³⁴ But he did not depart from his main conclusion, contradicting critics such as Beddoes and other writers who believed that he had placed too much emphasis on defective diet. As far as Beddoes was concerned, anything which reduced the oxygen content of the blood was capable of causing scurvy, including vitiated air, and the disease could be prevented solely by keeping vessels well ventilated (like Captain Cook on his famous voyages of discovery) or simply by change of climate.³⁵ Likewise, any substance containing what Beddoes believed to be large quantities of oxygen, including meat and elixirs of various acids, might work as well as citrus juices in restoring the depleted body to health.³⁶ Among Trotter's other critics was Dr Seguin Henry Jackson, a physician of Westminster General and St George's Hospitals. In his view, scurvy arose from an accumulation of 'phlogiston' in the blood (an element which was reckoned by some to impart combustibility to the air) and could be relieved by any number of methods, such as the provision of pure drinking water.³⁷ These critics lacked Trotter's first-hand experience of the disease and were in some senses marginal figures. Whereas Beddoes's radicalism put him beyond the pale as far as many more orthodox practitioners were concerned, Jackson's theories harked back to an earlier period of chemical philosophy and could be dismissed as anachronistic and idiosyncratic. Nevertheless, Trotter's opinion that scurvy was essentially a deficiency disease clearly provoked disagreement.

Soon, however, Trotter would have the opportunity to put his theory to the test, for he was appointed physician to the Channel Fleet in 1794. His experiences over the next 12 months appeared to confirm his earlier conclusions about the centrality of diet. In December 1794 the fleet had to put in unexpectedly at Torbay because of adverse winds. While it lay at anchor no fresh meat was issued, only salt provisions, and vegetables were scarce because of the season. By January 1795 the fleet had moved to Spithead, where fresh vegetables remained in short supply. Over the coming months, cases of scurvy began to appear on every ship and 200 men were sent ashore to hospital. The epidemic did not abate until after the arrival of summer and ample supplies of fresh fruit.³⁸ In Trotter's view, these facts proved that his theories were true: the outbreak was unquestionably related to dietary deficiency and was remedied by fresh provisions. Furthermore, the emphasis which Beddoes had placed on impure air was shown to be wrong because ventilation and sanitary conditions on all the ships were excellent.³⁹ But though Trotter attached more importance to diet than many of his contemporaries, he remained convinced that scurvy was aggravated – if not caused – by other factors; in this case, low temperatures, the abuse of hard liquor, and the debilitating effects of illnesses like the 'epidemic catarrh' which swept the fleet at Spithead during January and February 1795.⁴⁰ Trotter was by no means alone in holding such opinions. Dr R.T. Crosfield, who was confined among British prisoners of war in France, attributed an outbreak of scurvy among the captives to a combination of defective nutrition, cold, damp and low spirits. In his view, fresh vegetables were not in themselves capable of restoring the sick to health.⁴¹

This inclusive view of the causation of scurvy was shared by the other chief authority on the disease, the naval physician Gilbert Blane. Blane was somewhat unusual among naval

practitioners in that he entered the service already in possession of a medical degree. Most of those who claimed the title physician took their degree, like Trotter, after a period of service which left them with sufficient funds to do so. Blane, however, hailed from a prosperous merchant family and was able to complete his medical education. His family connections also secured him a position as physician to Lord Rodney's fleet in the Caribbean during the American War of Independence. Under Rodney's patronage, Blane acquired an enviable list of aristocratic clients and an appointment as head of the navy's Medical Board in 1796.⁴²

Blane's first contribution to the literature on scurvy was his *Observations on the diseases incident to seamen* (1785). This early work may be regarded as a transitional text: its descriptive vocabulary is largely drawn from humoral pathology but its explanation of causation resembles Milman's.⁴³ But while it placed slightly more emphasis on diet than Milman, Blane's treatise still emphasised the importance of predisposing causes such as sloth, moisture, filth and cold. Hard labour, however, did not seemingly contribute to its causation.⁴⁴ Each new edition of the work was rooted more deeply in nervous physiology than the last.⁴⁵ The third edition (1803), for example, drew heavily on the theories of Erasmus Darwin, which in some respects resembled those of Brown.⁴⁶ Accordingly, scurvy was regarded as a disease of debility rather than of putrefaction, and citrus juices were said to work because they administered a stimulus to the system. They provided what Blane termed 'refreshment' rather than nourishment.⁴⁷ However, the growing emphasis on nervous physiology did not come at the expense of predisposing causes. While the notion of stimulus invited practitioners to look outward to the environment or to certain properties in foodstuffs, it acknowledged the importance of factors which had a depressing effect on the mind and body, such as poor weather or lack of mental and physical stimulation. Indeed, the later editions of Blane's work show progressively more emphasis upon the role of such factors in causing the disease.⁴⁸ As Blane put it:

There seems to be something in the habits of life in a ship, whether at sea or in port, favourable to sea scurvy. The ships belonging to the channel fleet in 1794 and 1795, were subject to scurvy even when at Spithead, though the men were fed with fresh beef and drank beer. This would not happen to men in a garrison. The difference of these situations consists, partly in the superior dryness, cleanliness, and ventilation of the latter; but more, I apprehend, in the want of exercise and recreation on board a ship.⁴⁹

This stress on the non-dietary causes of scurvy may be surprising in view of Blane's crucial role in securing the standard issue of citrus juices to naval vessels in 1795. It is well known that Blane regarded that year as a watershed in the health of the navy and later produced statistics to illustrate the dramatic upturn in the navy's health.⁵⁰ But he recognised that the issue of lemon juice was only one of a series of reforms which had improved the health of sailors, including new regulations on hygiene and cleanliness introduced in the 1790s and enforced through the new 'divisional system', whereby such matters were overseen by midshipmen.⁵¹ Diet was seen as the principal factor in preventing scurvy but certainly not the only one, and the reforms of the 1790s addressed many of the hygienic and disciplinary concerns formerly raised by such practitioners as Lind. Scurvy was effectively brought under control by these actions, but naval views of scurvy remained relatively open and there was no administrative reason to insist that they be simplified. In some of the other contexts in which scurvy occurred, the situation was very different.

Scurvy in India

As cases of scurvy in the Royal Navy fell to negligible proportions, the disease was becoming more common elsewhere. One of these places was British India. Scurvy had occurred periodically among the East India Company's troops throughout the eighteenth century, especially when its forces were besieged in fortresses by Indian rulers and the French. But as the company began

to establish itself as a major territorial power and to deploy its forces further afield, cases of scurvy began to increase. It was a particular problem during the campaign in Burma in 1824–6, when some 20,000 deaths were attributed to disease and starvation. Many of these were said to be due to scurvy (directly and indirectly) and its familiar symptoms were present in many of those who died. In 1826, for example, the disease appeared among European and Indian troops in the vicinity of Rangoon. After the city was captured, Burmese forces compelled locals to move to the interior and cut off provisions, leaving the company's army without the means to replenish its dwindling supplies. Heavy losses among both British and Indian troops during the First Burma War prompted a rethink of many aspects of military policy, including victualling and disease prevention. Most of these have been discussed extensively elsewhere,⁵² but the incidence of scurvy has gone largely unnoticed, despite evidence of growing interest in this disease in Anglo-Indian medical circles. This is of particular significance because the observations of Anglo-Indian doctors do not accord with the findings of naval practitioners such as Trotter. On the contrary, Indian conditions led to the emergence of a distinct body of knowledge, some of which contradicted prevailing wisdom at the Admiralty.

Writing of the outbreak at Rangoon, the East India Company surgeon Dr G. Waddell acknowledged the effects of poor diet in causing the outbreak (the reliance of Europeans on putrid salt meats and of all races on diets deficient in vegetable matter), but he placed equal weight on meteorological factors.⁵³ The outbreak of scurvy was attributed as much to the 'inclement season' in which it occurred as to the deficiency of rations.⁵⁴ Waddell likened the atmosphere then to be found in Rangoon to that in some of the company's troopships, in which cases of scurvy were relatively common. In these vessels, he insisted, scurvy arose primarily from exposure to 'impure air'. Predisposing factors were also important; hence in both the company's ships and in its armies it was the laziest and filthiest who were affected first.⁵⁵

By the early 1800s, the East India Company had also established institutions such as prisons, hospitals and asylums, of which many were plagued by diseases, including scurvy. The most likely explanation for the poor health of prisoners is that many had subsisted for a long period on a diet lacking in ascorbic acid, and this was especially true in periods of dearth during which prisons tended to reach their capacity. In view of the increasing visibility of scurvy, professional bodies such as the Medical and Physical Society of Calcutta began to call for more papers on the subject. However, explanations of scurvy in these institutions were no more likely to ascribe them to an impoverished diet than accounts of outbreaks in the army. In his observations on scurvy as it occurred at the Murshidabad Insane Asylum in 1822, Dr Burt saw many similarities with the outbreak at Rangoon, which he had later witnessed as a military surgeon.⁵⁶ In both cases, the disease appeared to be caused by a combination of poor diet and impurities of the air arising from the marshes which surrounded the city.⁵⁷ The ill-effects of the location were evident in the stubborn nature of the complaints, for scurvy proved hard to eradicate even when the army had received supplies of fresh vegetables and lime juice. Indeed, the disease only disappeared once the weather became drier. As Burt put it:

I am inclined to suppose, from its breaking out during the rains and cold weather, and generally disappearing in the hot and dry – from its attacking the gloomy and dejected ... that *humidity and depression of spirits are the principal, if not the only exciting causes* [emphasis added]. These depress nervous energy, and induce a torpid action of the general functions of the body. The stomach and digestive organs partaking of this diminished action, do not secrete a sufficient supply of chyle [bile] to nourish the body; hence scurvy is induced.⁵⁸

Burt's account had the same physiological basis as works on scurvy in the navy and, like them, it continued to acknowledge the importance of predisposing factors such as melancholia

and imbecility. However, it differs substantially in that dietary deficiency was not the only or even the most important external cause: on the whole, meteorological and other environmental conditions were regarded as more significant. In some respects this is only to be expected in view of the overwhelming dominance of climatic explanations of disease in Anglo-Indian medicine at this time and, indeed, in contemporary accounts of the constitutions and habits of the people.⁵⁹ Although scurvy did not affect Indians exclusively, it seldom occurred among Europeans except when they had been subjected to abnormal deprivation. But instead of concluding that Indians must be more deprived than Europeans, British medical practitioners tended to attribute the disease to exposure to noxious influences. The vulnerability of Europeans increasingly disposed them to seek refuge from the climate, while Indians went about barely clothed. While they were less frequently prey to acute diseases, those who inhabited the least salubrious environments tended to show the progressive effects of debility. It was widely held that such influences, passed down through many generations, accounted for the 'degraded' state of many Indians in relation to Europeans and in relation to the more robust 'martial' races of the subcontinent. Natives of low, marshy areas, such as parts of deltaic Bengal, exhibited this tendency towards degeneration most markedly, it was claimed.⁶⁰

Dr D. MacNab, in his observations on scurvy among sepoys at Nusserbad, Bengal, in 1836, thus attributed the outbreak to depleted 'nervous energy', possibly due to poor nutrition (adulterated grain) but mostly to the effects of climate.⁶¹ 'Climate has always been admitted to exercise important bias over the character of disease, as well as over the constitution itself of man,' he opined. In his view, the atmosphere around the cantonment was deficient in 'electric qualities', which frequently led to 'debility' and 'degeneration' among the inhabitants.⁶² Four years later, Dr J. Bouchier also dismissed any notion that the outbreaks of scurvy he had witnessed were due to diet. Writing of an epidemic among prisoners in the Rutnagherry jail in the mid-1830s, he noticed that there were no cases among female prisoners despite them receiving the same food as the men. In his view, the most likely explanation was the scarcity and poor quality of water from the wells during the summer months. This appeared to explain why scurvy became more of a problem in times of drought.⁶³

What these interpretations had in common was that they drew attention away from diet and the shortages caused by famine. This was welcome for two reasons. First, it effectively naturalised scurvy, detracting from new methods of agricultural production and related issues such as the controversial revenue settlements recently imposed by the East India Company in many parts of India.⁶⁴ As MacNab put it: 'There are thousands, year after year, living on in poverty and wretchedness, yet when have we heard of its giving birth to Scorbutic disease, without a combination of other contingencies which are of rare occurrence.'⁶⁵ Second, rations for the company's soldiers, and the Bengal Army in particular, had often provoked unrest among sepoys, not least during the Barrackpore Mutiny of 1824, which was caused in part by the failure to provide appropriate items of diet.⁶⁶ If poor diet was officially declared the principal cause of scurvy then the company could be held responsible for this, too. These factors may help to explain why its surgeons were reluctant to regard scurvy as due to deficiencies of diet.

However, those who wrote about scurvy could not always agree on what, if not diet, was the most important factor. Climatic explanations tended to dominate, but some also blamed what they saw as the dissolute habits of many Indians. In his 1835 paper on scurvy among sepoys stationed near Cachar, Bengal, the East India Company's surgeon J. Hutchinson placed most emphasis on the widespread use of opium, which he claimed destroyed nervous power. Such explanations were also common when accounting for the high incidence of scurvy and other diseases in Indian jails. As one correspondent to the *India Journal of Medical Science* put it:

The prisoners . . . are most of them of the very lowest caste. They are generally men of debauched habits, who have led very irregular lives . . . the very scum of society enter our jails. And are not people of as depraved a nature particularly predisposed to disease on being subjected to the restraints of prison?⁶⁷

This tendency to ascribe the disease to factors other than diet did not really change until the 1840s, for reasons which will become clear in a moment.

Penitentiaries

If the logic of administration in India suited a view of scurvy which was broadly environmental, quite the opposite was true in Britain where 'land scurvy' had become common among prisoners. The problem was most evident in new penal institutions such as the National Penitentiary at Millbank – the reformatory designed by the Utilitarian philosopher Jeremy Bentham. Millbank was supposed to mark a radical departure in the treatment of prisoners. It was intended as an engine for the reform of miscreants rather than a repository for the refuse of society. These new institutions were supposed to be clean and free from disease, but their regime was also harsh and unyielding.⁶⁸ Prisoners were to be weaned away from a life of vice by 'well regulated labour and religious instruction'.⁶⁹ Like the workhouses which would later be introduced by the New Poor Law, food and other comforts were intended to be 'less eligible' than those normally available to the labouring poor.⁷⁰

Attempts to reform prisons preceded the Utilitarian-inspired reforms of the prison system and stemmed from the labours of philanthropists such as John Howard and Sir George Onesiphorus Paul. Their inspiration was as much moral as it was rationalistic. However, the idea of the clean prison, free from disease, was essential to the Utilitarian project. Reform of inmates could not be accomplished if they were sick, for disease impaired efficiency, but not long after Millbank began to reduce its 'luxurious' rations in line with Benthamite principles, disease began to show itself in the form of scurvy.

The recommendation to reduce rations was made by the prison's Principal Medical Officer Dr Hutchinson, who had been asked to look into the matter by the prison's management committee. According to Hutchinson, 'a general fullness of habit appeared to prevail among all the inmates of the Penitentiary', particularly the women.⁷¹ A reduced scale of diet was therefore advocated, following the precedent set by Dorchester prison. Instead of the former allowance of three-and-a-half ounces of meat and one pound of potatoes, the inmates were to receive one-and-a-half ounces of bread, two pints of broth and one pint of gruel.⁷² However, the committee first sought the opinion of Sir James McGrigor, the Director General of the Army Medical Department, presumably on account of his responsibility for the health of military prisoners. McGrigor agreed that the old diet was too 'full' and approved the plan proposed. In July 1822 the new ration was adopted.

Introduction of the new diet soon led to a serious deterioration in the inmates' health and the matter was brought repeatedly before the management committee and the prison's board of visitors. No substantial alteration was made, however, and the decision to persist with the ration placed Dr Hutchinson at odds with his subordinate medical officer, Mr Pratt, who was resident at the penitentiary, as well as with the matron, nurses and other officers of the establishment.⁷³ By January 1823 the first definite symptoms of scurvy appeared in one of patients seen by Hutchinson, and the disease became more common over the coming months among prisoners of all types and both sexes. On 14 February, McGrigor was brought in to inspect the prisoners and, having examined all the female and many of the male convicts, he reported that they were in reasonable health, the incidence of sickness being much as he would expect for the time of year. Most importantly, McGrigor concluded that none of the sickness was due to inadequate

diet or length of confinement. Thus reassured, the management committee took no further steps.⁷⁴

But within a few days scurvy began to gain ground rapidly, increasing from 53 cases in the middle of February 1823 to 118 by the end of the month. This disturbed the committee and it sought a second medical opinion from two other physicians, Drs Latham and Roget, who were asked whether the disease could be attributed to the new rations. Latham and Roget visited the prison on 1 March and found 110 inmates lying in the infirmary – 44 males and 66 females. Alongside Dr Hutchinson, they inspected each of the prison's pentagonal cellblocks and found that 448 of the total of 880 inmates showed signs of disease, often a combination of scurvy and dysentery. Women were more badly affected than men, on the whole, as were those who had been longest in confinement.⁷⁵

In the opinion of Drs Roget and Latham, the high incidence of scurvy among the inmates at Millbank was due primarily to their new diet, but they also concluded that cold, overwork, insufficient exercise and depression had been predisposing factors. Their opinion was largely, though not completely, welcomed by the Select Committee of the House of Commons which was appointed in 1823 to investigate the outbreak. The appointment of such a committee is worthy of note, for it shows the seriousness of scurvy in political terms. The high incidence of disease in Millbank reflected badly not only on the management of that prison but also on the new system of penal reform and, by extension, the political and economic principles on which it was based. But the fact that the Select Committee was willing to entertain a report critical of the penitentiary's dietary regime may seem surprising. It did so almost certainly because such a conclusion did not require a radical alteration or rejection of the penitentiary system but merely a relatively modest alteration of rations.

That the Select Committee had such an objective in mind is strongly suggested by the line of questioning to which Roget, Latham and other witnesses were subjected. While they had stressed the primacy of diet as a causative factor in scurvy, like most physicians at the time they continued to acknowledge the contribution of predisposing factors, mental and physical. Their insistence on these predisposing factors was less welcome to the Select Committee than their comments concerning diet. Accordingly, the questions asked of the doctors were such as to cast doubt on all factors other than the deficiency of rations. The Select Committee was particularly displeased with the assertion that cold weather had contributed to the outbreak, the implication being that many of the cells afforded inadequate protection from the elements. It pointed to the fact that prisoners who worked in the kitchens suffered less from scurvy and stated its opinion that this was due to additional food rather than to better heating: a point which Dr Roget later conceded. The committee also pointed to the fact that few other prisons had heated cells and yet did not suffer scurvy, forcing Roget to admit this, too.⁷⁶ Having elicited the opinion it desired, the committee concluded that there was no need to provide artificial heating for the cells – a provision which would clearly have been expensive.

Roget and Latham's contentions that the scurvy had been due in part to depression and exhaustion were more problematic. These observations called into question the reformatory regime itself, for it was founded on lengthy sentences and rigorous, repetitive labour.⁷⁷ The Select Committee questioned the assertion that these things were causes of scurvy by again referring to the absence of the disease in other jails. It seemed oblivious to the physicians' argument that it was a combination of causes rather than any single factor which was operative.⁷⁸ However, the committee did concede that the prisoners showed signs of low spirits and recommended more exercise and a wider selection of literature for them as an antidote – not merely works which had been selected to promote Christian knowledge.⁷⁹ This small concession was the only one made by the Select Committee other than the need to improve the prisoners' diet. It had effectively closed off all alternative or even supplementary explanations for the outbreak, thus leaving the

penitentiary regime intact. In the committee's view, there was nothing wrong with it that could not be fixed by a simple alteration of diet.⁸⁰

This was not the last time that parliament would hear of scurvy in prisons or, indeed, in Millbank. Throughout the 1820s and 1830s scurvy was still common in some prisons, especially those in London and East Anglia. The inspectors who had been appointed in the wake of the parliamentary enquiries of the early 1820s carefully recorded the views of prison surgeons, most of whom drew attention to the inadequacy of diet but also, in some cases, the contributory affects of cold weather and hard labour.⁸¹ The emphasis on hard labour called into question the logic of penal reform and was clearly unwelcome to advocates of the penitentiaries, but some surgeons persisted in their opinions. In 1837, for example, the Inspectors of Prisons noted that the surgeon of the Springfield House of Correction in Essex believed that 'the combined effect of hard labour, with inadequate and insufficiently varied diet, is further evident from the fact that no such disorder existing in the old gaol at Chelmsford, which is damp, and in local respects, less healthy than Springfield, but where there is no tread-mill labour'.⁸² But prison doctors had no power to compel their governors to alter rations. Indeed, they were charged with managing scorbutic symptoms by dietary manipulation, allowing rations to be kept to the bare minimum compatible with health.⁸³

By the 1840s, criticism of prison regimes was rare and dietary manipulation the norm.⁸⁴ Although there were still occasional references to dampness and other contributory factors, scurvy was represented medically and administratively as a deficiency disease. Ordered in this way, the problem of scurvy could be solved with comparative ease, for a cheap and effective remedy was at hand. Dr William Baly, physician to the penitentiary at Millbank and lecturer on forensic medicine at St Bartholomew's Hospital, was one of a number of medical men who recommended the addition of potatoes to prison rations and he did so after conducting an investigation of scurvy among military prisoners at Millbank. At that time, military prisoners had a lower allowance of potatoes in their diet than civilian inmates and it was this that accounted for the high prevalence of scurvy among them. There were practically no cases among civilians, the disease having been eradicated after Latham and Roget were permitted to alter their diet by adding potatoes. Whereas civilian prisoners were allowed five pounds of the vegetables weekly, the military prisoners were permitted only half a pound, and then only after six months' imprisonment.⁸⁵ Baly also examined the reports of the Inspectors of Prisons with a view to ascertaining whether scurvy was due to the absence of fresh vegetables. This was clearly the case in two instances – the Oxford and Northampton County jails – in which few fresh vegetables had been served.⁸⁶ Conversely, in several prisons scurvy had completely disappeared after the addition of a few pounds of potatoes to the weekly ration.⁸⁷ According to Baly, the value of the potato as a preventive of scurvy lay in the fact that it contained a high concentration of 'organic acids', including the citric and tartaric varieties. Potatoes thus appeared to contain the same active ingredients as other well-known antiscorbutics such as citrus fruits. However, they were far easier to obtain and store, as well as being considerably cheaper to supply. In view of this, he argued, they were not only well suited to prisons and other public institutions but might also be used by the Merchant Navy which continued to suffer badly from scurvy. In Baly's opinion, parsimony and laziness had led the Merchant Marine to neglect the naval practice of storing lemon juice on vessels, but potatoes might offer a cheap and practicable alternative.⁸⁸

Scurvy and the nineteenth-century navy

Having considered the situation in British prisons, it is instructive to return to the subject of scurvy in the Royal Navy. While scurvy was no longer an operational problem in the way that it had been

during the eighteenth century, naval medical officers still regularly faced the disease on convict vessels and occasionally on other long voyages and expeditions, most obviously those to the Arctic.⁸⁹ This section concentrates largely on outbreaks of scurvy on convict ships, to which Royal Navy surgeons were appointed. It was on these vessels that the majority of cases seen by naval surgeons occurred and on which we might expect to find similar concerns to those arising in penitentiaries. A good deal is already known about the health of ships designated to transport convicts and immigrants from Britain to Australia. After numerous voyages with high death rates, conditions on board such vessels improved following the imposition of regulations which stipulated the attention of surgeons and better provisions and medical supplies.⁹⁰ On convict vessels, the problems experienced with scurvy and other diseases were more often the legacy of malnourishment and infection prior to boarding rather than conditions on the vessels themselves.⁹¹ From the 1820s through to the 1840s, scurvy was among the most common diseases appearing on the prison hulks at Woolwich, Chatham and other locations, and many prisoners were probably borderline scorbutic cases before they were transported to Australia.⁹² The following discussion confirms this impression, but its chief purpose is to determine how far naval medical lore concerning scurvy had changed since the days of Trotter and Blane. In particular, it seeks to ascertain whether the scandals in civilian jails had much impact on the way in which scurvy was conceived and treated on convict vessels.

On the eve of the outbreak at Millbank, the views of naval surgeons on the causation of scurvy do not seem to have altered to any significant extent. Dietary deficiency appears to have been regarded as the principal cause of scorbutic complaints but by no means the only one, and in some cases it was expressly ruled out. In 1821, for example, the surgeon of HM Convict Ship *Lady Ridley* attributed the numerous cases of scurvy and debility on his vessel to 'the bad quality of our water which has been very putred [sic] for some time'.⁹³ Scurvy continued to be a regular feature of the convict voyages and it appeared so frequently that it was seldom the object of serious attention. But this is not to say that surgeons were uninterested in the disease or that they were unaware of what had been written about it in other contexts. Some had clearly read the report by Roget and Latham on the Millbank outbreak and compared their descriptions of the disease to that seen on convict vessels.⁹⁴ Latham and Roget's conclusions were not dissimilar to those espoused by the majority of naval surgeons, whose views of scurvy remained flexible and inclusive.

One of the more serious outbreaks of scurvy on a convict vessel occurred in 1833–4 towards the end of the *Southworth's* voyage to Van Diemen's Land. By the time the vessel had reached Hobart there were 30 on the sick list, mostly cases of scurvy or scorbutic dysentery. Some were so debilitated that they had to crawl ashore.⁹⁵ The surgeon, William Evans, gave the afflicted convicts citric acid and nitrate of potash (which had come into vogue as a treatment for scurvy and scorbutic dysentery) and paid close attention to the cleanliness of the prisoners.⁹⁶ These actions suggest that he did not regard the outbreak as due solely to dietary deficiency, but he noted that most cases were cured after they landed and were given plenty to eat. Evans commented that 'nothing short of liberal diet of fresh animal food and recent vegetables with soft bread, can affect that necessary change in the circulatory fluids so as to re-establish health'. It would therefore seem that Evans regarded diet as the principal factor in the causation of scurvy, but not the only one.⁹⁷ Furthermore, there is no evidence that he thought scurvy due to the absence of any particular dietary component or substance. In his opinion, this fresh diet seems to have acted principally as a stimulant which reinvigorated the body's vital power.

While most cases of scurvy treated by naval surgeons were confined to convict vessels, there were occasional outbreaks on long naval voyages. One such instance occurred on HMS *Atholl* in 1827, during operations off the African and Arabian coasts. The crew was said to have a disposition towards scurvy and three of the most severe cases died during the voyage. Of these men, two

were said to have ‘very bad habits of body’ prior to their showing evidence of scurvy and one had suffered several attacks of fever. The surgeon, William Aitken, was unsure to what he should attribute the outbreak, ‘except unwholesome water, bad biscuit, short allowance of spirits and provisions, and deficiency of vegetable diet, as the atmosphere was pure and the sky transparent’.⁹⁸ There is nothing in Aitken’s report to suggest that he regarded the outbreak as due to the deficiency of a single item of diet or some principle therein. Indeed, although atmospheric causes were excluded in this instance, Aitken’s remarks indicate a willingness to entertain such an explanation. The persistence of a multi-factorial view of scurvy is further indicated by the fact that a range of preventive measures was enjoined, ranging from the distribution of lime juice to the cleansing of drinking water with lime.⁹⁹ Forms of treatment used by other naval surgeons for scurvy exhibited similar eclecticism. In fact, throughout the 1830s and 1840s it appears that citric acid was not highly regarded as a treatment for scurvy. Nitrate of potash, sometimes mixed with vinegar and sugar, was reckoned by some to be more effective.¹⁰⁰ All seem to have agreed on the restorative effects of diet, especially a diet abundant in beef and fresh vegetables, but there is no indication that the problem of scurvy could be solved by the addition of any single item such as the potato.¹⁰¹

There is a clear difference between the outlook of most naval surgeons and that of the parliamentary Select Committee which enquired into the outbreak at Millbank. The same can be said of the Select Committee which later examined the transportation of offenders to Australia. That committee’s report in 1837 extolled the virtues of the potato as a cheap and nutritious item of diet and recommended that the proportion of this vegetable in the diet of prisoners working on chain gangs and penal settlements in Australia be considerably increased.¹⁰² Some surgeons on board prison hulks in British waters tried similar expedients using carrots, but their views on the causation of scurvy and similar diseases remained more diverse and they were prepared to entertain factors such as depression. Indeed, on occasion they directly criticised the regime in some of the prisons from which the convicts had been drawn prior to boarding the hulks. In his medical report for 1842, for example, Archibald Robertson declared that scurvy had been one of the principal complaints among the 436 patients sent from the convict hulk *Fortitude* to the hospital ship *Wyre*. In his view, the remote and exciting causes of the disease were to be found in ‘the previous prison discipline, solitary confinement, and low diet of the majority of our gaols, and which render very many of our convicts more fit for the hospital than for dockyard labour’.¹⁰³

Conclusion

The persistence of a reasonably broad view of scurvy thus presented few difficulties for the navy or, for many years, in India, but it posed a significant threat to the new penitentiary system in Britain and parts of scurvy’s etiological framework were excised as a result. Ultimately the politics of confinement in Britain were to have consequences for India, too. Growing scrutiny of the Indian prison system in the wake of the British enquiries forced the East India Company to take more account of the rations given to the inmates in its prisons.¹⁰⁴ Jails were ordered to revise their dietary scales and medical officers were assigned to devise new ones. More stress was now placed on the provision of fresh vegetables and other foods known to possess antiscorbutic qualities.¹⁰⁵ By the late 1840s, some Indian jails were already showing signs of improvement and some had established their own gardens specifically to provide prisoners with fresh vegetables.¹⁰⁶ Medical officers who continued to preside over gaols with a high incidence of scurvy were removed.¹⁰⁷ Although Indian soldiers were still permitted to obtain most of their own foodstuffs, the rations of British soldiers were altered in accordance with what had become the dominant view of scurvy in Britain. But the principal reason was that the old environmental paradigm of scurvy had been found inconvenient. Rather than abandoning remote stations previously reckoned to be prone to scurvy on account of their climate, an effort was made to cultivate vegetables

locally and to make up for deficiencies with extra supplies.¹⁰⁸ Better rations were also provided for invalids proceeding home to Britain, including on vessels which did not carry the East India Company's flag.¹⁰⁹

What these various initiatives show is that the natural history of scurvy was closely and inextricably linked to administrative considerations, be they the principles of penal reform or the strategic implications of withdrawal from remote but crucially important outposts of empire. By the middle of the nineteenth century there was a very loose consensus around the importance of dietary factors in the causation of scurvy, but this was more narrowly construed in British penitentiaries and Indian prisons than in the navy or even on board the convict vessels manned by its surgeons. In prisons, political expediency dictated a very definite view of what caused scurvy and how it was to be remedied. Lacking the constraints imposed by these administrative requirements, however, practitioners in other contexts continued to speculate widely over the causes of scurvy. Some still attributed it to unfavourable weather, fatigue, mental states and poor hygiene, in addition to inadequate diet, while others were later to claim that it was the consequence of bacterial infection. These widely differing accounts of the genesis and therefore the prevention of scurvy persisted into the First World War, when mounting evidence was adduced from the campaign in Mesopotamia to support the view that it was caused by the lack of a single substance, eventually to be known as vitamin C.¹¹⁰

Notes

1. See, for example, Carpenter, *History of scurvy*, 98.
2. Rodger, *Command of the ocean*, 308.
3. Lawrence, 'Disciplining disease'.
4. See Kelly, *War and the militarization of British Army medicine*; Tröhler, 'Quantification in British medicine and surgery'.
5. The literature on these subjects is voluminous but see, for example, Maclean, *Logic, signs and nature in the Renaissance*; French, *Medicine before science*; Wear, *Knowledge and practice in English medicine*.
6. See Lindeboom, *Herman Boerhaave*; Cunningham, 'Medicine to calm the mind'.
7. Lind, *Treatise on the scurvy*.
8. PRJ/1/10, Sir John Pringle, medical annotations, 58–61.
9. Pringle, *Observations on the diseases of the army*.
10. On Pringle and his circle, see Golinski, *Science as public culture*, 106–9.
11. Harrison, *Medicine in an age of commerce and empire*, 65–9.
12. Swieten, *The diseases incident to armies*, 90–1.
13. *ibid.*, 92.
14. *ibid.*, 91.
15. *ibid.*, 92.
16. For example, Hulme, *Libellus, causa*. This work stressed the importance of diet in the causation of scurvy and of the juices of citrus fruits as a remedy.
17. For an introduction to Cullen's life and work, see W.F. Bynum, 'Cullen, William (1710–1790)', *Oxford Dictionary of National Biography*.
18. Milman, *An enquiry*.
19. See Risse, 'The Brownian system of medicine'; Bynum and Porter, eds, *Brunonianism in Britain and Europe*. On the general influence of nervous theory, see Lawrence, 'The nervous system'; Rousseau, *Nervous acts*.
20. Milman, *An enquiry*, 8, 12–15, 16–27.
21. *ibid.*, 28–44.
22. On pneumatic chemistry and political radicalism generally, see Levere, 'Natural philosophers in a coffee house'. On the influence of pneumatic chemistry on medical practitioners in the armed forces, see Kelly, *War and the militarization of British Army medicine*; Harrison, *Medicine in an age of commerce and empire*. For pneumatic medicine in Britain generally, see Jay, *Atmosphere of Heaven*.
23. For example, Rymer, *Letter to the commissioners for the sick and wounded seamen*.

24. For example, Hayman, *Treatise on the scurvy*.
25. See Vale and Edwards, *Physician to the fleet*; Harrison, *Medicine in an age of commerce and empire*, 237–41.
26. Trotter, *Observations on the scurvy* (1786), xiii, 66.
27. For scurvy see, for example, Solomon, *Guide to health*, xviii–xix, 134–9. The first editions of this work were published around 1800.
28. Trotter, *Observations on the scurvy* (1786), 72.
29. *ibid.*, 59, 68.
30. *ibid.*, 98.
31. *ibid.*, 82–3.
32. *ibid.*, 83.
33. Trotter, *Observations on the scurvy* (1792), xii, xxiv.
34. Trotter, *Medical and chemical essays*.
35. Beddoes, *Observations on the nature and cure of calculus*, 52–3, 58, 94.
36. *ibid.*, 59, 84.
37. Jackson, *Dermato-pathological*.
38. Trotter, *Medicina nautica*, 404–7, 411, 414.
39. *ibid.*, 426–7.
40. *ibid.*, 406–7, 408, 416, 422.
41. Crosfield, *Remarks on the scurvy*.
42. See Harrison, *Medicine in an age of commerce and empire*, 260–1.
43. Blane, *Observations on the diseases incident to seamen*, 460, 468–9, 471.
44. *ibid.*, 460.
45. Blane, *Observations on the diseases of seamen* (1789).
46. Blane, *Observations on the diseases of seamen* (1803), 497–8.
47. *ibid.*, 488.
48. *ibid.*, 481.
49. *ibid.*, 482.
50. Blane, ‘On the comparative health of the British Navy’.
51. *ibid.*; Lawrence, ‘Disciplining disease’.
52. For example, Peers, ‘War and public finance’.
53. Waddell, ‘On the diseases which prevailed’.
54. *ibid.*, 273–4.
55. *ibid.*, 274–5.
56. Burt, ‘On land scurvy among the natives’.
57. *ibid.*, 18.
58. *ibid.*, 18–19.
59. Harrison, *Climates and constitutions*.
60. *ibid.*, chap. 3.
61. MacNab, ‘An account of the scurvy’.
62. *ibid.*, 112.
63. Bouchier, ‘Notes on the scurvy’.
64. See Guha, *Rule of property for Bengal*. For the relationship of later settlements to prevailing doctrines of political economy, see Stokes, *English Utilitarians and India*.
65. MacNab, ‘An account of the scurvy’, 113.
66. Harrison, *Climates and constitutions*; Peers, ‘Habitual nobility of being’.
67. Anon., Letter from ‘D.W.’, ‘On the management of Indian jails’.
68. Ignatieff, *A just measure of pain*.
69. *Report from the Select Committee on ... the penitentiary at Millbank*, 1.
70. See, for example, Boyer, *Economic history of the English poor law*; King and Tomkins, *Poor in England*.
71. *Report from the Select Committee on ... the penitentiary at Millbank*, 5.
72. Carpenter, *History of scurvy*, 99.
73. *Report from the Select Committee on ... the penitentiary at Millbank*, 10.
74. *ibid.*, 7.
75. *ibid.*, 7–8.
76. *ibid.*, 22–3.
77. *ibid.*, 23.

78. *ibid.*, 23.
79. *ibid.*, 4.
80. *ibid.*, 18.
81. For example, *Report of Inspectors of Prisons of Great Britain II*, 4, 39, 49, 85.
82. *Second report of the inspectors appointed ... to visit the different prisons of Great Britain*, 304.
83. For example, *Report of Inspectors of Prisons of Great Britain II*, 4.
84. For example, *Seventh report of the inspectors*, 179.
85. Baly, 'On the prevention of scurvy', 1–5.
86. *ibid.*, 5.
87. *ibid.*, 6.
88. *ibid.*, 7. See also Cook, *Disease in the Merchant Navy*, 197–9.
89. As well as occurring in the Arctic, scurvy also broke out during operations in the River Plate and Black Sea in the 1840s and 1850s. See McLean, *Surgeons of the fleet*, 40; Lambert, *Franklin*; Bayliss, 'Sir John Franklin's last Arctic expedition'.
90. See, for example, *Convict vessels. Copies of instructions to the surgeons*. Surgeons were charged with ensuring that the convicts were given rations without deduction, that they received an allowance of lemon juice daily, and that they carried out daily medical inspections.
91. See Foxhall, 'Disease at sea'; Haines, *Doctors at sea*.
92. For example, *Convicts. Two Reports of John Henry Capper* (1826–7), 1, 6; *Convicts. Two reports of John Henry Capper* (1843), 8.
93. TNA, ADM 101/42/2B/3, fo 16, Medical and surgical journal of HMCS *Lady Ridley*, from 17 Nov. 1820 to 26 Oct. 1821.
94. See TNA, ADM 101/27/4/6, fo 45, General remarks of Surgeon Charles Cameron, medical and surgical journal of HMCS *Ferguson*, from 23 Feb. 1828 to 8 Apr. 1829.
95. TNA, ADM 101/68/10/4, fo 28, Medical and surgical journal of HMCS *Southworth*, from 31 Aug. 1833 to 28 Jan. 1834.
96. *ibid.*, fos 22–3.
97. *ibid.*, fo 28.
98. TNA, ADM 101/88/2/1, fos 18–19, Medical and surgical journal of HMS *Atholl*, from 1 Jan. 1827 to 19 Oct. 1827.
99. *ibid.*, fos 19–20.
100. TNA, ADM 101/22/4/3, fo 28, Medical and surgical journal of HMCS *Eden*, from 3 Mar. 1842 to 13 July 1842; TNA, ADM 101/8/1, Medical and surgical journal of HMCS *Bengal Merchant*, from 6 Sep. 1834 to 20 Feb. 1835.
101. See, for example, TNA, ADM 101/40/3/fo 65, Medical and surgical journal of HMCS *Katherine Stewart Forbes*, from 26 Jan. 1832 to 24 July 1832. The 36 convicts who arrived at Hobart showing symptoms of scurvy (many very severe) reportedly looked much better after being fed a diet of beef and fresh vegetables for 10–12 days.
102. *Report from the Select Committee on transportation*, 8.
103. Quoted in *Convicts. Two reports of John Henry Capper* (1843), 8.
104. Dutta, 'Disease and medicine in Indian prisons'.
105. Leith, 'Contribution to dietetics'.
106. BL, IOR, E/I4/837, India and Bengal despatches, July 1856, Medical no. 98, 331.
107. BL, IOR, E/4/854, India and Bengal despatches, 1858, Judicial no. 2, 371–2.
108. BL, IOR, E/4/814, India and Bengal despatches, Feb.–Mar. 1852, Military no. 4, 458–9; BL, IOR, E/4/813, Bengal and India despatches, Nov. 1851–Jan. 1852, Military no. 7, 61; BL, IOR, E/4/820, India and Bengal despatches, May–June 1853, Military no. 7, 501.
109. BL, IOR, E/4/843, India and Bengal despatches, Apr. 1857, Judicial no. 142, 1442.
110. Harrison, 'Fight against disease', 479–89.

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