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The Scottish Scurvy Epidemic of 1847

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Abstract

This paper examines the emergence of scurvy in several parts of Lowland Scotland during 1847. At first the condition was not recognised because of a mistaken, persistent belief that scurvy was only seen at sea, despite the work of James Lind who showed that sea and land scurvy were one and the same. Professor Christison failed initially to recognise the disease and wrongly thought it was caused by a lack of milk in the diet; colleagues elsewhere correctly attributed scurvy to the loss of the potato in the diet of the poor.

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Introduction

In 1847, accounts in the medical press spoke of scurvy appearing in many parts of the United Kingdom. The Scottish publication, *Monthly Journal of Medical Science* in June 1847 reported an outbreak of scurvy in Edinburgh and another at Perth Prison in the autumn of 1846, commenting that the epidemics had found an able historian in Professor Christison, 'who at the last meeting of the Medico-Chirurgical Society, read an elaborate memoir, abounding in novel views of the etiology(sic) and mode of treatment of the disease.' Robert Christison, (1797–1882), later Sir Robert Christison Bart, Professor of Materia Medica, President of the Royal College of Physicians of Edinburgh and Vice President of the Royal Society of Edinburgh had a special interest in nutrition, delivering a course of lectures on dietetics, the handwritten notes of which are preserved in his papers. His advice on institutional diets was sought after: the April 1843 Minutes of Edinburgh Royal Infirmary, expressed thanks to Dr Christison who had prepared eight diet tables; in 1846 he was appointed by the Board of Supervision for the Relief of the Poor in Scotland to advise poorhouse managers on diets and he was consulted by the Perth prison authorities concerning the scurvy outbreak in the autumn of 1846. Christison's opinion as to the cause of the Edinburgh outbreak will be analysed and contrasted with that of his colleagues in other centres.

The Potato Blight

A leading article in *The Lancet* attributed the outbreak of scurvy

'…to the absence of the potato (as it formerly was to the limited use of all vegetables) from the food of the poorer classes, and the consequent deprivation of an acid principle, which appears essential to the healthy condition of the blood.'[1]

The potato crop had been destroyed by a fungus, *Phytophthora infestans*, probably brought to Europe from the Andes when the newly independent South American states began to trade with Europe. Significantly, among the varieties of potato affected in the first recorded European attack in the Courtrai area of Belgium in June 1845, were three bearing the names 'Lima', 'Péruviennes' and 'Cordillières.'[2]

The first reports of blight in Britain were from the Isle of Wight in August 1845 and by mid-September most English counties, the eastern counties of Ireland, and the southern parts of Scotland were affected. The crop in the Highlands and Islands escaped in 1845 but prolonged wet weather the following year caused widespread disease and by the time of the potato harvest it was evident that the whole of the country was affected, the extent of the crisis in Scotland becoming clear only in the last quarter of 1846. Devine reckons that 90 per cent of the crofting districts lost their entire crop but the farming districts in the east of the Highlands were not spared with 80 per cent badly hit. It was ironic that a greater acreage than normal had been planted in the hope of repeating the profits of 1845 and early 1846, when potatoes from the Highlands fetched high prices; in April 1846 five thousand barrels of potatoes were exported from North Uist to Glasgow selling at almost twice the usual rate.[3]

There were many theories as to the cause of the blight and equally numerous were the proposed cures. Few were quite as pessimistic as the editor of *The Gardeners' Chronicle and Horticultural Gazette*: 'as to cure for this distemper there is none… man has no power to arrest the dispensations of Providence. We are visited by a great calamity which we must bear.'[4] The Highland and Agricultural Society of Scotland was less pessimistic although some reports echoed an identical resignation to the will of the Almighty. In November 1845,

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James Johnston (1796–1855), chemistry lecturer in Durham, was asked by the Society to analyse reports on the failure of the potato crop in Scotland, offering 'a premium' for analyses of diseased potatoes and setting up a 'Chemical, Botanical, Entomological and Practical' inquiry. A Clackmannan farmer replied to the Society quoting Rob, his foreman 'who says "may a' crack as they like about it, but it's just a punishment sent on us for our sins, for we've never been half thankfu' for our mercies."' The famer opined that 'this is one of the truest and most sensible remarks that I have yet fallen in with about the matter'.[5] The Church of Scotland Assembly of 1848 not to be outdone proclaimed that 'having taken into serious consideration the widespread distress which has long prevailed in this country, as well as in other lands, so strikingly manifesting the displeasure of the Almighty…' and praised 'the exemplary patience with which it [distress] has been borne by the poorer classes.'[6]

The Edinburgh Scurvy Epidemic

There were significant differences in the reaction of the medical profession in Scotland to the scurvy outbreak with reports indicating that the views of Christison were by no means general and did not fit with contemporary theories of causation and treatment.[7]

Christison told the Medico-Chirurgical Society of Edinburgh in May 1847 that from September 1846 many cases of scurvy had appeared 'throughout the general population of the city, both among the working classes, and even in the middle ranks.'[8] His first case, a master upholsterer, was seen in mid-September and by December he had been asked to see 'two other cases in the middle ranks of society'. The case of the master upholsterer had puzzled him, and it was not until he was consulted about scurvy at Perth prison in October that the diagnosis became clear. The three men of 'the middle ranks' had lived for many months almost entirely on bread and meat, coffee and tea, without fresh vegetables, milk or malt liquor.

The first 'working class' case was a shoemaker, age thirty six, admitted to the Edinburgh Royal Infirmary on 17 February 1847 with 'spongy, livid bleeding gums, pains, stiffness, and oedema of the limbs, purple petechiae on the arms and thighs, extreme feebleness and a remarkable waxy paleness of complexion.' For seven months he had lived on bread and coffee with sugar but no milk. He was prescribed lemon juice, turnips and carrots, extra meat, wine and the Infirmary Common diet, namely bread and coffee for breakfast, bread and broth for dinner, and bread and tea for supper. On this regimen he recovered and went home after forty four days.

Christison commented that 'during thirty years… since I have been attached with little intermission, in one capacity or another, to the Royal Infirmary of this city, I have not seen a single case of scurvy in its wards until February last.' By late May 1847, 149 patients with scurvy had been admitted: railway labourers, a master upholsterer, a blacksmith, two shoemakers, a tailor, a tanner, a porter and a barber. He emphasised the lack of milk in their diet but made no reference to potatoes. Milk was scarce at this time because of an epidemic of pneumonia in milch cows, which Christison attributed to 'the epidemic constitution' affecting humans and animals alike but 'by far the most remarkable circumstance connected with the health of the community has been the appearance of scurvy.'[9]

A Royal Infirmary ward journal, maintained by a senior medical student, (in this instance HD Littlejohn, later Sir Henry Littlejohn (1826–1914), Edinburgh's first medical officer of health), recorded the history of a 50-year-old tailor, Donald Baillie, admitted on 23 April 1847:

> 'he had been much exposed to privation during the last few months from want of employment and his food consisted entirely of bread and coffee which he had frequently only once a day and never more than twice. He never tasted animal food or milk.'[10]

There was no record of treatment or of diet prescribed but analysis of the Infirmary full diet shows that it provided 65.59 mg of vitamin C daily, sufficient to cure scurvy and so it was that he went home cured on 10 May 1847.[11]

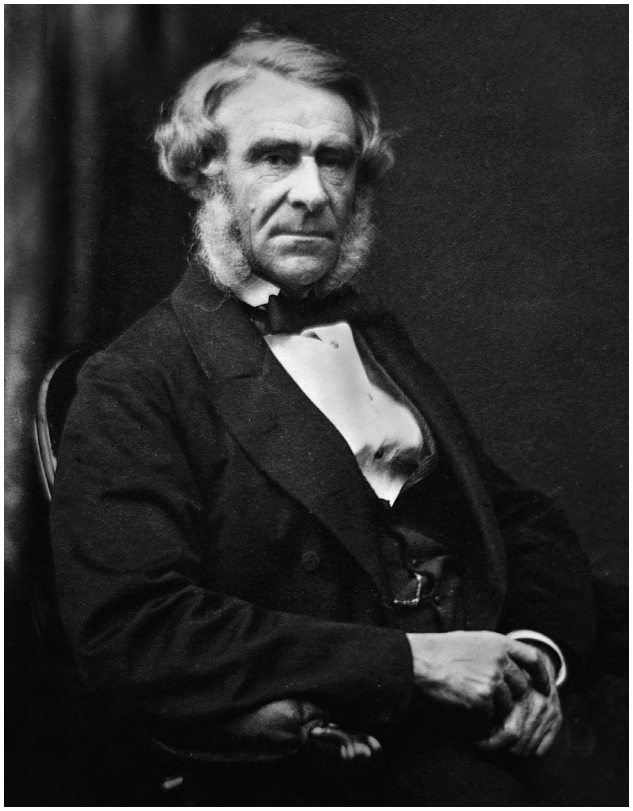
The full diet was one of eight arranged in 1843 by Christison but there is no indication as to whether a specific diet was prescribed for certain illnesses or whether one diet was thought to be more nutritious than another, but the full diet must have been close to the top of the nutritional scale. That so many diets were considered necessary implies that adequate nutrition was seen to be an important part of treatment.

Of the seventeen men with scurvy in Ward 1 at this time, eleven were railway labourers and six local working men.[12] Christison wrote:

> 'who could have expected such a disease as scurvy among the labourers on our railways, men mostly in the prime of life, engaged in an athletic occupation, working in the open fields and breezy moors, earning ample wages, and whose extravagance in good living was a frequent subject of remark in their neighbourhood.'[13]

The ward clerk commented that two local men with scurvy, a weaver and a blacksmith, belonged to trades known to be affected by the recession. Among the others was a weaver who had 'lately been working on the railway', a collier unable to work through injury but supporting his brother, sister-in-law and family on two shillings and sixpence per day, and a fifty five year old shearer who had been living on shellfish for the previous six months.[14] Some of the railway workers were forced by the company to purchase their food and other necessities from the company store, the cost being deducted from their wages. This system, known as 'truck', has been called 'a notorious fraud practised by certain employers upon their servants', a fraud that enabled the employer to avoid paying a full wage and forced the employee to buy inferior goods, often at inflated prices. It has been suggested that

Figure 1 Sir Robert Christison



the Truck Act of 1831, intended to apply in both England and Scotland, 'proved particularly ineffective in Scotland, since to a Scottish lawyer much was unintelligible, in particular with regard to procedure.'[15]

Christison's Milk Deficiency Theory

Christison thought that the diet of Mr Sherry, an Irish labourer, was different to that which he was accustomed to in Ireland, inasmuch as he had no milk. He was aware that Sherry no longer ate potatoes but does not deduce that this might be responsible for the scurvy. The passage quoted below tends to suggest that Christison was adjusting the evidence to fit preconceived ideas. He described the Irish labourers' diet at home in Ireland where they had potatoes, oatmeal, porridge and milk and commented:

> 'It has been commonly represented, and is generally understood in this country, that until the late failure in the potato crop, the food of the Irish peasantry consisted in most parts almost exclusively of that root. But this is one of the many vague statements of practical men, which require to be received with some limitation. On questioning a great number of the labourers who have recently come from all parts of Ireland to work on the railways around Edinburgh, I find that to a man they had an abundance of skimmed milk in their native country.'[16]

Christison concluded that lack of milk was a major cause of scurvy, quoting as evidence the 1840 report of the English Poor Law Commissioners which stated that the food of labourers in Ireland comprised at that time, on average, nine and one quarter pounds of potatoes and two and a half pints of skimmed milk daily. He acknowledged that several factors might have been responsible for scurvy among the railway labourers: the failure of the potato crop, the epidemic among milch cows, the cost of all provisions and the distance from the place of work to their lodgings but he was convinced that milk was a powerful antiscorbutic and that there was a 'tendency of a saccharo-farinaceous diet to engender scurvy.'[17]

Doctors from neighbouring districts had patients with scurvy. Dr Brotherston of Alloa had twenty six cases among ironworkers and labourers and established that their diet had deteriorated in recent months: 'the food had consisted for some time of bread, oatmeal porridge, and tea, without any meat or milk.' Christison used this as evidence that lack of milk and meat was responsible, together with 'a diet too purely farinaceous, saccharo–farinaceous, or saccharo–farinaceous and fatty'. He admitted that potatoes may have prevented scurvy but was convinced that animal nitrogenous nutriment had good antiscorbutic properties. He explained that three 'appearances' of scurvy had occurred: among city tradesmen; among the prisoners in Perth prison and among the railway labourers around Edinburgh. In only one of these groups, the city tradesmen, had the food been defective in quantity whereas in all three groups the food had as its 'main peculiarity… a great want of animal nitrogenous principles.'

He reminded his readers of other scurvy epidemics in which the lack of animal nitrogenous nutriment had been responsible, questioning Dr Budd's article on scurvy in the Library of Medicine in which Budd claimed that 'antiscorbutic properties are possessed exclusively by substances of vegetable origin' and that 'we have no evidence that milk is antiscorbutic.' Christison refuted this, claiming that milk was a powerful antiscorbutic and that the cause was 'a too pure farinaceous aliment.' He also criticised the work of Dr Baly who concluded that the potato had antiscorbutic properties when investigating an epidemic of scurvy in the Milbank Penitentiary in 1840 and 1841 where the prisoners had been well on a diet which included potatoes but developed scurvy when potatoes were withdrawn.[18]

Christison developed his theory of 'a scurvy to be engendered by a diet too purely farinaceous or saccharo–farinaceous or saccharo–farinaceous and fatty' , and on the influence of a due admixture of animal food, and especially of milk, in curing the disease but he also found it necessary 'to advert briefly to certain other views as to its causes and treatment, in order that the precise object of my remarks may not be misconstrued.' These other views included a belief that patients had misrepresented their diets, claiming to have taken meat and milk regularly, when in fact he believed that they had lied, 'unwilling to reveal the state of abject poverty to which they had been reduced.' Christison postulated some other cause, which, in conjunction with a faulty diet, had been responsible for the scurvy seen in Edinburgh and its neighbourhood. He believed that 'no other mode will appear adequate to account for the facts, except the assumption of some particular "epidemic constitution."' He concluded

that 'as various errors in diet may occasion scurvy, so may it be treated by sundry dietetic remedies', including milk and meat, succulent vegetables, lemon juice, oranges and malt liquor among them. Christison who yearly gave a series of lectures on diet to medical students claimed in one of his lectures that scurvy, 'the terrible scourge of fleets, armies, and society at large', was favoured by confinement, want of exercise and by a moist, cold atmosphere but the main cause was a combination of damp air and inactivity, with faulty food, particularly lack of milk, playing a part in the condition also.[19]

Opinion of Christison's Edinburgh Colleagues

It was not until the May meeting of the Edinburgh Medico-Chirurgical Society that the scarcity of potatoes was mentioned, first by Dr Paterson who commented:

> 'that sufficient stress had not been laid upon the absence of potatoes in the production of the epidemic… it was only in this year that there had been an absence of potatoes, and the presence of scurvy. This at least was a very singular coincidence.'

Despite Christison's reputation several of his colleagues were not totally convinced by his 'novel views'. At a meeting of the Edinburgh Society in April 1847 Mr Spence reported cases of scurvy affecting several persons in the middling ranks of society; Dr Hughes Bennett had seen several cases of scurvy at the Royal Dispensary; Dr Paterson had seen three cases; Dr Peddie had treated two cases in the Minto House Hospital and Dispensary and two unnamed physicians had also seen several cases. The diet of the patients Paterson treated in the infirmary had been deficient in milk, potatoes and green vegetables; Dr Hughes Bennett had been able 'to trace the disease in every case to a want of fresh vegetables, more especially potatoes, or to an insufficient diet. In the light of these opinions Christison said that he 'did not wish to have it supposed that, in his opinion, the want of potatoes might not be the cause, or that other antiscorbutics besides milk were useless.' He considered the want of potatoes a most important circumstance.

Scurvy in Glasgow

Dr Charles Ritchie, (1798–1878), physician at Glasgow Royal Infirmary, wrote in May 1847: 'the prevalence among our land population of a distinct disease presenting the precise features, aetiology, proximate nature, and general indications of treatment of the scorbutus of our navies.' He had treated in the Infirmary eighty three cases, seventy males and thirteen females and at the dispensary, thirty nine cases, thirty three males and six females and cited other instances of the condition, which 'some may be disposed to call purpura'. Ritchie concluded that:

> 'the general fact in regard to the food of all was, *that it failed in variety, and in the quantity of its animal constituents,* and, that, in all but a fraction of the cases, in which they were very deficient, *the patients had been exposed for months to a total deprivation of fresh succulent vegetables.*' [Original italics].

Figure 2 Dr Charles Ritchie

He went on to say:

> 'It is familiar to everyone that the failure of the potato crop for two successive seasons, did, with the stunted growth of our pastures and other crops, lead to a rise of between 30 and 40 per cent on all kinds of provisions during last winter. One effect of this was to render all kinds of fresh succulent vegetables unattainable by nearly every class… accordingly of the infirmary patients in this epidemic, about 95 per cent had suffered a total deprivation of fresh succulent vegetables for more than six months.'

The treatment included a diet of oatmeal, milk, animal broth made with vegetables, fresh meat, turnips or carrots, an orange, crystallised citric acid or two ounces of lemon juice and one pint of porter daily. On this regime all except one of the scorbutic patients recovered.[20]

Dr John Steele (1821–1892), Superintendent of Glasgow Royal Infirmary, commented on 'the want of employment and the high price of provisions, to which the lower orders were subjected during the bygone year, increasing their liability to disease.' These 'irregularities' were also the primary cause of the outbreak of scorbutus which 'appeared almost simultaneously in an endemic form in various parts of Scotland.' The average length of stay in hospital was twenty seven days, a figure that does not compare well with the six days described by James Lind.[21]

Scurvy in the Borders

Henry Lonsdale (1816–1876), MD Edinburgh 1838, physician to the Cumberland Infirmary, Carlisle, wrote in August 1847 on cases of scurvy in his locality and among workers on the Caledonian Railway in the south of Scotland. In the southern

part of the county of Dumfries, agricultural workers were the group most affected. Lonsdale confirmed that potatoes had always been a food staple for these farm workers and observed that: 'During three winters the potato crop has been failing, but the supply was never entirely cut off till last autumn and winter.' Dr Bogie of Annan, who had previous experience of scurvy as a surgeon at sea, diagnosed between ninety and one hundred cases of scurvy amongst the pauper class and the workers on the Nithsdale Railway. Dr Walker, also of Annan, had treated a dozen cases. Lonsdale recorded a conversation with Dr Browne of the Crichton Institution, Dumfries, who said that every spring he had scorbutic cases but now (1847) the disease was more common. The consensus among these practitioners was that the lack of potatoes was the chief cause of the outbreak, an opinion with which Lonsdale concurred, blaming an error of diet for the disease, adding 'as vegetables became plentiful, scurvy disappeared from amongst us. I do not hear of fresh cases at this date (9 July).'[22] Andrew Anderson, (1817–1870), surgeon to the Glasgow Eye Infirmary, in a letter to the *Monthly Journal of Medical Science* also took issue with Christison, considering that milk was not 'essentially antiscorbutic, at least not in all cases…' and agreeing with Dr Lonsdale.[23]

Figure 3 William Gregory

Nutritional Science in Scotland

Christison's nutritional knowledge was no doubt influenced by Scottish protégés of Justus von Liebig (1803–1873), Professor of Chemistry at Giessen, whose research in the field of organic, physiological and agricultural chemistry laid the foundations of nutritional science. He established the first practical chemistry teaching laboratory where he developed methods of organic chemical analysis and where 'the university laboratory was transformed… into a major teaching device, into a training ground for practical scientists, and into the home of research schools.' His approach was:

'to assess quantitatively the relation between what was consumed as food, chemically analysed, and what was expended as work and heat in the animal and human body. This concern with work, in relation to physical efficiency, remained central to much of nineteenth century nutritional science, and could be readily adopted by the state to promote its interest in having a strong army and navy and a productive labour force.'[24]

His laboratory attracted students such as Thomas Thomson (1773–1852) and his nephew, Robert Dundas Thomson (1810–1864) who both taught chemistry in Glasgow and Thomas Graham, Lyon Playfair and James Johnston, all members of the 'clan of Scottish chemists'. A colleague of Christison in Edinburgh was William Gregory (1803–1858), Professor of Chemistry, one of von Liebig's 'earliest British disciples' who translated Liebig's works into English as did his successor, Playfair.[24] Lyon Playfair, later Lord Playfair (1818–1898), studied with Liebig, translating Liebig's seminal work, *Organic Chemistry in its Applications to Agriculture and Physiology*, in September 1840 ready for the Glasgow meeting of the British Association for the Advancement of Science. The main thrust of Liebig's work was his contention that fats and carbohydrates were fuel foods and that protein was a plastic food, which formed new tissue as the body grew and replaced the tissue destroyed in muscular work. He taught that work involved the breakdown of muscle and that its repair and renewal required rest and plastic food. These conclusions about the singular qualities of animal protein had long term, perhaps not entirely beneficial, effects on nutritional thought and practice.

Liebig was convinced that animal nutriment was superior to all other food and by far the most valuable item in any diet, a view shared by Christison writing in 'The Proper Food for Man in Various Circumstances of Life' gleaned from 'the best established modern rules in dietaries' he recommended a diet for a person doing heavy physical work, based on the training diet of prize fighters as communicated to Sir John Sinclair of Lybster. The main object, Christison maintained, was 'to throw into the body as much animal food as the stomach can digest.' He recommended beef and mutton as superior to veal and pork; vegetable food was not considered suitable, but could be eaten 'in small proportion, especially potatoes.' Fish was a watery food and was not considered very nutritive. Perhaps his most harmful claim was that:

> 'even under great starvation, man will generally survive an extraordinary length of time, without any other distinct disorder except some diarrhoea, oedema and ulcer of the mouth – provided he be so placed as to be able to struggle for existence by travelling, or otherwise labouring, in the open air; and when death takes place at last, it arises from gradual excessive exhaustion, rather than positive disease.'[25]

Conclusion

The scurvy cases recorded in the *Monthly Journal of Medical Science,* together with press reports amount to around 400 cases in four parts of Scotland: Edinburgh, Glasgow, Ayrshire and Dumfriesshire, but was Christison the able historian the *Journal* assumed him to be?

There is an explanation for the early and frequent appearance of scurvy among the Irish railway labourers. The rapidity with which they developed scurvy was the result of their previous high levels of vitamin C from the enormous amount of potatoes they ate which provided far in excess of the daily requirement of thirty to sixty milligrams. Research has demonstrated that people accustomed to saturated levels of vitamin C become depleted of the vitamin more quickly than those used to a low intake. It has also been shown that 'higher physical activity and higher whole grain consumption have been associated with higher plasma vitamin C concentrations', both likely to have been the case in Scotland. Moreover, the condition of haemochromatosis reputedly has a negative effect on vitamin C absorption thereby increasing the risk of scurvy and it is relevant that the Irish are genetically predisposed to haemochromatosis through a gene mutation. It is not known if Highland Scots are similarly predisposed.[26]

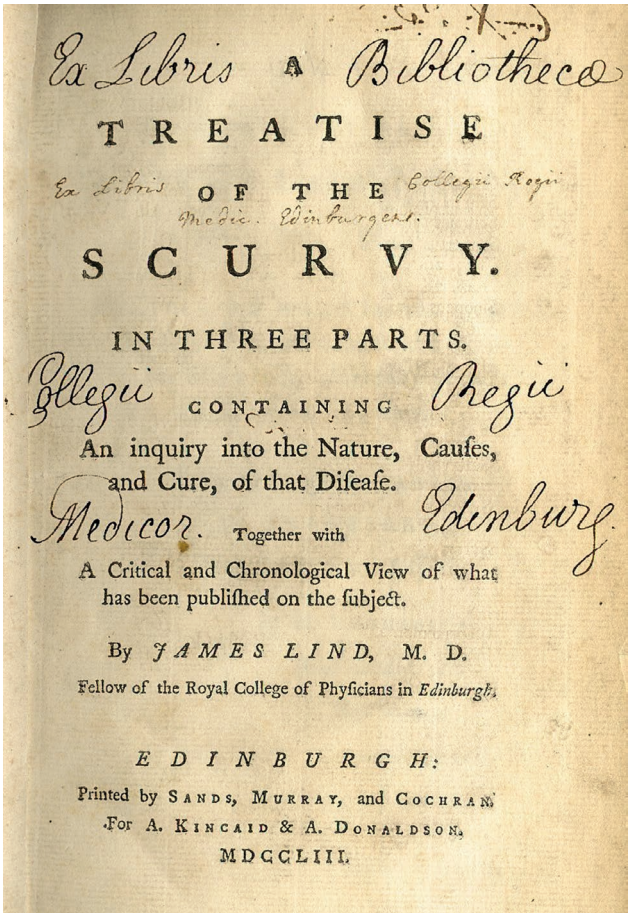
The disease has been known since the thirteenth century, but the complete syndrome became recognised only when long sea voyages of exploration became common, thus explaining how the association with life at sea came to dominate accounts of the disorder. The first true account of the condition, however, described a land based outbreak affecting the French army fighting the Saracens in Egypt in 1249–50.[27] It was the frequency with which scurvy appeared at sea that persuaded many observers that there was a specific association between factors peculiar to life afloat and the condition. This belief was widespread; for example, an 1821 nosology defined scurvy or scorbutus as:

> 'A septic state of the system induced by the excessive use of salted animal food, the want of fresh vegetables, a foul, cold and moist air, and bad water: manifesting itself by general debility and depression of spirits, livid spots on various parts of the body, especially affecting the roots of the hair, an offensive breath, gums spongy, and occasional haemorrhage from the mouth, nose, intestines and other parts of the body.'[28]

The emphasis Hosack placed on salted animal food, foul, moist and cold air, lack of vegetables and bad water, all of which were associated with life at sea before refrigeration enabled a better diet, is evidence of the general belief that these conditions were responsible for disease, a conviction which persisted well into the nineteenth century.

Civilian doctors were slow to accept Lind's work, (Lind himself had doubts about the disease and its cure), because there was little interest in scurvy or its treatment in the first half of the nineteenth century; scurvy was uncommon.[29] Opinion was divided as to whether conditions at sea had so improved that the disease no longer appeared or whether the scurvy described by earlier writers was in fact an entirely different disease.[30]

Figure 4 Frontispiece of Lind's treatise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TREATISE

Ex Libris OF THE Collegii Regii Medic. Edinburgen.

SCURVY.

IN THREE PARTS.

Collegii CONTAINING Regii

An inquiry into the Nature, Cauſes, and Cure, of that Diſeaſe.

Medicor. Together with Edinburg.

A Critical and Chronological View of what has been publiſhed on the ſubject.

By JAMES LIND, M. D.

Fellow of the Royal College of Phyſicians in *Edinburgh*.

EDINBURGH:

Printed by SANDS, MURRAY, and COCHRAN, For A. KINCAID & A. DONALDSON.

MDCCLIII.

In many parts of Scotland, where scurvy had previously been endemic, the disease had disappeared by 1800, at about the time the potato became an important item in the diet. Perhaps it is not surprising that doctors failed to diagnose scurvy in the 1840s; only the oldest practitioners or those who had served at sea had any experience of the disease, and there persisted a conviction that scurvy did not occur on land except in conditions of close confinement as in prisons. It was forgotten that Lind had disproved the theory that there was any difference between sea and land scurvy, stating 'as to the cause of this disease; they are the same on both elements: for it will be fully proved that there is not to be found any one cause of it at sea, which is not also to be met with at land…'.[31] One explanation put forward for these medical shortcomings was that 'medical learning was so constricted by Galen's classical pathology of "humours" that the conception of a deficiency disease was not realised till long after.'[32]

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The images of Christison, Gregory and the frontispiece of Lind's treatise are reproduced courtesy of the Royal College of Physicians of Edinburgh; the image of Dr Charles Ritchie is reproduced courtesy of the Royal College of Physicians and Surgeons of Glasgow.

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