Food, Poverty and Epidemic Disease, Edinburgh: 1840–1850

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Declaration

I declare that this thesis has been composed by me and is entirely my own work

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Abstract

The thesis first examines the link between nutrition and disease, focusing on the poor of Edinburgh during the 1840s, a time of economic depression and food shortage. The development of nutritional science and the level of dietary knowledge amongst the medical profession are considered in the light of current nutritional guidelines. An assessment is made of the relationship between the 1947 scurvy epidemic and nutritional deficiency amongst the poor whose diet is then analysed. Institutional diets from contemporary tables of nutrition are subjected to computer analysis and their significance in terms of nutritional status discussed.

There follows a description of the living conditions of the poor in the Old Town, emphasising the degree of overcrowding from inward migration and loss of housing stock following the demolition of streets and wynds in the course of city improvements and industrial developments. The history of the city’s water supply is explored and the absence of sewage provision is described in conjunction with an account of the foul burn controversy stressing the fact that the lack of water in the Old Town was critical in creating the fetid and hazardous environment where only the most primitive and inadequate methods of sanitation existed.

Finally epidemic disease is studied, concentrating on the fever epidemics of 1841–44, 1847–49 and the cholera outbreak of 1848–49 but reviewing also the lesser epidemics of measles, whooping cough and scarlet fever. The history of the identification of typhus fever, relapsing fever and typhoid fever and their causation is described and the contagion–miasma debate is examined, assessing the contribution of Edinburgh physicians to the question. The lack of statistical information on Edinburgh’s morbidity and mortality is one of the factors discussed in a critical appraisal of the reaction of the medical profession and the city administration to these epidemics and to the social conditions in the midst of which they worked but few lived. In studying the cholera outbreak a database of 740 cholera victims has been prepared from the cholera returns maintained by the Royal College of Physicians of Edinburgh and the information contained therein evaluated.
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Introduction

This thesis is primarily concerned with the poor of Edinburgh during the 1840s, examining their diet and living conditions in order to establish whether there was a link between these factors and the epidemics of infectious disease that appeared in the city with monotonous regularity at this time. The study also examines the position of the medical profession in relation to these outbreaks of disease and their response to them. The scale of these epidemics in a city which was not on the whole industrialised in the way of Manchester or Glasgow is worthy of examination in conjunction with analyses of nutritional status and living conditions. Scholars have noted the association between rapid, massive urbanisation and industrialisation on the one hand and epidemic disease on the other — in the words of George Gordon, ‘cities which were expected to be places of civilisation were increasingly found to be full of the most loathsome problems... the industrial towns’ vulnerability to the trade cycle produced widespread distress of a kind hard to ignore...’. ¹ Hitherto the social deprivation associated with increased morbidity and mortality in a city with Edinburgh’s particular structure has not been subjected to scrutiny.² Nutritional deficiency disease in the city will also be explored, examining the epidemic of scurvy in the wake of the potato crop failure and concentrating on a computer analysis of diets of the period. It is not intended in this thesis to consider the incidence and causation of the chronic, endemic disease, tuberculosis, which remained a scourge for generations, but instead to concentrate on the acute infections which took epidemic form, particularly fever and cholera, and to review the less dramatic but nonetheless lethal outbreaks of infections such as measles, whooping cough and scarlet fever. The imprecision of the terms used for tuberculosis does not permit a detailed analysis of its effects; for example, in 1841 St Cuthbert’s burial

ground recorded the following deaths which may have been caused by tuberculosis: decline; water in chest; consumption; debility; water in head; inflammation lung; inflammation chest; decay; inflammation of brain; bowel hives. Flinn argues that 'as killers, however, both cholera and typhus were dwarfed by tuberculosis; and tuberculosis scarcely stirred the imagination of any social group in this period. It was so much a part of life, so inevitable, so little understood, that it was accepted mutely.'

Edinburgh's mid nineteenth century social problems have not received the attention that they merit, possibly because of the city's reputation as being relatively immune from the ills that were common in industrial cities with large populations engaged in manufactures. The historiography of the city has focussed therefore on other aspects of the city's development, for example, the development of the New Town in Youngson's The Making of Classical Edinburgh and more recently Richard Rodger's comprehensive account of Edinburgh's expansion in the second half of the nineteenth century, The Transformation of Edinburgh: Land, Property and Trust in the Nineteenth Century. These works did not set out to be analyses of social conditions amongst the poorest of society although both works touch on the subjects of city improvement, water and building. Rodger does however make the point that a considerable number were employed in industry and accordingly were subject to the same cycles of commercial depression as those labouring in the factories of Glasgow or Lancashire. Other earlier writers preferred to describe the more picturesque and quaint aspects of the city and its inhabitants, with a tendency to romanticise, ignoring the appalling overcrowding in the Old Town. The inclination began early in the century with writers such as Robert Chambers who in his Traditions of Edinburgh of 1825 wrote fondly of times past, making no comment on the squalor that had replaced the charming buildings which he remembered so well and was continued well into the second half of the century by Dr John Brown whose essays on the city

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3 Edinburgh City Archive, St Cuthbert's Churchyard Cash Book, 1841-49.
and its personalities are charming vignettes. The popularity of these books has left a legacy in which the social problems of the nineteenth century have been largely neglected by historians and other commentators.

There have been studies, however, which have examined distinctive features of Edinburgh’s social development focussing on living conditions; one of the most disturbing of these for the middle classes of the city was written by Edinburgh’s first Medical Officer of Health, Dr Henry Littlejohn, as the *Report on the Sanitary Condition of the City of Edinburgh*. The difference between this report and earlier descriptions of the worst streets of the Old Town lay in Littlejohn’s careful use of statistical data to show that areas with the highest population density had the highest rates of morbidity and mortality; earlier works, such as Dr George Bell’s *Day and Nights in the Wynds of Edinburgh* and *Blackfriars Wynd Revisited*, were dramatic and no doubt accurate descriptions of atrocious living conditions and were intended to shock the middle classes into greater charitable efforts. Many were written to gather support for societies with specific objectives; Dr Bell had connections with a temperance organisation with common aims to those expressed in the pamphlet series, *Social Reform*, produced by the Society for the suppression of drunkenness. The tendency of the poor to spend what money they had on spirits rather than on food was a common theme in these polemics, betraying at least an awareness of the value of a good and wholesome diet as well as a willingness to criticise the class being condemned.

During the 1840s Edinburgh, like many other areas of Britain, was affected for a period of time by a nutritional deficiency crisis caused by the potato blight. This occurred when the living conditions of the poor were deteriorating and when a series of epidemics of fever and other infections were responsible for a dramatic increase in sickness and death. The thesis will examine the relative contributions of nutrition and

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9 *Social Reform*, (Edinburgh, 1851).
environment to this increase in mortality and although it is not the intention to advance evidence in support of either side in the debate briefly summarised below, it is hoped that an investigation of the possible link between nutrition, poverty and epidemic disease in Edinburgh during the 1840s may shed some light on the controversy. The debate referred to is centred on the McKeown thesis which attributes the decline in mortality in England and Wales between 1850 and 1914 to improved living standards, particularly better nutrition. The revisionist school take a different view, ascribing the reduction in mortality to the public health movement and locally administered preventive health measures which they claim were successful in combating the ills of urban congestion caused by industrialisation.

The first section of the thesis will therefore concentrate on nutrition, a term which in this study means diet or food intake, while nutritional status means the balance between food intake and the demands on that intake by the body's metabolism, the energy expended in physical and mental activity and that used in fighting infection whether acute, chronic, viral, bacterial or parasitic. Scholarly interest in diet and research into the nutritional status of specific populations in Scotland has been sparse — certainly when compared to the attention paid to English and Irish nutrition. Among the early studies on English diet were those of Dr Jonathan Pereira and Dr Edward Smith in the nineteenth century at a time when there were no equivalent Scottish publications. Descriptions of rural diet in Scotland in the late eighteenth and the first half of the nineteenth centuries can be found in the two Statistical Accounts and details of country diet is tabulated in the answers given to the Scottish Poor Law Commission Enquiry of 1842. It is much more difficult to find

13 J.Pereira, A Treatise on Food and Diet, (London, 1843); E.Smith, Practical Dietary for families, schools, and the labouring classes, (London, 1864).
descriptions of urban diet in Scotland, particularly that of the working class, and references to the food eaten by the poor of Edinburgh are non-existent. This is in contrast to the analyses of English urban diet in the classic work *The Englishman's Food: A History of Five Centuries of English Diet* written by Drummond, a biochemist, and Wilbraham, a nutritionist, first published in 1939.\(^\text{15}\) Kitchen and Passmore, two Edinburgh medical researchers, in 1949 published a monograph entitled *A Scotsman's Food* which attempted, less successfully, to do for Scottish diet what Drummond and his colleague had done ten years earlier, the content of their work being to a large extent dictated by the availability of primary source material. There have been other scholarly works on British nutritional history containing chapters devoted to Scottish food and diets: Professor R.H.Campbell, an economic historian, analysed Scottish eating habits in *Our Changing Fare*, stressing the variations in regional diets and criticising the imprecision of most of the dietary detail in the *New Statistical Account* but his is a rare contribution to the historiography of Scottish nutrition.\(^\text{16}\) D.J.Oddy and J.Burnett are two English historians whose writings on diet in a British context have included references to Scotland but the difficulty under which they laboured is plain when their bibliographical study of British diet since industrialisation is reviewed. The authors imply that the lack of primary sources for British food history compared to those available to their European colleagues is a major handicap, explaining that 'the researcher is obliged to extract bits and pieces here and there from secondary sources.'\(^\text{17}\) The position in Scotland is even less helpful and the detail that is accessible generally concerns rural diet with little accurate information on the day to day diet of the urban poor, an omission that says much about nineteenth century attitudes to the new and often deprived city dwellers.

The review of nutrition that follows will draw on the secondary sources already mentioned but will also make use of other primary sources and contemporary

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descriptions in order to build a picture of the types of food likely to have been eaten by the Edinburgh poor. A particularly valuable source, hitherto unused, discovered among the papers of Sir Robert Christison, professor of materia medica in Edinburgh University from 1832 until his retirement forty-five years later, contains several dietaries of the period among which are two from the Edinburgh Infirmary and the Edinburgh Charity Workhouse. These are similar in structure to the diets which were analysed by Drummond and his colleagues and recorded in Appendix A of The Englishman's Food in which the levels of the main nutrients were calculated and compared to recommended norms. A similar but more complex analysis was carried out by J.C. McKenzie on the quality of diets in Manchester and Dukinfield in 1841, estimated by William Neil and presented as a paper to the statistical section of the British Association, later published in the Journal of the Royal Statistical Society in 1841–2.18 Based on Neil's work which 'never dealt specifically in terms of food quantities', McKenzie calculated from family expenditure on food the total calorie content, the amount of protein, iron and vitamin C, forming conclusions as to the adequacy of working class food intake, basing his calculations on the nutrient content of present day foods according to McCance and Widdowson. This kind of assessment, although useful, has limitations, in that food quantities were extrapolated from the prices of food available and the wages of those buying the food, a drawback that is not present in the detail of the diets recorded by Christison. These have been subjected to computer analysis, a technique that up till now has not been used in the assessment of past nutritional standards in Scotland.19

Amongst the factors of importance in any discussion of nutritional status during this period is the loss of the potato crop as a result of fungal infection. This had catastrophic consequences for the Irish peasantry and if, in the Scottish highlands and islands there was no comparable mortality crisis, the prolonged potato famine had far-reaching social and economic consequences. The historiographical focus on

19 Edinburgh University Special Collections, Christison Papers, Table of Nutriment in Various Dietaries, (Edinburgh, 1854).
these nutritional disasters in specific parts of the British Isles has diverted attention from the effects of the loss of the potato in other areas of Scotland and England and has overlooked the importance of the potato to the urban working class. The significance of the potato as an urban dietary staple and the consequences of its loss became apparent when studying reports of scurvy in Edinburgh in the course of research into the medical aspects of the potato famine in Ireland and Scotland and some of the results of this study will be introduced.20

The relationship between nutritional status and infectious disease has until relatively recently been accepted as a matter of common sense. The link has now been established scientifically by advances in immunological and nutritional research stimulated by the Aids epidemic. The writer of the foreword to Nutrition and Immunology: Principles and Practice, a distinguished worker in the field of nutrition, immunology and infection, stressing the importance of the two disciplines, speaks of the reality that in Africa 'large sums of money are being spent on nutritional intervention in the hopes of improving immune responsiveness.'21 It is not intended to review in detail current theories of the interaction between malnutrition and the immune response but to concentrate on specific deficiencies, bearing in mind that 'human malnutrition is almost always a syndrome of multiple nutrient deficiencies compounded by genetic influences, age, gender, and the superadded effect of infection.'22 One of the complications of research into this link is that those who are malnourished often exist in overcrowded, insanitary conditions and it is difficult, if not impossible, to separate the effects of poor food from those of a poor environment. These themes are explored by Treble in Urban Poverty in Britain 1830–1914 in a chapter entitled ‘The Socio–Economic Characteristics of Poverty 1830–1924: Food and Housing’ and by Hamlin in Public Health and Social Justice in the Age of Chadwick. Anne Hardy highlights the dilemma in her paper ‘Urban Famine or Urban Crisis? Typhus in the Victorian City’ and this idea is also explored

by Rotberg and Rabb, in ‘The Relationship of Nutrition, Disease and Social Conditions: A Graphical Presentation’ in *Hunger and History: The Impact of Changing Food Production and Consumption Patterns on Society*.23

Whereas the relationship between malnutrition and disease is well established the effect of a poor environment on health is less clear cut. Uncertainty in the 1840s about these factors led to the uncritical acceptance of erroneous theories of fever causation which were only shown to be wrong with the development of bacteriology. The concepts of contagion and miasma are examined with reference to the contributions of Arnott, Southwood Smith and Alison and the eventual success of the sanitary movement. M.W.Flinn’s edition of the *Report of the Sanitary Condition of the Labouring Population of Great Britain* is an invaluable depiction of the urban environment and Pickstone in ‘Dearth, dirt and fever epidemics’ discusses what he calls two views of public health, that of Chadwick and the ‘ultra-sanitarians’, and the death model in which agricultural failures are seen as the cause of fever.24 Edinburgh’s congested Old Town is the focus of the chapters describing living conditions, sanitation and the water supply. Of considerable importance in this analysis are the Minutes of the Police Commission; the Minutes of the Cleaning Committee; and the Edinburgh Council Records.25 The scarcity of water in this part of the city is in marked contrast to the wealth of material on the history of the city’s water supply, including books written by Lewis and Colston, both authors having at one time served on the Edinburgh Town Council.26 The papers relating to the “foul burn controversy”, although at the time a side issue, are an important source of


25 Edinburgh City Archive, Minute Books of the Police Commission; Minute Books of the Police Commission Cleaning Committee; Edinburgh Council Records.

information about Edinburgh’s sanitation and reveal a great deal from both sides of the argument about contemporary views on sewage held by doctors and others.\textsuperscript{27} Surviving papers, such as the open letter dated 1856 from Henry Johnston to the Council, concerning the condition of the houses and drainage in the Lawnmarket, High Street, Canongate and Cowgate with an Appendix in which 159 closes are described in considerable detail are invaluable giving a picture of the squalor and dangerous state of some of the worst affected closes.\textsuperscript{28}

Johnston in his introduction refers to the different responsibilities of the Town Council and the Commissioners of Police, writing: ‘although the subject of this communication belongs more properly to the province of the General Commissioners of Police, yet I have preferred to address it to you...’.\textsuperscript{29} Until the Police Commission was created in 1771 ‘the public concerns of the city were almost entirely under the control of the Town Council’ but thereafter two departments of local government co-existed with the Council located in the City Chambers and the Commissioners in Parliament Square. The concept of police and more specifically medical police was of European origin, based largely on the work of Johann Peter Frank (1745–1821), who in the late eighteenth century ‘collected in his System einer vollständigen medizinischen Polizey (1779–1819), the most important principles of State hygiene’, a system of a complete medical police.\textsuperscript{30} Roberton, an Edinburgh physician, wrote A Treatise on Medical Police and on Diet, Regimen, etc (1809), in which, discussing the causes of diseases among other matters, he criticised the filth accumulating in the east of the city and the failure to water the streets in summer because of water shortages.\textsuperscript{31} It is significant that Roberton combined dietary advice with the work of medical police whose role was largely in disease prevention by ensuring that towns

Other works include Q.J., The Supply of Water to Edinburgh, (Edinburgh, no date) and A.Leslie, An Account of the Water Supply of Edinburgh, (Edinburgh, 1883).
\textsuperscript{27} W.Tait, An Examination of the Statements Contained in the Papers Relating to the Fetid Irrigations around the City of Edinburgh, (Edinburgh, 1839); R.Forsyth, Foul Burn Agitation, (Edinburgh, 1840).
\textsuperscript{28} H.Johnston, Letter to the Lord Provost, Magistrates, and Council of the City of Edinburgh on the State of Closes in the Lawnmarket, High Street, Canongate, and Cowgate, (Edinburgh, 1856); Royal College of Physicians of Edinburgh, Cholera Returns 1848–49.
\textsuperscript{29} Johnston, Letter to the Lord Provost, Magistrates, and Council of the City of Edinburgh, p.3.
\textsuperscript{31} J.Roberton, A Treatise on Medical Police and on Diet, Regimen, etc., (Edinburgh, 1809), volume 2, pp.278 & 290. Comrie says that this was the first treatise in English on the subject.
and cities were cleaned, that nuisances were not allowed to develop and that "fever nests" were fumigated. The Police Commissioners, whose jurisdiction extended not only over the burgh but also over the new suburbs, dealt with cleansing, lighting and watching and had legal power to levy rates but as the century wore on their work began more and more to resemble that of medical police. The work of the Commissioners was devolved to a Cleaning Committee whose weekly deliberations are recorded in their minutes kept in the Edinburgh City Archive. These are an important source, recounting the day to day management of street cleaning and manure disposal and of the reactions of the administration to the epidemics of fever and cholera.

The lack of data on morbidity and mortality hinders an accurate assessment of the effect of fever and other infectious diseases and the reasons for this absence of statistics are explored by reviewing the work of Cleland, *A Historical Account of Bills of Mortality*; Hawkins, *Elements of Medical Statistics*; and the Report of a Committee of the British Association for the Advancement of Science on the Vital Statistics of Large Towns in Scotland*. There are contemporary papers with some statistical detail written by Alison, Christison, Robert Deuchar, the Secretary of the Fever Board, and the reports of James Stark, Edinburgh’s first official recorder of mortality, which were published in the *Edinburgh Medical and Surgical Journal* between 1846 and 1849. The cash books of St Cuthbert’s Churchyard in the Edinburgh City Archive are in effect a detailed register of burials in which age, occupation, cause of death and type and cost of interment are recorded. Although the

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33 Edinburgh City Archive, Minute Book of the Cleaning Committee, 1840–1850.


number of burials is only a small proportion of the total Edinburgh mortality, the books are a useful source in that they show trends which in turn reveal the onset and duration of epidemics. The change in the terms used for diseases causing death can be ascertained with, for example, the first mention of influenza being in February 1842. The ages of those dying from measles and whooping cough are revealed and the magnitude of child mortality is demonstrated.

A significant discovery was the day-to-day record of patients treated at home and in hospital of all those who contracted cholera in the epidemic of 1848–49. The two volumes in the archives of the Royal College of Physicians of Edinburgh contain the names, ages, addresses, occupations, type and state of accommodation of 740 cases with replies to enquiries as to whether the victim had been in contact with other cholera sufferers, their habits and state of nutrition, length of illness, the outcome, treatment and whether a post mortem was performed. These have been recorded in a database and since a full detailed analysis of their contents from a social, epidemiological and medical historical viewpoint is beyond the scope of this thesis, the volumes will form the basis of further collaborative research. The minutes of the College are key sources of information as to how the Fellows responded to the recurrent fever epidemics, to the imminent arrival of cholera and to local and central management of these mortality crises.

In assessing the nature of cholera epidemics and the history of the disease the works of Pollitzer, De, Ackerknecht and Speck have been particularly useful. The accounts of the earlier British outbreak by R.J. Morris and M.Durey are valuable as is Margaret Pelling’s study, Cholera, Fever and English Medicine 1825–1865. The monograph by Asa Briggs compares the reaction to cholera in Britain and France while Richard Evans’ analysis of cholera in Hamburg is a wide ranging and comprehensive survey. Finally the Town Council and Police Commission cleaning committee

records for the 1840s have been studied, noting the disquiet expressed by citizens to the Commissioners about the dangers from nuisances such as slaughter houses, pig sties, byres, privies, necessaries, and accumulations of dung and excreta in stairs, closes, wynds and streets. The first hard evidence available to the city authorities and the medical profession of the association between epidemic disease and squalor is contained in the College cholera returns. If the evidence was lacking the link was well known; the committee asked Dr Glover to provide details of ‘the localities in which fever most prevailed and showing how far the cholera has followed the tract of that disease.’

The cholera returns bring into view the circumstances of women in the city. Except for burial records, women appear as shadowy figures, if they appear at all, whereas in the College returns female occupations are recorded if the woman was single or if she was a widow with an occupation. As Neff has stressed, ‘women as workers did not harmonise with the philosophy of the Victorians’, the ideal woman was a wife and mother but when this role evaded them they became marginalised, their economic status declined, they became malnourished and many were driven to prostitution. The difficulties in establishing ‘the pattern and location of women’s employment’ is noted by Eleanor Gordon when she stresses the unreliability of the decennial censuses as a source of information, where work was defined as full–time, permanent and outside the home. Seasonal employment of women was common at this time and it follows therefore that seasonal unemployment was also common; where there were job creation schemes for unemployed men there were none for women, perhaps explaining why there were 10,000 female paupers in Edinburgh in the late 1840s. The women whose occupations were recorded in the cholera returns were generally unmarried or widowed and included servants, outworkers, seamstresses, washerwomen and prostitutes. The cholera returns provide invaluable


37 Edinburgh City Archive, Minute Books of the Cleaning Committee, 15 December 1848.
background information on the position of women in the Old Town, on occasion supplying anecdotal reports on nutrition from attending doctors who were aware of the link between lack of food and ill health. The fact remains, however, that the nutritional status of women cannot be separated from that of men and children nor can their living conditions be assessed in isolation or their fever case fatality calculated. Unlike the situation with fever, however, their case fatality can be ascertained from the cholera data and will be recorded. Further work on the situation of women based on this source will be carried out in the collaborative investigation already mentioned.

There are no equivalent records of those who contracted fever during the epidemics which were the single greatest cause of sickness and death during the 1840s. The minutes and records of the Fever Board, the body with responsibility for management of the epidemics, cannot be traced but figures for morbidity and mortality can be acquired from a variety of other sources. The published annual reports of the Royal Infirmary, the two Public dispensaries and the contemporary analyses of the epidemics by medical practitioners such as John Rose Cormack, Robert Paterson, Robert Christison and others furnish sufficient detail of the size and nature of the outbreaks.41 The Ward Journals of the Royal Infirmary, although incomplete, are a register of the patients admitted to these wards with detail of diagnoses, age and occupation. Secondary sources studied include the reviews of Creighton and Murchison which, although written some years after the events

41 Edinburgh University Library Special Collections, P.107/1, Report by the Managers of the Royal Infirmary for the Year from 1 October 1840 to 1 October 1841, (Edinburgh, 1841); Edinburgh University Library Special Collections, Statistical Tables of the Royal Infirmary, 1841–42; Edinburgh University Library Special Collections, P.107/12, Annual Report for 1843 of the Royal Public Dispensary of the City and County of Edinburgh, (Edinburgh, 1843); Edinburgh University Library Special Collections, P.115/19, Annual Report for 1845 of the Royal Public Dispensary of the City and County of Edinburgh, (Edinburgh, 1845); Edinburgh University Library Special Collections, P.115/20, Annual Report for 1846 of the Royal Public Dispensary of the City and County of Edinburgh, (Edinburgh, 1846); Edinburgh University Library Special Collections, P.107/13, Annual Report of the Edinburgh New Town Dispensary, (Edinburgh, 1842); J.R.Cormack, Natural History, Pathology, and Treatment of the Epidemic Fever at Present prevailing in Edinburgh and other Towns, (London, 1843); R.Paterson, 'An Account of the Epidemic Fever of 1847–48, more especially as deduced from the Statistical Records of the Extra Accommodation with a Suggestion and Plan for Improved and more Economical Temporary Erections for Future Occasions', Edinburgh Medical and Surgical Journal,
described, are useful if not always entirely reliable.\textsuperscript{42} This material will be used in
the review of the fever epidemics, showing how the change from the use of umbrella
term “fever” to the more accurate typhus, relapsing fever and typhoid fever took
place and explaining why the recognition of these diseases is interesting in terms of
the involvement of Edinburgh medicine and how slow many physicians were in
adopting the new terminology. The puzzle of the apparent rarity of typhoid fever in
Edinburgh will be investigated, contrasting this with the situation in other cities.

The importance of fever and cholera to the subsequent changes in medical and
administrative response is reflected in the amount of space devoted to them. The
other epidemic diseases, smallpox, measles, scarlet fever and whooping cough are
examined but their impact, although significant, was less than that of typhus and
cholera and arguably had less influence on perceptions of disease and the
development of public health as a medical speciality and an administrative
responsibility. It was fever above all others that was feared by the middle classes
whose apprehensions were often exploited by charities seeking donations as in the
appendix to the \textit{Third Report of the Edinburgh Lodging House Association}; a letter
from an Edinburgh clergyman to the secretary stressed the absence of fever from the
association’s two lodging houses when ‘fever has been raging’ and the importance of
this contribution to the public health.\textsuperscript{43} The cholera epidemic of 1848–49 for the first
time brought together representatives of the medical profession, the town
administration and the Parochial Boards. This unusual degree of co-operation,
despite occasional instances of disagreement, resulted in an agreed plan of action to
deal with the threat and an official recording of cholera cases.

The thesis is therefore concerned with the effects of food, poverty and epidemic
disease on the population of Edinburgh during the period 1840–1850, focusing on the
poor of the city and examining how their nutritional status and living conditions

\textsuperscript{70} (1848), pp.371–407; Christison, ‘On the Changes in the Constitution of Fevers, 3, (1857–58),
\textsuperscript{42} C.Creighton, \textit{A History of Epidemics in Britain}, (London, 1965, reprint of 1891 edition); C.
\textsuperscript{43} Edinburgh Central Library, \textit{Third Report by the Committee of the Edinburgh Lodging House
Association}, (Edinburgh, 1848), p.10.
contributed to the high levels of morbidity and mortality. Particular emphasis will be placed on new primary source material relating to nutrition, nutritional disease and on the cholera returns of 1848–49, an invaluable source whose contents will be presented in database form. The efforts of the medical profession to come to terms, both practically and rationally, with their new quasi public health role are described as are their failures to adapt to changing circumstances.
Chapter One

The Science of Nutrition

This chapter deals with a number of topics concerning the development of nutritional science. Advances in food chemistry will be reviewed and the attitude of doctors and scientists in Edinburgh to this new discipline will be explored in order to establish the extent of interest in and knowledge of food during the middle of the nineteenth century. The chapter will continue by reviewing current nutritional science, dietary recommendations and essential dietary constituents and will conclude by examining the effects of living on a diet lacking certain essential nutrients. The interaction between malnutrition and disease has now been accepted as a significant factor in the increased morbidity and mortality from infectious diseases and the aim of this chapter therefore will be to describe current nutritional recommendations emphasising the crucial role of certain essential nutrients and the inevitable sequelae which result from deficiency.

The concept of a relationship between poor diet and increased susceptibility to disease has long been accepted in a general way; those who are poorly fed are more liable to infection and are less able to fight that infection once it is established. The idea that an improvement in nutrition might have had a major impact on demographic change was first articulated in 1976 by McKeown when he argued that this was the main reason for the increase in expectation of life at birth and the growth of population in Britain during the eighteenth and nineteenth centuries. Until ‘the McKeown thesis’ it had been accepted, fairly uncritically, that sanitary improvement and advances in medical science had been responsible for these changes. For several years the improved standard of living explanation was in its turn accepted until the 1980s when several authorities put forward other reasons for the apparent connection between dearth and disease. Galloway demonstrated a correlation between epidemic

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disease mortality in London and high grain prices but suggested that the link could be explained by the movement of people into the city because of poor harvests thereby increasing the risk of infectious disease through overcrowding.\(^2\) Szreter, in a critical evaluation of McKeown's research, suggested 'an alternative interpretation of the same epidemiological evidence then forms the basis for a revisionist account which directs attention to the leading role played by the public health movement and its locally administered preventative health measures in combating the urban congestion created by industrialisation.' Szreter, in support of his contention, first cited the work on English population history of Wrigley and Schofield, which established that the steady improvement in expectation of life at birth beginning around 1730 ceased in the 1820s and did not resume until the late 1860s. Secondly, he quotes the anthropometric research of Floud, Wachter and Gregory, which demonstrated that the increasing average height attainment (an indication of improving nutritional status) of cohorts of children born in successive decades in the United Kingdom during the late eighteenth and early nineteenth centuries was not maintained from the 1820s to the 1850s. For these decades there was an actual fall in height attainments relative to the preceding decades.\(^3\)

It is not the intention of this thesis to advance evidence in support of either side in this controversy, but it is hoped that a study of the relationship between nutrition, poverty and disease in Edinburgh during the 1840s may shed some light on the debate. The aim will be to concentrate on the emergence of diseases caused by sub-standard nutrition and to examine the possibility that there was an increase in morbidity and mortality from faulty immune responses caused by malnutrition. It is evident, however, that demonstrating an association between nutrition and disease


does not necessarily confirm a causal link. As Livi–Bacci pointed out ‘...recognition of these two relationships is not proof in itself of the existence of a direct correlation between demographic or mortality cycles and the fluctuating availability of sustenance’ but in the case of a well-proven deficiency disease this reservation does not apply. ⁴ During the period under review ‘the fluctuating availability of sustenance’ was associated with first, economic hardship during cycles of industrial depression and secondly, with the scarcity of food following the potato famine. Edinburgh in the 1840s, and for many decades thereafter, was not thought of as an industrial city—as Gray comments ‘non-industrial employments have always been regarded as a distinctive feature of Edinburgh’. He goes on to quote from Groome’s *Ordnance Gazetteer of Scotland* (1886) in which is found the following passage:

> The city has a calm, steady character, in keeping with the predominance of legal, educational, literary and artistic pursuits, from which it derives its chief maintenance, and contrasts broadly with the fluctuations, excitements, and mercantile convulsions, which produce so much vicissitude in manufacturing towns.⁵

R.J.Morris draws attention to a different aspect of the city, writing ‘Edinburgh was a service and trading centre but it also gathered a significant industrial sector.’ ⁶ Although the city may have escaped significant industrialisation nevertheless there were many who were employed in small to medium sized industrial units and who were just as vulnerable as factory workers to ‘mercantile convulsions.’ Edinburgh was, therefore, a city with its own distinctive features and social problems and if an association between nutrition, morbidity and mortality does exist, the decade under review might provide evidence in support of such a connection. The period between 1840 and 1850 was, in Edinburgh (as elsewhere in Britain), a decade when epidemic disease was unusually common, there were typhus and relapsing fever epidemics in 1841–1844, typhus, relapsing fever and typhoid fever in 1847–1849, influenza in 1847–48, epidemics of measles and whooping cough in 1846–47 and cholera in 1848–49. It was also a time of economic hardship with a downswing in the business

cycle which began in 1837 and lasted until 1842. A radical Edinburgh periodical declared in 1840 that 'much distress prevails; thousands are everywhere out of employment, and subscriptions have, in many places been raised to protect the people from absolute starvation.' The potato blight caused major hardship between 1845 and 1847, although its effects lasted longer in the Highlands and Islands, continuing into the 1850s. In 1847, there was another cyclical business depression and, although Edinburgh was not industrialised to anything like the same extent as Glasgow, nonetheless, the city suffered economically during the recession.

Urbanisation in Scotland was rapid. It has been estimated that of the ten largest Scottish towns in 1851, immigration accounted for 53 per cent of the inhabitants, the figure for Edinburgh being 49.7 per cent, both figures being based on the 1851 census. The increase in population of the city (including the extended royalty) from 82,560 to 160,511 between 1801 and 1851 indicates a significant degree of urbanisation. How much of this increase was due to permanent inward migration cannot be calculated; census returns during the first half of the century 'were in essence statistical summaries', information about place of birth was not at the time thought to be important and enumerators simply recorded residence at the time of the census. Devine and Withers who have studied temporary and permanent migration from the Scottish highlands to lowland towns and cities make clear the difficulties experienced in trying to make a quantitative analysis of this movement of people.

It is difficult to quantify the extent of this migration but it is clear that this movement of people to a new and strange environment involved enormous social changes in day to day living and particularly in eating habits. Oddy says that 'the displacement of

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7 Tait's Edinburgh Magazine, March 1840, p.204.
11 D.V.Glass, Numbering the People, (Farnborough, 1973), p.93.
traditional foods from the diet and the alteration of food patterns is an essential part
of the process of social change that accompanies the spread of industrialisation and
urbanisation. The three factors referred to above, unemployment, food shortages as
a result of poor harvests in the early 1840s and the loss of the potato crop in the
middle years of the decade together with dietary change consequent on urbanisation,
were in action in Edinburgh during the 1840s; therefore, nutritional deficiencies are
likely to have occurred. There were physicians and other professionals in the city
who were concerned about the effect of the poor diet on which many were forced to
live and the contribution of these men will be addressed later. However, it was not
only the quantity of food that was increasingly of interest, the quality of food was
also being explored at this time using the new techniques of food analysis. These
analytical methods owed a great deal to the new discipline of organic chemistry
developed by men like Justus von Liebig (1803–73), professor of chemistry at
Giessen, whose research in the field of organic, physiological and agricultural
chemistry laid the foundations of the science of nutrition. The section that follows
will review the nineteenth century advances in nutritional chemistry and physiology,
assessing the position of the new science in Edinburgh.

The History and Development of the Science of Nutrition

This section is primarily concerned with the development of the science of nutrition
but will also address the state of nutritional knowledge in Edinburgh where there was
a growing interest in the chemistry of nutrition but little organized exploration of the
effects of poor diet on the health of the people. As will be shown later, Edinburgh
physicians did give dietary guidance to institutions in the city but on the whole this
kind of advice was intended for the individual. Nutritional advice was not yet seen

12 T.M.Devine, The Great Highland Famine: Hunger, Emigration and the Scottish Highlands in the
13 D.J.Oddy, 'The paradox of diet and health: England and Scotland in the nineteenth and twentieth
centuries' in A.Fenton (ed.), Order and Disorder: The Health Implications of Eating and Drinking in
the Nineteenth and Twentieth Centuries, (East Linton, 2000), p.45.
14 E.Mccollum, A History of Nutrition, (Boston, 1957), p.92; A.Castiglioni, A History of Medicine,
& 36 which describe a diet prepared by Edinburgh physicians for the Charity Workhouse; A.Combe,
as a key aspect of public health although in the 1840s there were physicians who believed that lack of food was related to disease. On the other hand, one authority has suggested that in the nineteenth century attitudes to food were concentrated more on food adulteration than nutritional adequacy. Food purity was increasingly a common worry but there was also a growing awareness of the science of nutrition; the advances in chemistry that enabled food to be analysed and so prevent food adulteration were also used to investigate the chemical nature of food. This new interest in nutritional chemistry arose from research being carried out both in mainland Europe and Britain, but it was only in the 1840s that scientists began to relate the chemistry of food to physiology and nutritional requirements. A.P.den Hartog argues that there were two main approaches to the science of nutrition: there was research based on physiology and chemistry and there was research that was centred on epidemiology and its effect on the public health. It has been stressed that 'the chemistry of foods had been under investigation for many years, but the investigation of physiological chemistry, or chemical physiology, became a reasonably systematic scientific endeavour only around 1840, and its focus at that time was on nutrition.' The groundwork and stimulus for this work was laid in the 1820s and 1830s by two men, an Englishman and an American. One of these was William Prout (1785–1850), a London physician and scientist whose work changed the way people thought about food. Prout was prominent also during the 1830s in the debate on the relationship between science and religion as one of the eight scholars who wrote the series of tracts, known as the Bridgewater Treatises, to which the Rev Dr Thomas Chalmers was also a contributor. William Whewell (1794–1866), scientist and theologian, author of the first treatise to be published (1833) explained the purpose behind the tracts as an effort 'to lead the friends of religion to look with

**The Physiology of Digestion and the Principle of Dietetics**, (Edinburgh, 1836), a treatise intended for the medical practitioner and the interested layman.


confidence and pleasure on the progress of physical sciences, by showing how admirably every advance in our knowledge of the universe harmonizes with the belief of a most wise and good God." Prout was therefore recognised as an outstanding scientist and as a man who could rationalise the often-conflicting claims of science and religion. However, it was his work on animal chemistry that was of the greatest importance, although the concept of the 'vital principle' did detract somewhat from the rigour of his research. Porter described him in these terms:

Like most of his contemporaries, Prout was a vitalist who believed the chemistry of living beings was altogether different from that of unorganized materials, such differences being explained by a principle of organization. Imbued by the Creator with a faculty little short of Intelligence, this vital principle synthesized organic substances from inorganic elements into combinations unlike those in the mineral kingdom. Living systems could be sustained only by the constant and unremitting agency of the vital principle; otherwise death resulted, releasing the atoms into their original inorganic state.

In a lecture on food to the Royal Society of London in 1827 Prout first explained his view that the objective of food analysis was 'to determine the saccharine, oily and albuminous divisions in which the alimentary substances of the higher animals may be comprehended...'. He suggested that these three constituents of foods, now called proteins, carbohydrates and fats, were essential for human health, a statement thought to be one of the earliest attempts to describe an adequate diet in chemical terms. Prout, in his classification, used the term saccharine or saccharinous substances for carbohydrates, oleaginous materials for fats and albuminous or nitrogenous matter, proteins. His work was of the utmost importance for until then no understanding of the chemical nature of food existed, it had been believed from the earliest times that food contained only a single nutritional principle—aliment. This was the first time that food had been classified and its three main constituents, fats, carbohydrates and proteins described. Prout also established that the stomach contained hydrochloric acid, which acted in some fashion to dissolve these compounds but the exact mechanism remained unknown until the publication of an extraordinary piece of research in 1833.

The process of digestion was not understood until William Beaumont (1785–1853), a surgeon in the United States army, published the results of his work. In 1822 Beaumont was asked to treat Alexis St Martin, a young trapper, who had been shot in the abdomen. Remarkably, considering the nature of the injury, the youth survived but with an open track from the exterior of the abdominal wall to the interior of the stomach, allowing observation and analysis of the digestive processes. Over several years, Beaumont studied the response of the stomach lining to a variety of chemical and physical stimuli, publishing his findings in 1833. 22 His Experiments and Observations on the Gastric Juice and the Physiology of Digestion (1833), a landmark in medical literature, confirmed the presence of hydrochloric acid in the stomach, as Prout had claimed, and identified a ferment, which later analysis identified as pepsin, an enzyme responsible for the breakdown of protein.23 The importance of Prout’s classification and Beaumont’s observations on digestion in stimulating interest cannot be overestimated. A new era of chemical research began and was developed mainly in Europe.

There were several scientists in the Netherlands and Germany whose main research interests lay in the field of physiological chemistry and who were actively investigating the composition of nutrients and how the body utilised food. One of the most prominent of these was Gerrit Jan Mulder (1803–80), a Dutch physician and professor of chemistry in Utrecht, who published what has been described as the first scientific study on nutrition with a strong public health component. He did not confine his observations to purely scientific matters and rejected middle class prejudices on the imagined laziness of the labouring class, attributing their lack of vigour to faulty diet. In 1838 he coined from the Greek the word protein, which replaced the terms albuminous or nitrogenous matter and remains in use. Carl Schmidt in 1844 made up the word carbohydrate to replace the term, saccharinous

22 W. Beaumont, Experiments and Observations on Gastric Juice and the Physiology of Digestion, (Plattsburg, 1833).
23 Castiglioni, A History of Medicine, p.686.
substances, and this remains in use also. Jacob Moleschott (1822–1893), a Dutch physician and pupil of Mulder, wrote several popular scientific works, the first on nutrition entitled *The Science of Foodstuffs, for the People*, (1850). His writings reveal his philosophy of the science and culture of nutrition; he wrote of ‘the importance of food and diet for the physical, intellectual and social well-being of mankind.’ For Moleschott, nutrition was not simply a science, he related his scientific discoveries to philosophy and to liberal political ideas and he believed that healthy eating was essential for the development of ‘human well being, dignity and freedom.’ These two Dutch scientists had only one equal in the field of physiological chemistry, the German scientist, Justus von Liebig who, as suggested earlier, has a genuine claim to have been the most influential of all in extending the boundaries of nutritional science.

Justus von Liebig (1803–1873), professor first at Giessen and later at Munich, began to study the chemical processes of living organisms at the University of Giessen during the 1830s; the publication of his work *Animal Chemistry* in 1842 is considered to be the beginning of modern nutritional science, establishing him as a major figure in this field. One of the British Association’s most far reaching actions was their request that Liebig should investigate agricultural chemistry. His *Chemistry and its Applications to Agricultural Physiology* (1846) laid the foundations for the sciences of soil chemistry and nutrition. He is credited with having established the first practical chemistry teaching laboratory where he developed methods of organic chemical analysis and in his hands ‘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

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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.²⁸

His laboratory attracted students from many countries including Scotland and among
the Scotsmen who studied with him were, Thomas Thomson (1773–1852) and his
nephew, Robert Dundas Thomson, (1810–1864), both of whom taught chemistry in
Glasgow, Thomas Graham, Lyon Playfair and James Johnston, all members of this
‘clan of Scottish chemists’, who made sure that Liebig’s methods were known to a
wider public. Liebig’s seminal work, Organic Chemistry in its Applications to
Agriculture and Physiology, translated and edited by Playfair, was published in
September 1840 to coincide with the Glasgow meeting of the British Association.
The main thrust of his 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 protein had long term, and not
entirely beneficial, effects on nutritional thought and practice.²⁹

The work of Liebig, Mulder and Moleschott was crucial to the development of the
basic sciences of chemistry and physiology and in Britain the influence of the work
of Jonathan Pereira (1804–1853), a London physician who was among the first to
study and write on the subject of nutritional diets, was substantial. Pereira wrote of
Prout’s ‘beautifully simple and generally admitted classification into aqueous,
saccharine, albuminous and oleaginous’, but criticised its limitations since there was
no mention of salt, which Pereira considered essential, or of lemon juice, ‘one of our
most valuable anti scurbutic foods’.³⁰ McCollum suggests that ‘at the time of
publication of his treatise, no other writer had so carefully examined and weighed all
the experimental evidence available concerning the essential constituents of an
adequate diet.’³¹ Pereira’s book is certainly extremely detailed; his analysis of the

³¹ McCollum, A History of Nutrition, p.95.
classes of alimentary principles included aqueous, mucilaginous, saccharine, amylaceous, ligneous, pectinaceous, acidulous, alcoholic, oily, proteinaceous, gelatinous and saline and a chapter on dietaries had eight sections with recommendations for feeding children, sailors, soldiers, paupers, prisoners, the sick, the insane and puerperal women. 32

Much of the interest in the new science was at the outset limited to the notion of food as fuel but in the politically turbulent 1840s the adequacy of the food supply became an issue of great importance, especially in 1848, a time of revolutionary fervour in Europe. The role of science and nutrition in promoting and achieving social change, as advocated by men like Moleschott, was acknowledged by some but as yet the relationship between food and physical health, although accepted as common sense, was studied scientifically by few, Pereira being a notable exception. The extent to which in Edinburgh this relationship was looked upon as a matter worthy of investigation and discussion will form the subject of the next section.

Nutritional Science in Edinburgh

It is against this background of research both at home and abroad that nutritional knowledge and new theories on diet and health, in Scotland, and more particularly, Edinburgh has to be viewed. There is no evidence that scientists in Edinburgh considered that their research had any part to play in ensuring adequate food supplies; nutritional chemistry was mainly a scientific discipline, with few if any political overtones and, perhaps influenced by Liebig rather than Moleschott, it never became a subject with far reaching implications for society. If there was a political or economic dimension, this was manifested among political economists as a Malthusian anxiety about the nation's ability to feed a growing population as much as a concern for human dignity and freedom. It is impossible to say whether the men with medical degrees who had studied chemistry and nutrition in Germany and who taught and carried out research on returning to Scotland had any related political views; if they had they did not express them publicly. There were men in Edinburgh

32 Pereira, A Treatise on Food and Diet, pp.vii–xvi.
who did have opinions on the plight of the destitute and who expressed them forcibly, William Pulteney Alison being the most notable, but the majority of scientists and physicians avoided controversy.

The men with a particular interest in and knowledge of nutrition were William Gregory and Robert Christison who during the 1840s held the chairs of chemistry and materia medica respectively. In the Edinburgh school of medicine, a course of lectures on dietetics was given by Robert Christison (1797–1882), later Sir Robert, who was professor of materia medica from 1832 to 1877. He graduated MD, Edinburgh, in 1819, studying thereafter in London and Paris where he developed an interest in chemistry and toxicology. On his return to Edinburgh in 1822 he was immediately appointed professor of medical jurisprudence, in time gaining a distinguished reputation as a forensic scientist.33 His university course of lectures on dietetics and his detailed records of cases of scurvy treated during the late 1840s are a valuable source of material on nutrition and medical knowledge at this time.34

In his lectures on “Dietetics” Christison discussed food and nutrition in very general terms, for example, he compared the nutritive qualities of oatmeal and wheat, he criticised what he saw as the neglect of the potato in Germany and France and, describing the nutritive qualities of milk, he commented: ‘in Scotland with little else than oatmeal, it forms the food of hard–working and thriving labourers.’ Christison’s analysis of the cases of scurvy, which appeared in the city in 1847, will be discussed in a later chapter. Christison was interested in chemical physiology but he was not an original thinker or researcher in the rapidly developing field of organic chemistry. William Gregory (1803–1858), the fourth son of James Gregory (1753–1821), professor of medicine at Edinburgh from 1790 until his death in 1821, graduated at Edinburgh in 1828. He succeeded Charles Hope as professor of chemistry at Edinburgh in 1844 and was one of Liebig’s ‘earliest British disciples’ and, like his successor, Playfair, was a translator of his work. His successor, Lyon Playfair, later Lord Playfair (1818–1898), had studied with Thomas Graham (1805–1869) a

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33 J.D.Comrie, History of Scottish Medicine, (London, 1932), volume 2, pp.616 & 617.
34 Edinburgh University Special Collections, Christison Papers.
Glasgow graduate and professor of chemistry at University College, London and also with Liebig in Giessen.\textsuperscript{35}

These Scotsmen were all members of the chemistry section of the British Association, the most influential scientific body of the time. It is unlikely therefore that Christison was unaware of the new developments in nutrition and it is against this background that his ideas on diet should be judged. In his lectures on diet (c.1847) at the university many of his ideas and methods appear very similar to those of Jonathan Pereira, particularly the section devoted to the subject of dietaries. These dietaries and their nutritional content will be examined in a later chapter.\textsuperscript{36}

My intention in this review of historical developments in nutritional chemistry has been to demonstrate that Edinburgh scientists were involved in this expanding discipline; Liebig's books had been translated into English by Gregory and Playfair, men with influence and connections in Edinburgh academic circles. It is likely that the bias in Liebig's work towards the importance of animal nutriment, particularly protein, persuaded many experts in Scotland, as elsewhere, that animal food was superior to all other foods. Liebig had become convinced, without confirmatory evidence, that extract of muscle, especially beef, possessed special nutritional qualities and he was so certain of this belief that he popularised his eponymous beef extract, recommending it for patients suffering from weakness or those who were convalescent. The preparation was extremely popular for many years, largely because of his fame and reputation, 'until it was proved that the only value was in its flavour.'\textsuperscript{37} The popularity of 'Liebig's extract of meat' is an example of a product of no nutritional value gaining an undeserved reputation and as a result of Liebig's advocacy many believed that animal protein was essential and by far the most valuable item in any diet. This was certainly the case with Andrew Combe, an Edinburgh physician and author of a text on digestion and dietetics, in which he wrote, 'among the poorer classes ...diet being chiefly of a vegetable nature and

\textsuperscript{35} The biographical details are from the Dictionary of National Biography, Comrie, History of Scottish Medicine, volume 2, pp.472 & 623–625 and Morell and Thackray, Gentlemen of Science, pp.489–491.  
\textsuperscript{36} Edinburgh University Special Collections, Christison Papers, Tables of Nutriment in Various Dietaries, (Edinburgh, 1854).  
\textsuperscript{37} McCollum, A History of Nutrition, p.94.
consisting of porridge, potatoes and soups with very little butcher meat, proves far from adequate to carry on vigorous growth in the one [the child] or repair waste in the other [the parent].

The defects in contemporary nutritional knowledge are clear from Christison’s lectures on dietetics; in a lecture entitled on ‘The Proper Food for Man in Various Circumstances of Life’ gleaned from ‘the best established modern rules in dietaries’ he recommends a diet for the person carrying out heavy physical work. This he based on the training diet of prize fighters as communicated to Sir John Sinclair of Lybster and published by him in his work on longevity. The main object, Christison insisted, 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 he allowed (in an addition dated 1850) that they could be eaten ‘in small proportion, especially potatoes.’ Fish was a watery food and was not considered very nutritive. Perhaps the most harmful advice was his claim 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.

In a reference to relapsing fever, however, he thought that unemployment among the working classes ‘occasioning’ a lack of food was the prime cause of the disease. Christison was later appointed by the Board of Supervision for the Relief of the Poor in Scotland to advise the managers of poorhouses on diets for the inmates.

This review of mid nineteenth century nutritional theory does not prove or disprove whether doctors advising the various charitable institutions and dispensaries in Edinburgh had any great interest in or deep knowledge of new developments in chemistry, physiology and nutrition. There were physicians like William Alison, Robert Christison and Andrew Combe who did have some special knowledge and

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38 Combe, The Physiology of Digestion and the Principles of Dietetics, p.249.
39 Edinburgh University Special Collections, Christison Papers, Faulty Food and its Effects.
40 The Board of Supervision was created under the terms of the Act for the Amendment and better Administration of the Laws relating to the Relief of the Poor in Scotland, 8 and 9 Vict. cap 83, 1845; Levitt, (ed.), Government and Social Conditions in Scotland 1845–1919, p.35.
expertise but they were the exception rather than the rule and, as has been shown, their advice was not necessarily correct. Later in the century it became widely accepted that health depended on adequate energy intake and that could be supplied by any of the three main groups, carbohydrate, protein or fat with the proviso that some protein was essential. It was only in the twentieth century that the need for hitherto unsuspected components, vitamins, minerals and trace elements, was recognised. The likelihood is that Edinburgh doctors at this time were no better and no worse than doctors in the rest of Britain, possibly with the exception of Jonathan Pereira, as far as their knowledge of contemporary nutritional theory and practice was concerned. The scientific developments in chemistry and physiology that helped to shape their beliefs have been reviewed and the aim of the next section, therefore, will be to review current nutritional science so as to place in context nineteenth theory and practice.

Current Nutritional Theory

The main purpose of this section will be to describe current nutritional knowledge and to review the dietary recommendations of present day authorities. The concepts of nutritional essentiality and nutritional status will be defined and reference will be made to the nutritional status of historical populations. Finally theories of the interaction between malnutrition and disease will be examined with particular emphasis on infectious disease. These reviews will allow comparisons to be made between current and mid nineteenth century dietary practice and standards.

Before reviewing current theory and practice the terms nutrition and malnutrition will be defined. Nutrition is the way the body uses food, the process by which nutrients that are essential for growth and health are absorbed and provide the energy that the body needs to maintain the metabolism, the circulation, respiration, digestion, and cerebral function. Exercise in whatever form uses energy, also provided by food, but even at rest the body uses energy in the physiological processes referred to above. Malnutrition, strictly defined, means bad nutrition, but has come to mean undernutrition, a change from a qualitative concept to a
quantitative one and will be used in the latter sense in this thesis. The first and most important questions to consider are how the body converts energy from food and how that energy is used.

Plants and animals cannot create or destroy energy but they are able to change it from one form to another. Plants unlike animals can make use of solar energy directly by the process of photosynthesis, whereby solar energy is converted into chemical energy, which is then stored by the plant. Animals cannot use solar energy directly but they are able to use the chemical energy, bound in molecules of carbohydrate, protein and fat, present in plants or animals. By the process known as oxidation, carbohydrate, protein, fat and alcohol are converted into energy, carbon dioxide and water. The energy taken in as food is used to maintain body functions, to perform mechanical work, to sustain the tissues of the body and to promote growth. However, the conversion of this chemical energy into mechanical energy is an inefficient process with most of the energy lost as heat and only about 25 per cent converted into mechanical work. A moderately active person will use about half of this available 25 per cent in maintaining the body, this being the basic metabolic rate, the remainder being used by the muscles in doing work.

It is essential to be able to measure energy intake and output in order to recommend suitable levels of nutrition for various ages, physical states and degrees of physical activity. Accordingly two units of energy measurement have come to be used, joules and calories, one calorie being roughly equivalent to four joules but in practice nutritionists use larger units such as the kilocalorie which is equal to 1,000 calories. Using this unit it has been calculated that the average daily energy intake in the United Kingdom is 2450 kilocalories for men and 1680 kilocalories for women but, of course, each person's energy requirement varies according to their level of activity and to their basal metabolic rate. Even at complete rest energy is used up by the activity of internal organs, the basal metabolism; the basal metabolic rate is therefore defined as 'the rate at which a person uses energy to maintain the basic functions of

proportionately higher the body.' In order to maintain the basal metabolism, the average adult will use about 1 kilocalorie every minute, approximately 60 per cent of the total daily energy expenditure but the rate does vary from person to person with the young having a proportionately higher basal metabolic rate and the old a lower rate.  

Energy balance is achieved when energy intake as food equals energy expenditure. As has been emphasised, slightly more than half of energy output is expended in maintaining the body and the rest is utilised by the muscles in work, which may be external work or work in supporting body posture or in moving the body. The energy expended in carrying out external work is of course extremely variable as a person in a sedentary occupation expends much less energy than one carrying out heavy physical labour. Theoretically energy intake should balance energy output in order to maintain the body in a healthy state and is measured in calories, by a formula devised by W.O.Atwater (1844–1907) following dietary studies carried out in the United States around 1900. Atwater measured the heat produced by foodstuffs burned in a sealed container and in so doing obtained the familiar calorie values of food: four calories for each gram of protein burned, the same for each gram of carbohydrate and nine calories for each gram of fat.

Many different formulae have been created to calculate the ideal food intake, usually by recommending diets which contain a certain number of calories, the number varying according to individual circumstances, for example, 2,500 calories for an average sized male carrying out light physical work. One of the more accurate and more complex is the Harris–Benedict formula which first estimates resting energy expenditure having taken into account body weight, height, age and gender and bases its recommendations accordingly. A simpler rule of thumb formula is often used in which body weight in kilograms is multiplied by twenty five to give a figure for resting energy expenditure in calories. To this figure a further 10 per cent is added for the energy involved in digestion and an exercise factor which is variable, ranging from 10 per cent for minimal activity to 40 per cent for the highly active. Using this

44 Lowenberg, Food and Man, p.187; McCollum, A History of Nutrition, pp.189 & 190; Davidson, Passmore, Brock and Truswell, Human Nutrition and Dietetics, p.18.
equation the suggested calorie intake for a man weighing 80 kilograms, doing heavy physical work, would be 3000 calories daily. Martin Eastwood, an Edinburgh physician, in a recent work suggests that 5 to 7.4 kilocalories per minute is required for someone carrying out moderate work, for example, general labouring with a pick and shovel or heavy agricultural work; the figure he recommends for extremely intensive labour, for example lumber work or furnace stoking, is 10 to 12.5 kilocalories per minute. In the case of the former there would be a daily calorie intake of 3000 calories but for the latter the figure would be nearer 4000 calories per day.

There are occasions when the energy intake has to increase because energy output is greater than normal; the food intake of children has to be sufficient to allow for growth; in pregnancy extra energy is needed to enable foetal growth to take place and to provide for the placenta; during lactation extra energy is required for the production of milk. In a healthy human adult, the energy intake should, in theory, equal output and failure to achieve this balance will result in wasting of the body, if intake is less than output; if output is less than intake, fat will accumulate with inevitable gain in weight. However, nutrition is not simply a matter of balancing energy intake and output, there are other equally important factors to be considered and these will be examined next.

The nutrients taken in as food include not only carbohydrates, proteins and fats, whose importance was recognised as early as the 1820s, but also minerals and vitamins. Each of these groups has specific functions in the body and no single food contains all the nutrients essential for growth and health. All humans require the same nutrients throughout life but in varying amounts that are influenced by age, sex, body size, activity, state of health and heredity. Carbohydrate, protein and fat are

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46 Eastwood, Principles of Human Nutrition, p.90.
48 Lowenberg, Food and Man, p.165.
the major energy containing constituents of food and are referred to as the macronutrients and these substances will be studied next.\textsuperscript{49}

The Macronutrients

The most common of the macronutrients is carbohydrate and this will be analysed first. Carbohydrates provide most of the energy in almost all diets and frequently provide as much as 90 per cent and more of the energy needs of the poor in the form of cereals, rice and other plants. These provide a source of carbohydrate that is cheap, relatively easy to produce and widely available and it was the successful cultivation of cereals that allowed man to store food from which to obtain energy easily and rapidly.\textsuperscript{50} Most dietary carbohydrate in agricultural societies comes from cereals whereas in industrial societies carbohydrates from sugar provides an increasing amount of energy. This process had already begun in the first half of the nineteenth century — as early as 1838 sugar had become worthy of comment, Thomson writing that ‘sugar has now become an essential part of the food of the Europeans. It contains perhaps a greater proportion of nourishment than any other vegetable substance in the same bulk.’\textsuperscript{51} Although carbohydrate is of fundamental importance as an energy source and is always a major component of the diet it does not appear to be a dietary essential. In other words, man can survive in reasonably good health on a diet of meat alone, as was shown by the experience of the Arctic explorer, Stefansson who with a colleague was forced to live for a year on meat alone and survived in good health.\textsuperscript{52}

The second of the macronutrients is protein which contributes less than 20 per cent to the energy value of the diet and commonly forms between 10 and 20 per cent of the


\textsuperscript{50} Troyer and Fernandes, ‘Caloric Intake: Sources, Deficiencies, and Excess — An Overview’, pp.35 & 36.


total. As an energy source, protein is of secondary importance but it is essential for the formation and maintenance of the protein of the body. Every cell in the body contains protein, which is broken down regularly and has to be replaced by new protein from food. Carbohydrate can be metabolised by the body to create fat, and carbohydrate can be produced from protein but protein can come only from the protein in food. The tissues of the body are constantly undergoing repair and renewal, a process involving protein replacement at a rate greater than the normal dietary intake with the shortfall being made up by the reutilisation of old protein. Proteins in food are broken down into their constituent amino acids by the processes of digestion; it is these amino acids that are required by the young for growth and are essential for tissue restoration during the continual process of breakdown and renewal. Plants can synthesise amino acids but animals cannot and are therefore dependent on external sources for the nine essential amino acids that are said to be 'nutritionally relevant'.53 Low protein levels in cells and organs are the result of low protein or amino acid intake and affect function with the result that in the young, growth falters and may cease; in all individuals, increased morbidity and eventually death occurs if the dietary deficiency is not corrected. In the course of the body's normal physiological functions, protein and amino acids are turned over and some are lost by excretion; to maintain positive protein and amino acid status these losses have to be balanced by dietary intake. There is also an important qualitative element to protein metabolism in that nine essential or indispensable amino acids must be provided by food to ensure satisfactory nutritional status. This qualitative element has been defined thus:

The qualitative requirement for protein is defined as the lowest level of intake that will balance the losses from the body in persons maintaining energy balance and a modest level of physical activity. In the case of children and pregnant and lactating women the requirement also includes the needs associated with the deposition of protein in tissues or the secretion of milk at rates consistent with good health.54

The foods that provide protein are meat, fish and pulses with cereals as average providers and fruit and vegetables low. The complexity of protein metabolism is

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54 Young, 'Protein and Amino Acids', p.54.
reflected in the many calculations, equations and figures that have been used to recommend protein requirements. The daily protein intake recommended by the World Health Organisation is calculated according to age and physiological status, thus 1.47 grams per kilogram body weight for a six month old infant, 0.75 grams per kilogram body weight for a full grown adult, during pregnancy and lactation as much as 16 grams extra protein daily is recommended.\textsuperscript{55} The British standard recommendation is 69 grams of protein daily compared to that of the United States, which advises 56 grams daily, levels much lower than those advocated earlier in the twentieth century, when protein was still thought of much as it had been during the nineteenth century, influenced by the work of Liebig. Another authority advocates as a safe target for daily protein intake one in which 10 per cent of the body’s energy intake comes from protein.\textsuperscript{56} Livi–Bacci argues that the wide variations in present day advice as to safe daily protein intake are the result of ‘an incomplete understanding of the precise relationship between nutrition, health and sickness.’\textsuperscript{57}

Protein is nutritionally indispensable, providing the nine essential amino acids that the body cannot synthesise. If protein intake is low a deficiency of protein in the cells results and function deteriorates. In the young growth is affected and in the adult, unless the protein deficit is corrected, increasing debility occurs and death is the outcome. The amount of protein needed varies according to age and state of health. These recommendations apply to healthy individuals only, the protein needs of the sick being greatly increased.\textsuperscript{58} Many diseases increase the metabolic rate, infection being a typical and dramatic example in which fever is a sign of the increased metabolic turnover; as the metabolic rate rises body protein is used as fuel and at the same time there is both an increased utilisation of protein and a decrease in the appetite for food. When this happens to someone whose nutritional status is already compromised the effect can be catastrophic.\textsuperscript{59}


\textsuperscript{56} Davidson et al, \textit{Human Nutrition and Dietetics}, p.70.

\textsuperscript{57} Livi–Bacci, \textit{Population and Nutrition}, p.29.


The third of the group of macronutrients is fat which has a high energy value yielding twice as many calories per unit of weight as carbohydrate or protein; it is a concentrated source of energy and, moreover, enjoyable because fat gives flavour to food and increases its palatability. Fat includes not only the distinct, visible fats such as butter, margarine, lard and vegetable oils, but also less obvious fats found in some foods as an emulsion or in the tissues, meat being an example. Dietary fat varies from 35 to 45 per cent of the calorie intake in more prosperous countries to 15 per cent or less in economically poorer countries. It is now thought that the minimal requirement for fat may be very low and it has been suggested that 'in any country social requirements of fat for good living are far higher than physiological requirements for good health.' 60 In the thirty years since that assessment was made, research has shown that the quantity of fat is not the sole criterion, certain fatty acids are beneficial to health and play a significant role in the prevention of cancer and cardiovascular disease.61

The Micronutrients

The three macronutrients, carbohydrate, protein and fat are not the only important elements in the diet, trace elements, minerals and vitamins are needed for vital cellular function. These three make up the group known as the micronutrients, named because the amount required by the body to maintain health is much less than that of carbohydrate, protein and fat. The quantity of trace elements necessary is extremely small whereas certain minerals such as calcium, phosphorus, magnesium and potassium are needed in considerably larger amounts. There are fifteen trace elements considered to be essential for health: iron, zinc, copper, selenium, chromium, iodine, fluoride, manganese, molybdenum, boron, nickel, silicon, vanadium, arsenic and cobalt. Many have multiple roles but several have specific functions; for example, iron is needed in the synthesis of haem, an essential component of the blood, facilitating oxygen transport. Bread, fruit and vegetables are good sources of the mineral but the iron contained in meat is a major source being

60 Davidson et al, Human Nutrition and Diabetics, p.88.
absorbed more readily because much of it is in already in the form of haem. Iodine is essential for normal thyroid function and is well absorbed from food, the only rich source being seafood; fruits, vegetables, cereals and meat do contain iodine in small amounts but the quantity is variable, being determined by the environment in which the substance is grown. Iron, zinc, copper and selenium have an important effect on the function of the body’s immune system and therefore on the response to infectious disease.

The final group of dietary constituents to be considered is a group of chemicals which have become known as vitamins, defined “as organic substances present in minute amounts in natural foodstuffs that are essential to normal metabolism, the lack of which causes deficiency diseases.” 62 This definition, although accurate and useful, could apply equally to several nutrients, for example, amino acids and fatty acids. It is the lack of any similarity in the chemical structure of vitamins and their varied functions which makes classification difficult and this definition is a useful way of describing these important substances. Another way of defining them is as ‘a diverse array of molecules, which have equally diverse functional roles.’ 63 The existence of such dietary factors had long been suspected but evidence was lacking until F.G.Hopkins (1861–1947), an English scientist, in 1906 showed that young animals failed to grow when fed on purified nutrients. He wrote “there are many minor factors in all diets, of which the body takes account. In diseases such as rickets, and particularly in scurvy, we have had for long years knowledge of a dietetic factor; but though we know how to benefit these conditions empirically, the real errors in the diet are to this day quite obscure.” 64 There are two groups of vitamins, separated according to their solubility in fat or in water, the type of solubility affecting the relative degrees of absorption and transport within the body and the sites at which absorption takes place. In the fat-soluble group are vitamins A, D E and K; in the water-soluble group are vitamin C (ascorbic acid), vitamin B2

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63 Rucker, ‘Vitamins’, p.95.
(riboflavin and thiamin), vitamin B5 (niacin), vitamin B6 (pyridoxine), vitamin B12 (various cobalamines), folate, biotin and pantothenic acid. It is not proposed to describe in detail the history of the identification, the function and the availability in nature of all the vitamins but to concentrate on those which have been identified in dietary analyses and by case records as deficient in certain diets in Edinburgh during the 1840s. Vitamin D will be referred to in this exercise in view of its role in the immune response although it is not intended to investigate the incidence of its deficiency and that of rickets during this time.

If it is not easy to define vitamins, their nomenclature is equally confusing, a confusion that has arisen for historical reasons. When each was discovered it was given a letter and only with the identification of the structure of the compound was it given a chemical name. The discovery in 1913 by E.V. McCollum (1879–1967) and M. Davis (1888–1967) of two factors, essential for normal growth, which at first they called fat soluble A and water soluble B was the first step and the identification of C, D, E soon followed. Both the letter and chemical names continue to be used and are interchangeable. The term “vitamine” was coined in 1911 by C. Funk (1884–1967), a Polish biochemist, but was soon changed to vitamin and from about 1920 the alphabetical suffixes were added. Funk put forward his theory, later shown to be correct, that beriberi, scurvy, pellagra and possibly rickets were caused by a deficiency in the diet of “special substances which are of the nature of organic bases, which we will call vitamines.”

The first of this group to be described is Vitamin A which is not a single chemical compound but a group of fat–soluble compounds of which retinol or pre–formed vitamin A is one of the most active and is found in animal foods such as liver, milk and eggs; fish is a ready source and many plants also provide a precursor. Dark coloured pigments, provitamin A carotenoids, which the body converts to vitamin A, are present in carrots, spinach, kale, turnips, tomatoes, oranges and other fruits and vegetables to the extent that about 30 per cent of the body’s vitamin A comes from

66 Porter, The Greatest Benefit to Mankind, p.554; Castiglioni, A History of Medicine, pp.955–957.
this source. The vitamin is important in vision, bone growth, cell function and the maintenance of the surface lining (the mucosa) of the lungs, the urinary tract and the intestines. The integrity of these surface linings is a vital first line of defence against bacteria and viruses and its loss has serious consequences. It has an essential role also in the immune system and in the function of white blood cells when fighting infection. Deficiency is rare in the developed world, appearing only after a prolonged period without dairy produce or vegetables but in the developing world deficiency is not uncommon and is responsible for childhood xerophthalmia and blindness. The damaged surface lining of the lung allows micro-organisms to invade and since the immune response is also compromised what might have been a mild infection can become lethal. Deficiency has been associated with an increase in the severity of respiratory syncytial virus infection which causes lower respiratory infections and is responsible for worldwide epidemics every year. It is thought that damage to the lung and the immune system is the reason that measles becomes deadly in children deficient in vitamin A. It is now recommended that Vitamin A is given to patients with measles who are ill enough to require hospital admission, a measure that has reduced the mortality in such cases from 5 per cent to 1.6 per cent. In Asia infants with low vitamin A levels have an increased incidence of respiratory infections and diarrhoea and several studies have shown that routine periodic administration of vitamin A reduces mortality from these and a number of other causes in infants and children. The effect of possible vitamin A deficiency on infection in children and adults in Edinburgh will be discussed in a later chapter.

The D vitamins are a group of fat soluble chemicals, one of which can be synthesised in the skin from the action of ultra violet light. There are also small amounts in dairy produce but many people obtain their vitamin D from sunlight, the only rich sources

of vitamin D being the liver oils of fish, hence the provision of cod liver oil to children during World War II. Vitamin D is needed for the formation of normal bone and lack adversely affects bone growth causing rickets. The D vitamins also influence the development of blood cells that have a role in combating infection. The theory that a poor environment was the cause of rickets was disproved by E. Mellanby (1884–1955) in 1919 when he showed that certain dietary constituents, especially cod liver oil, had an anti–rachitic capability and that rickets was a deficiency disorder.71 The dietary factors were identified in 1926 and 1927 by A.F.Hess, O.Rosenheim, T.A.Webster and M.Weinstock after earlier work by P.G.Shipley, E.A.Park and E.V.McCollum had revealed the therapeutic value of sunlight in rickets.72

Vitamin E is the generic term given to a group of chemical compounds, tocopherols, ‘that exhibit functional activity in biological or chemical tests of antioxidation’.73 The discovery of this dietary factor in 1922 is credited to H.M.Evans (1905–83) and K.S.Bishop (1889–1976); Evans and his co–workers G.A.Emerson and H.P.Emerson isolated and identified the active principle in 1936.74 The most active form of the vitamin is found in plant oils, particularly in germ seeds, eggs, cereals and some green vegetables such as broccoli. Their role as anti–oxidants helps to protect against cardiovascular disease but signs of deficiency in the human are not obvious, although recent research suggests that vitamin E is important for the immune system.75

Among the water-soluble group, vitamin C (ascorbic acid) acts as an anti–oxidant and an anti–scorbutic, hence its importance in the human. Its distribution is limited, the richest sources being citrus fruits, currants, berries and green vegetables with root vegetables and potatoes containing smaller amounts. The potato is an important source in many European countries because of the quantity eaten daily and often forms a large part of the total intake. The best animal sources are liver and fish roe,

73 Rucker, 'Vitamins', p.82
evidently the reason why the Eskimos, until recent times living on a diet wanting fruit and vegetables, did not develop scurvy.\textsuperscript{76} Deficiency causes scurvy is a form of scurvy, which will be discussed in a later chapter, stressing the work of James Lind. In the nineteenth century A.Hirsch (1817–94) demonstrated the antiscorbutic properties of the potato and by the second decade of the twentieth century the antiscorbutic factor had been recognised as an essential element of the diet. A.Szent–Györgyi (1893–1986), W.A.Waugh and C.G.King (1896–1988) are credited with isolating the active principle and the Lancastrian, W.N.Haworth (1883–1950), established its chemical structure, later synthesizing vitamin C in the laboratory and naming the compound ascorbic acid because of its antiscorbutic properties. Haworth, King and Szent–Györgyi were awarded the Nobel Prize for their work on ascorbic acid.\textsuperscript{77}

The B vitamins, thiamin, riboflavin, niacin, pantothenic acid, biotin, folacin, Vitamin B6 and cobalamin are a diverse group of compounds with equally diverse functions and are widely distributed in plant and animal foods. There are several clinical syndromes associated with deficiency of the B group of vitamins. Beriberi results from thiamine deficiency, a syndrome associated with certain methods of cooking rice; in Europe, alcoholics are the only group in which thiamine deficiency is commonly seen but biochemical evidence of thiamine deficiency has been reported in the elderly living at home subsisting on an inadequate diet.\textsuperscript{78}

Vitamins therefore represent a broad range of chemical compounds with diverse functions in human metabolism. The absence or lack of these substances not only causes specific deficiency disorders such as scurvy but also results in less obvious but potentially serious malfunction of the immune response. Vitamin deficiencies most often occur in a monotonous diet based on a restricted food supply and it is on such deficiencies and curtailed food sources in Edinburgh in the 1840s that succeeding chapters will concentrate.

\textsuperscript{76} Davidson et al, \textit{Human Nutrition and Dietetics}, p.161.
This review of the macronutrients, carbohydrate, protein and fat, and the micronutrients, the minerals and the vitamins, all of which are essential to health, does not complete the analysis of nutritional needs. The difficulties encountered in the assessment of nutritional status and the concept of nutritional essentiality will now be considered.

**Nutritional Essentiality and the Interaction between Nutritional Status and Disease**

Calculations of daily food intake do not give any information as to nutritional status, a complex subject and one for which many techniques have been employed in its evaluation. For example, measurements of the body's tissue nutrient reserves can be considered as true indicators of nutritional status whereas 'dietary intake data reflect only the likelihood of low (or high) intake and the consequent risk of undernutrition (or overnutrition).' Dietary intake can appear adequate but nutritional status may in fact be poor if the person's energy needs exceed intake; such an imbalance can arise if there is high energy output at work or if extra energy is required to combat illness. Nutritional status is the outcome of nutrient intakes since conception, balanced against the demands on those nutrients for health, growth, work, play, warmth and happiness.

Nutritional science allows current researchers to study the nutritional status of historical populations whose theoretical average nutritional requirements can be estimated once the age and sex structure of the population has been taken into account. Such an exercise has been carried out by Livi–Bacci for 'a range of populations, imagined and real, historical and contemporary.' He found that the differences in theoretical requirements for three historical populations and for three theoretical populations studied were not marked. Using the United Kingdom

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78 Basu and Dickerson, *Vitamins in Human Health and Disease*, pp.21 & 22.
recommended standards for present day diets, the average daily calorie requirements for the three theoretical cases were 2,147 calories, 2,195 calories and 2,272 calories per day. The figures for the three historical examples, Tuscany in 1427, England in 1681, and England in 1821 were 2,153, 2,252, and 2,178 respectively. Livi–Bacci wrote ‘if we ignore variations in distribution, we can conclude that a population which could rely on a normal consumption of 2,000 calories per head would have been, in centuries past, an adequately fed population, at least from the point of view of energy.' As he infers, the fact that a population was adequately fed in terms of energy does not prove that it had a good nutritional status. It is relatively simple to calculate the calories available to population groups but assessment of the nutritional status of the population in question and the balance of essential constituents in their diet is much more difficult.

An average daily intake of 2000 calories supplies sufficient energy for all but the most active but without a breakdown of the constituents that furnish these calories no accurate assessment of the diet can be made. It is essential that a diet contains the elements that are fundamental to health, namely, protein, certain fats, vitamins, trace elements and minerals; if any of these is absent or deficient ‘pathological conditions may arise and worsen, no matter how high the calorie intake may be.’ Harper, discussing nutritional essentiality, suggests that there were some pre–nineteenth century descriptions of disease which hinted at this notion, one of them being Lind’s discovery that citrus fruits prevented scurvy. The concept of essential foods arose from observations that particular diseases could be prevented by the addition of certain substances to the diet, the basis of another early experiment on food essentiality, this time in France. Food scarcities in the early years of the nineteenth century was the motivation for the setting up in 1815 of the French Gelatin Commission to establish whether the gelatin extracted from bones could replace meat in the diet of the poor. The physiologist employed by the Commission, F. Magendie (1783–1855), found that dogs could not live on gelatin alone or on bread and gelatin

82 Livi–Bacci, *Population and Nutrition*, p.27.
but they survived when protein was added to their diet and from these observations he concluded that protein was an essential element. 83

The concept of nutritional essentially has been defined by Harper whose five criteria of essentiality are:

1) The substance is required in the diet for growth, health and survival.
2) Its absence from the diet or inadequate intake results in characteristic signs of a deficiency disease and, ultimately, death.
3) Growth failure and characteristic signs of deficiency are prevented only by the nutrient or a specific precursor of it, not by other substances.
4) Below some critical level of intake of the nutrient, growth response and severity of signs of deficiency are proportional to the amount consumed.
5) The substance is not synthesized in the body and is required for some critical function throughout life. 84

These five criteria define nutritional essentiality, the earliest evidence of which were Lind’s discovery that lemon juice cured scurvy and Magendie’s observation that protein was indispensable. By 1850, several mineral elements, calcium, potassium, sodium, iron and chloride had been recognised as essential, largely through the work of Liebig but not all of Liebig’s opinions were correct; he argued that carbohydrate, fat and protein together with a few minerals were sufficient for a nutritionally adequate diet but also that only nitrogenous foods had real nutritional value. Such was his eminence that few were prepared to disagree with the result that many of his principles, now known to be wrong, dominated nutritional thinking for much of the nineteenth century. It is now accepted that the essential nutrients include nine amino acids (derived from protein), vitamin A, the eight compounds in the B group of vitamins, vitamins C, D, E, K, certain fatty acids, minerals, trace minerals, electrolytes (sodium, potassium and chloride) and energy sources. 85

It has been said that the complexities of protein metabolism are such ‘that there is a great deal of uncertainty and imprecision attached to the amino acid requirement

values for all age groups.' However, there is no such uncertainty about the effects of a lack of protein on the health of the individual and, in particular, the child. Protein–energy malnutrition (PEM) is the term used for the clinical condition that results when dietary protein is insufficient for the body’s needs. Malnutrition and PEM are frequently used interchangeably, with good reason for the one is seldom found without the other. PEM is a misnomer since there is often a co-existing deficiency of other essential dietary constituents, such as vitamins, minerals and trace elements. The clinical features of PEM are complex and follow not only from a failure of protein synthesis with dysfunction of various organs but also from the biochemical effects of vitamin and mineral deficiencies. At one end of the spectrum is marasmus, a condition of partial starvation in children (the equivalent of starvation in adults), resulting from prolonged lack of dietary energy, protein and other essential nutrients. Wasting of the limbs from muscle atrophy and a shrivelled "old man" face are among the most obvious signs of the condition. The potential for a vicious circle of diminishing nutritional status and decreased resistance to infection is seen in protein-energy malnutrition. Loss of appetite and mobilisation of tissue nutrients to combat the infection reduce nutritional status still further and, in turn, damage to the immune response permits a higher level of infection than might otherwise have been the case. Thus a relatively mild infection like measles can become a dangerous illness with a high mortality rate in a malnourished child.

Nutritional status can be defined as the balance between food intake and energy output but there are other elements which have to be taken into account. An individual in less than perfect health may use energy to feed parasites and fight infection in addition to that expended in physical activity and in the maintenance of basic metabolism. Therefore, someone carrying out hard physical labour while combating parasitic and low grade or chronic bacterial infection on a diet rich in calories may actually have a poor nutritional status. A person free of disease and not carrying out physically demanding tasks, while existing on a meagre diet, may have good nutritional status. This downward spiral from the interaction of malnutrition

86 Young, ‘Protein and Amino Acids’ p.62.
and disease, when infection diminishes nutritional status, a state which in turn may increase susceptibility to further infection, has led to the concept of synergy, a condition that can exist not only between malnutrition and disease but also between disease and disease.\textsuperscript{98}

It was not until 1959 that the case for the interaction between nutrition and infection was argued scientifically by Scrimshaw and his colleagues. Their groundbreaking article was followed in 1968 by their World Health Organisation monograph, which confirmed their 1959 conclusions and brought their findings up to date.\textsuperscript{89} These and later studies showed that ‘the concept that malnutrition adversely affects immune responses’ was correct. The nature of the relationship is highly complex and is still not fully understood but there is general acceptance of the notion that malnutrition has an unfavourable influence on the body’s ability to combat infection. Poorly nourished individuals rapidly exhaust protein and calorie reserves in the process of fighting infection. This ‘synergistic package’, a vicious cycle of under nourishment and infection, is the cause of the high morbidity and mortality of populations in the developing world and, as Taylor suggests, the idea that ‘the behaviour of most diseases is shaped by the nutritional state of the affected host,’ is now widely accepted.\textsuperscript{90} In 1982 a multi-disciplinary conference of historians, demographers, nutritionists and economists concluded that synergy between malnutrition and disease did exist, that all infections adversely affected nutritional status and that malnutrition could reduce resistance to infection and thereby increase the severity of many infections. The conference grouped infections according to the degree of synergy thought to exist; smallpox, plague, typhoid, yellow fever and poliomyelitis.


were considered to be so virulent that nutritional status has little influence on outcome; with measles, diarrhoea, tuberculosis, cholera there was thought to be a high degree of synergy; typhus, diphtheria and influenza were considered to be variable or equivocal in their response to nutritional status. Over the following twenty years research into the body’s defence mechanisms against infection including the function of immune response has confirmed these conclusions and this will be discussed later.

Malnutrition is often equated with under nutrition, but its true meaning, as defined earlier, is "bad nutrition" especially in the context of ‘imbalanced nutrition.’ When considering protein and energy nutrition there is a range of clinical syndromes caused by both dietary imbalance and deficiency, for example, nutritional marasmus from pure protein lack, kwashiorkor which appears when protein and energy are both deficient, with protein lack the greater. These are gross manifestations of macronutrient lack but now deficiencies of micronutrients and the specific needs of the immune system are receiving attention. It is these less florid presentations that have been subjected to scrutiny in recent years as scientists investigate the relationship between nutrition and immunology. The authors of a recent text on nutrition and immunology wrote:

The last twenty years have seen an explosion in research, expanding on the molecular requirements of the immune system. From the role of minerals and cofactors in transcriptional regulation of immune maturation, through the specific protein, vitamin, and lipid needs of immune responses, to the increased demand for antioxidant protection and tissue repair created by the consequences of immune activation, these fields of the molecular nutrition of the immune system are the focus of multiple chapters in the text.

Early research into the relationship between malnutrition and immune function concentrated on antibody mediated immunity but now the focus is on other


parameters of the immune response. In evaluating the immune system there are at least ten components to consider, including the following: the physical barriers of the skin and mucous membranes; the white blood cells, which consume invading cells and are the second line of defence; lymphocytes, also part of the white blood cell system; B cells, part of the lymphocyte group, are essential in the formation of immunoglobulins, which in turn form antibodies; the complement system made up of proteins has a vital role in the elimination of invading microorganisms. Each of these varied and complex immune responses to infection is impaired by malnutrition, and more specifically by protein and vitamin deficiency.93

Vitamins and proteins are vital to a properly functioning immune system but mineral and trace elements, copper, selenium and zinc, are also important and their lack damages antibody production, phagocytosis, lymphocyte formation and the complement system. The balance of macronutrients and micronutrients is fundamental to the body’s ability to defend itself against invading microorganisms and it has to be remembered that the immune system, in common with all physiological processes, requires energy to function and to create certain structural mechanisms, all of which are derived from food. ‘Without adequate nutrition, the immune system is clearly deprived of components needed to generate an effective immune response.’ 94 Many of the interactions between specific nutrients and the immune system have still to be clarified but it is now accepted that there is a high correlation between immune status and nutritional status. In praising the pioneering work in nutritional immunology of Chandra, Beisel, Scrimshaw and Newberne, Fraker wrote ‘these early studies, along with more recent investigations to be discussed, provide compelling evidence that nutritional status and immune status are closely linked and that immune integrity can be rapidly altered by changes in nutritional status.’ 95

The relationship between malnutrition and infection has been examined in the light of current knowledge of immunological mechanisms, a review that is critical to understanding how poor nutrition during the 1840s might have altered individual resistance to infectious disease with increased morbidity and mortality. The link between nutritional status and immune status has long been accepted in a fairly uncritical way but there is now solid scientific evidence which confirms the relationship; those who are badly fed are more likely to develop infections and having done so are more likely to succumb. There is now a realisation that specific nutrients, particularly protein, vitamin A and Vitamin C, have a vital role in the immune response and it is on the amounts of these essential nutrients in working class diets during the 1840s that succeeding chapters will focus. There is little hard evidence available about the food eaten by the poor at this time but, if accurate records do not exist, there remains sufficient detail from which to build a picture and form conclusions about their diet, taking into account regional differences and contrasts in urban development which make estimating calorie and essential nutrient intake extremely problematic. The next chapter, therefore, will explore existing sources in a review of the diet of the Edinburgh working class and will appraise the methods that have been used to calculate the nutritional status of historical populations.
This chapter considers the nutrition and nutritional status of the people of Edinburgh and more especially, that of the poor and the destitute during the 1840s, analysing the sufficiency of food available in the city during a decade that has been named "the Hungry Forties" — with good reason according to one historian. ¹ The assessment of a population’s nutritional status is not simple and no technique is entirely satisfactory. One of the most accurate methods used to calculate nutritional status is the collection of dietary data over a period of time, employing both quantitative and qualitative techniques. This yields acceptable results but since there is no record of the daily amount and type of food eaten by the poor in Edinburgh in the 1840s this technique cannot be used. There are institutional diets which will be analysed in a later chapter producing information on the nutrition of some of the population at certain periods of their lives.² This analysis does not, of course, deal with the average daily food intake of the majority of the population, but from accounts written by doctors, clergymen and others an impression of the type of food eaten by the poor can be obtained.

The second technique that is used to calculate nutritional status is anthropometry in which measurement of the mass and dimensions of the body at a known age give information about nutritional status, both past and present. The method is based on the fact that the height of an individual is determined both by genetic inheritance and environment; thus, the growth of a child is the result of a number of factors that have been in action from conception, including levels of nutrition. The potential of anthropometry was recognised in the 1890s but it was not until World War I that the modern era of nutritional anthropometry began when comparison of the attained

² Edinburgh University Special Collections, Christison Papers, Tables of Nutriment of Various Dietaries, (1854).
growth of a group of known age to a set of known reference standards provided estimates of nutritional status. The research on which anthropometry is based showed that height and weight measurements reflect prior nutrition; the height an individual achieves is determined by past nutritional status and quality of food intake, particularly of protein. Weight can be lost but attained height, once achieved, cannot be diminished by 'environmental insult.' Thus an individual whose caloric intake equalled or exceeded output and whose diet included high quality protein, essential vitamins and minerals would be likely to attain his or her maximum height. Failure to achieve this occurs when caloric intake is exceeded by output, particularly if this happens at times of maximum growth or when the diet is deficient in the constituents necessary for growth. When anthropometry is employed as an indicator of past nutritional status, it is based on height measurement when growth is presumed to have ceased, but, as has been pointed out 'the limits of accuracy for such data, including the pitfalls inherent in recorded ages, have long been recognised...'. This is not the only parameter on which anthropometry has been criticised, as will be discussed next, but Floud and his colleagues in their work on British living standards at different periods in the past claim that height measurements do give valid results and discuss at some length the anthropometric studies carried out by J. Beddoe in 1870 and some years later by the British Association for the Advancement of Science. Anthropometry has other limitations in that it does not reflect recent changes in nutrition and it is not specific to particular nutritional deficiencies. It has been criticised also on the grounds that recorded age is not always accurate, that there are ethnic differences in growth patterns and that nutritional status determined by height for age can be different from that derived from weight for age. A recent study has emphasised the importance of excess caloric output through physical exertion producing a negative nutritional balance as the cause of a decline in

heights. An underfed youth carrying out hard physical work for extended periods on an average dietary intake may have a low nutritional status and as a result will not achieve his full growth potential. A further element that is open to criticism is the gender bias in most studies; Floud and his collaborators based their research on the growth of males, measuring the height of male military recruits in the nineteenth century; they admit that the absence of data for females is a shortcoming but is unavoidable. The gender imbalance may in fact disguise the extent of sub-nutrition because it has been suggested that at certain times the male’s nutritional status was superior to that of the female. Oddy has drawn attention to this quoting Edward Smith’s assertion that the working class believed that “the husband wins the bread, and must have the best food.” If this was so the nutritional status of women and children may well have been lower than that of the male.

The third technique used to calculate nutritional status is clinical appraisal and the recording of biochemical data to establish the concentration of certain nutrients in the body, ascertaining physical condition and the presence or absence of signs of illness. For a nineteenth century population biochemical investigation is not possible but surviving reports and records, especially those from physicians, provide details of diets and of particular illnesses. Moreover, records of morbidity and mortality should in theory provide evidence of nutritional deficiency disease but, as Petty and Rivers have pointed out, it is rare for this type of data to be of the quality and quantity necessary to make valid judgements. Estimation of the extent of malnutrition from such surveys is difficult, not simply because the data are of poor quality, but because frank clinical deficiency is comparatively rare.

Contemporary accounts of the consequences of food shortages on the poor are of value when considering the relationship between malnutrition and disease and it is

9 Brown, 'Application and Interpretation of Commonly Used Nutritional Assessment Techniques,' pp.3–12.
here that the experience and knowledge of physicians is valuable. The work of the
author of a monograph on the harm caused by malnutrition, the Manchester doctor,
Richard Baron Howard (1807–1848), is worth recalling. Howard, a medical graduate
of Edinburgh, explaining his special knowledge and expertise, wrote ‘the more
severe and aggravated effects of defective nutrition come under the notice of the
physician, and, in periods of distress, he is often called upon to apply the resources of
his art for the relief of diseases then arising.’ His experience was gained in the
slums of Manchester during the autumn and winter of 1837–38 when many of the
working class were out of work.11 Howard used statistics from the dispensary in
which he worked and official figures for mortality in the city in his review of the
effects of malnutrition on the poor showing that lack of food and increased mortality
were related. As will be discussed in a later chapter the absence of mortality statistics
in Edinburgh prevented and prevents any such analysis but there are contemporary
descriptions of food and food shortages which will be reviewed.

The nutrition of the labouring classes and the unemployed was ignored by most
contemporary observers who on the whole were more concerned with the sanitary
condition of the poor. One of the few who took a different view was William
Pulteney Alison (1790–1859) who graduated at Edinburgh in 1811, becoming
physician to the New Town Dispensary in 1815, professor of medical jurisprudence
in 1820 and professor of medicine in 1842. He made a special study of fevers in the
city and from this and his experience of treating the poor at the Dispensary became
convinced that destitution encouraged the development and spread of disease.12 In a
paper on the fever epidemic of 1843 Alison described the plight of the destitute of
Edinburgh, highlighting the suffering caused by lack of food and quoting an extract
from a letter written in April 1843 by Mr Johnston, chairman of the committee
established to relieve the destitute:

During the last five months, I have had the best means of ascertaining
the amount of poverty and destitution amongst the industrious poor of
this city; their privations from want of food, clothing, and household
furniture, greatly exceed the belief of those who have not the same

means of knowing them. I have known many instances of sober and industrious tradesmen who, after having sold or pawned every article within their dwellings (their fire-grate not excepted), were driven at last to apply to the committee for a scanty supply of bread or meal to appease the cravings of hunger, many of them urging the plea (which their emaciated appearance too truly confirmed), that for eighteen, twenty-four or twenty-eight hours they had not tasted food.\(^\text{13}\)

It is likely that the contemporary belief that Edinburgh was not industrial convinced many observers that the city did not have the problems common to the rapidly urbanising industrial cities like Glasgow and Manchester and that the scale of deprivation in Edinburgh was somehow manageable. This conviction may have grown because of the scarcity of reliable data about the nutrition of the people, their morbidity and mortality and the fact that: 'British historians have been confronted with an insuperable obstacle: they do not possess any trustworthy sources of information for the consumption of food.'\(^\text{14}\) A review of European food history published in 1992 contained the following passage:

> Early in the 1980s, the compiler of a bibliography on the history of food wrote: 'Comparatively few works of scholarly excellence have been written on the history of food. The researcher is obliged to extract bits and pieces here and there from secondary sources whose overall merit may be slight.' While some of us may shrink from accepting the full weight of that judgement, there is a ring of truth about it that we recognise: it remains a matter of surprise that the creation of a bibliography of historical studies of food supply, consumption and nutritional status of the British people is a relatively sparse affair and that the amount of current work in the field is limited.\(^\text{15}\)

In the bibliographical study cited, references to food and the nutritional status of the people of Scotland are few and there are none that deal with the diet of the citizens of Edinburgh. The explanation as to why social and economic historians of nineteenth century Scotland have on the whole ignored nutritional standards in their analyses of life in Scottish cities may lie in the fact that there has been an tendency among social commentators, perhaps dating from Edwin Chadwick's report of 1842, to attribute


the high rates of morbidity and mortality in British cities to bad housing, poor water supplies, non existent sewage disposal, overcrowding and bad ventilation.\textsuperscript{16} A quantitative analysis of the nutrition of Edinburgh’s citizens is hampered by a lack of data, but an appraisal of the food available will be carried out although such an exercise will not reveal the nutritional status of that part of the population most likely to have experienced periods of substandard nutrition. During periods of economic crisis, measures were taken to help the destitute and figures for the numbers relieved are available; the reports of various charitable organisations also record the number of paupers and people relieved. It is intended to use this information to demonstrate that there were many people unable to obtain sufficient food at certain periods during the 1840s. The effect on nutritional status of the potato blight of 1845–47 will be reviewed and the general standard of nutrition throughout the 1840s of groups most at risk will be examined.

Neither the \textit{Report from the Commissioners appointed for inquiring into the Administration and Practical Operation of the Poor Laws in Scotland} nor the new Statistical Account gives any information on the diet of the poor of the city.\textsuperscript{17} The latter includes accounts by parish ministers on the agriculture and horticulture in districts near Edinburgh and these will be used to build up a picture of the food available to the city. The Poor Law Report has references to diet but mainly to the quantities of food provided in various institutions such as the Charity Workhouse, the West Kirk Charity Workhouse and the House of Refuge.\textsuperscript{18} In their seminal work on \textit{The State of the Scottish Working Class in 1843} Levitt and Smout analysed the types of food common in various parishes based on the Poor Law report. Their findings will be reviewed and compared with other sources, both primary and secondary and conclusions made as to the diet eaten by the poor of Edinburgh.

\textsuperscript{17} P.P.\textit{Report of the Royal Commission appointed for inquiring into the Administration and Practical Operation of the Poor Laws in Scotland}, 1844, [557.] xx–xxvi; \textit{New Statistical Account of Scotland} (Edinburgh, 1845).
\textsuperscript{18} \textit{Report of the Royal Commission on the Poor Law (Scotland)}, 1844, xx, pp.1, 20, 26 & 105.
Kitchen and Passmore quote Sir John Sinclair who maintained in 1813 that Scottish agriculture could now be considered pre-eminent in support of their thesis that the diet of the Scottish peasant was improved by ‘the coming of scientific farming.’ The new methods ‘meant for the peasant the addition of potatoes and other vegetables, more cheese and more meat to his diet of oatmeal and milk.’ 19 It is doubtful if these improvements were sustained over the decades that followed, particularly in view of Mitchison’s claim that in the 1840s a farm labourer in a well-paid area could not adequately feed a wife and three children. She implied that the skilled urban workman was better off but the unskilled man was worse off and less secure than he had previously been. 20 What is certain is that in time of economic crisis and of poor harvests both the rural worker and his urban counterpart were hard pressed to feed their families and at such periods their nutritional status must have been compromised. It is easy to identify years during the 1840s when food was scarce but it is more difficult to identify the types and amounts of food eaten by the urban poor during this time. There are sources which refer to the diet of the average Scot during the mid nineteenth century and these will be used to give a broad picture of the diet of the people we are concerned with in this chapter.

One of the few scholarly works to attempt an analysis of Scottish food during the nineteenth century refers to the diets of the working class, as recorded by Hutchison in his survey of 1868 and concludes that they were generally of the following type:

- **Breakfast**  
  Porridge and Milk.

- **Dinner**  
  Tea, oatcakes, Fish, Cheese or Eggs.
  Broth with fresh meat or salt pork on Sundays.

- **Supper**  
  Porridge and Milk, or Potatoes and Fish
  Milk or Tea. 21

In their investigation of Scottish eating habits, using data from the Poor Law Commission, Levitt and Smout deduce that oatmeal and potatoes were the dominant items in the diet of the working class. This was based on reports from the 93 per cent

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of parishes that responded to the Commission’s questionnaire but the authors acknowledge that the absence of detailed answers from Edinburgh and Glasgow prevent a valid comparison being made between urban and rural working class diets. Nevertheless, they conclude that oatmeal and potatoes were important in both, inasmuch as reports from urban parishes in Aberdeen, Dundee, Greenock and Kilmarnock suggest that diet in the towns was very like that in the country, with oats and potatoes predominant. There is no mention of bread in the answers from towns and cities, surprising in view of statements made in other studies. The Poor Law Commission findings on which Levitt and Smout based their conclusions differ from those in the New Statistical Account in which ministers from parishes close to Edinburgh gave quite a different picture of the eating habits of the working class, indicating that bread was now common. There is no obvious explanation for this unless it is assumed that the eating habits of the population of the urban areas in the north and west of the country remained traditional and were markedly different from those of Edinburgh. This might be so bearing in mind the advanced agricultural economy of the Lothians.

The change had begun in the eighteenth century. In the old Statistical Account, the minister of Mid-Calder wrote ‘not many years ago, loaves made of flour were rarely to be found in the country, unless in gentlemen’s families; but now they are more frequently made use of by all ranks of the people.’ Earlier in the Account a letter from William Creech to Sir John Sinclair recorded the quantity of wheat made into flour at the Water of Leith mills belonging to the incorporation of bakers and showed that it had grown from 22,762 Midlothian bolls in 1750 to 48,257 bolls in 1791. Besides these mills, Creech emphasised, there were several others grinding flour for the city as well as that sent to the city by the millers and bakers of Dalkeith, Musselburgh and Lasswade.

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Edinburgh was close to the developed farms of the Lothians where wheat was grown and with new forms of transport being introduced there was an increase in the availability of this cereal. The descriptions of agriculture by ministers of parishes near to the city in the *New Statistical Account* invariably contain references to the cultivation of wheat, although there are frequent statements which emphasise the continued importance of oats. In Dalkeith, 18,000 quarters of wheat and 43,000 quarters of oats were brought to the local market from farms in the parish, wheat yielding £2,187, as compared to £1,650 for oats. The thirty-four farms in the parish of Liberton grew 685 acres of potatoes, 850 acres of oats and 738 acres of wheat. The same pattern is repeated in all of the parishes for which detailed reports are available and it is fairly representative of many of these that mention is made of crops ‘raised for the Edinburgh market.’ The proximity of these farms to the city enabled a two-way traffic to be carried on; manure for the fields came out from Edinburgh and oats, potatoes and wheat were taken in to the city. The new modes of transport, especially the railways, made this process simpler and more efficient but with these improvements appeared changes in eating habits and in expectations. Mokyr points out that ‘the modernisation of transportation affected the supply of agricultural products in a way similar to improvements in agricultural productivity…’. The development of canals and railways and the improvement in roads and shipping removed the local element from food; wheaten bread became popular in areas at a distance from where it was cultivated and may well have replaced oats as the staple in the south of Scotland and in the towns and cities. As Teuteberg points out, data showing an increase in the amount of wheat eaten, quoted above, reveal only tendencies in consumption, they do not tell us who was now eating wheat nor do they furnish details of social class, work, age, gender and income. If these facts are taken into account, the findings of the Poor Law Commission and later interpretations of the food of the Scottish working class based

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25 In 1842 the Edinburgh and Glasgow Railway was opened, in 1846 the North British line from Edinburgh to Berwick opened, and in 1848 the Caledonian line from Edinburgh to Carlisle was completed, A.W.Ritchie, ‘Social Conditions in the Nineteenth Century’, *Studies in the Development of Edinburgh*, (Edinburgh, 1939), p.37.
on the Poor Law Report may be correct when applied to the rural rather than to the urban working class and, more particularly, the Edinburgh working class. It is noteworthy in this context that twenty five years later The Report on the Dietaries of Scottish Agricultural Labourers (1869) estimated that oatmeal was the staple of 90 per cent of the working class in Scotland. This is at odds with a modern analysis, which estimated that 40 per cent of the population of Scotland was eating wheat by 1841, the author suggesting that the growth of wheat eating in Scotland and the North of England was in proportion to the degree of urbanisation but that the movement away from oats may have been interrupted by periods of unemployment and trade depression in 1838–43 and 1847–48. No breakdown of the geographical or social distribution of this percentage is given but the likelihood is that wheat consumption in the form of bread was substantial in Edinburgh by the 1840s.

The emphasis on the importance of oatmeal in the Scottish diet has been criticised. Gauldie contends that oatmeal porridge and bannocks became 'the improved food' of the rural population who remained on the land after the change in farming methods. She maintains that before agricultural improvement the food of the common people was bread, made from the kind of meal that was available in their own region. In Scotland, 'mashlum, a mixture of peas, beans, bere or oats was still generally eaten by all classes, until the end of the eighteenth century and by working people after except in some Lowland areas.' Fenton cites evidence to suggest that mashlum was found generally in the main farming areas of eastern and southern Scotland, from the Highlands to the Lowlands, and maintains that the ingredients of mashlum were sown by farmers in order to make bread to feed their servants. It was not only in Britain that a mixture of rye and wheat was used to make bread, in Belgium around the middle of the nineteenth century 'maslin', a similar mixture, was eaten. This type of mixture was known as maslin in England where the mixture was composed of wheat and rye or barley, often with boiled mashed potato added. There were differences in the type of bread eaten even within a single county—in Moray, the

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30 Lis and Soly, 'Food Consumption in Antwerp between 1807 and 1859', p.471.
coastal districts used a mixture of bere meal, pulses and rye or wheat flour whereas inland, oatmeal was used to make oatcakes with no such mixing being practised.  

The relative importance of bread and oatmeal, in whatever form, cannot be established with any degree of certainty, but it is probable that the move away from a rural way of life, where a subsistence economy predominated, to an urban existence changed the types of food that people ate. There were many reasons for this; berries, herbs, plants and wildlife, generally available in the countryside, were not so easily obtained in urban surroundings; there were foods that were too expensive for the working man and the possession of the means with which to prepare food was often missing. The high price of fuel and the greater convenience of the loaf determined the baker’s success and ‘as his monopoly increased so that of the mealman diminished, leaving the consumer with fewer alternatives as to choice of product and raw material.’ In Edinburgh, in 1844, there were 201 bakers, thirty-one listed as having premises in the Old Town; there were eight in the Canongate, three in the Lawnmarket, two in the Cowgate and two in the Grassmarket. Williams records that there were 207 bakers in 1835, falling to 188 in 1866. As a comparison, Glasgow in 1845 had 230 bakers. It is unlikely that these bakers sold their bread other than in their immediate locality.

It was not merely convenience that encouraged the change from oatmeal to wheat. It has been calculated, for example, that the price of oatmeal in Glasgow fell by about 20 per cent between 1810 and 1831, compared to a fall of 45 per cent in the cost of white wheaten bread and 40 per cent for the brown “household” bread. The relative merits of oats and wheat may have influenced the places and the people who changed from oats to bread. The ease with which the former can be prepared with a minimum of equipment must have persuaded many to stay with oats whereas one

33 Collins, ‘Dietary Change and Cereal Consumption in Britain in the Nineteenth Century’ p.108.  
advantage of wheat was the ease of its storage and transportation as bread. Bread, however, requires baking in ovens and this became a specialised activity in villages and towns. Thus oatmeal, which by the nineteenth century formed the staple of the agricultural worker, gradually gave way to wheat, which in the form of white, low extraction flour was poor in minerals and vitamins. The great increase in the use of wheat is apparent from the statistics recorded by Porter for the quantity of wheat and wheat flour imported; in the decade from 1811 it amounted to 459,578 quarters; between 1841 and 1849 the total was 2,588,706. If the increase in population is taken into account this represents a six fold increase per person in Britain.

Food Adulteration

Cooking implements, a fire on which to cook and fuel to feed it were necessities but these were not always readily available in the homes of the urban poor. Therefore many were forced into buying prepared food, most commonly bread, which was cheap, available, apparently nutritious and above all had the advantage that it required no cooking. There was a drawback, however, because of the extent to which food and bread in particular was adulterated, a scandal that was not corrected until, in 1860, legislation was introduced to prevent the worst excesses. Engels wrote in 1844: ‘producers and shopkeepers adulterate all foodstuffs in a disgraceful manner with a scandalous disregard for the health of the ultimate consumer.’ It was understandable that common concerns during the early Victorian period were food adulteration and food quality. Filby, an authority on food adulteration gives the legal definition of adulteration as follows: ‘an article is adulterated when any substance

37 P.P.An Act for preventing the Adulteration of Articles of Food or Drink. 23 and 24 Vict.Cap.84, 1860. Before this Act became law the following reports and bills had been presented to parliament: First Report of Select Committees on Adulteration of Food, Drink, and Drugs with Evidence and Appendix, 1854–55, viii.221; Second Report of Select Committee on Adulteration of Food, Drink, and Drugs with Proceedings. Evidence and Index, 1854–55.viii.373, Parliamentary Bills: Prevention of Adulteration of Food or Drink, 1857, (session 2), 1.9; 1859, (session 1), 1.5; 1860.1.49. After the Act further Bills were presented in 1857, 1859, 1860, 1867–68 and 1868–69.
other than that which the article purports to be is mixed with, or added to, or placed upon it, either to increase the bulk or weight or apparent size, or to give it a deceptive appearance.' There is ample evidence that substances were added to food during the first half of the nineteenth century with the express purpose of deceiving the consumer and producing more profit for the seller. Filby says that there was a marked increase in contamination of food after 1800 and that it was only the development of analytical chemistry that allowed controls to be instituted. Drummond believed that the extent of 'flagrant adulteration' was at its maximum between 1800 and 1850. As Wohl points out adulteration of food was not unique to the nineteenth century but there were factors that 'increased its scale and range.' Among these factors, he suggests, were mass production, marketing techniques, the anonymity of urban life and 'the scale of operations called for by the large, densely packed urban populations.' The shift from a predominantly rural to an increasingly urban society changed a subsistence agricultural economy in which the origin of food was usually local and less likely to be adulterated to an urban one where most food was purchased from shops and dealers. In cities the opportunities for tampering with food were greater and it is not surprising that the section of society thought to be most vulnerable to impure food was the working-class. As Burnett remarks, 'for the poor, tied by indebtedness to the local grocer and baker, there could be little improvement until the advent of effective legislation.' He points out also the important role played by the Co-operative movement in England at this time; the Rochdale Pioneers in 1844 beginning the process that led to the modern co-operative retail society. This kind of self help organisation did not exist in Edinburgh during the period under review, presumably leaving the poor at the mercy of unscrupulous merchants; there is no surviving evidence of food adulteration in the city during the middle of the nineteenth century but it is difficult to believe that it did not exist.

The types of food and drink most commonly adulterated were bread, beer, tea and milk, all basic items of a working class diet and critical if the nutritional status of the


poor was to be maintained at adequate levels. Richard Evans in a study of cholera in Hamburg makes the point that 'a decline in quality could easily nullify an increase in quantity.' The quality of milk supplied was often appalling; watering and removal of the fat were very common practices and butter was sold with as much water added as it could hold. Bakers have been responsible for introducing a variety of contaminants to bread and at one time or another turnips, potato flour, meal, lime, chalk, alum, bean meal and acorns have all been found with the commonest contaminant alum, introduced to make the loaf whiter and therefore more expensive. Adding alum and potatoes was such a common practice that Pereira in a section headed 'Wheat—Bread' wrote 'bakers generally employ, in addition, potatoes and alum.' He disapproved of alum, its use being prohibited by law, but he did not object to the potato flour being added. The addition of alum to flour by millers was common and they were also accused of making flour from substances other than wheat, for example, potato, Indian corn, rice flour and rye. Unfortunately, no direct information has survived on food adulteration in Edinburgh, or indeed in Scotland, but it is probable that similar deceptions to those described above were practised in Scottish cities, to the detriment of the nutrition of the poor who were forced to rely on the cheapest food, often the most susceptible to adulteration. An already meagre diet was further reduced by unscrupulous shopkeepers with consequences to health that are incalculable.

Diet of the Urban Poor

The type of food eaten by the poor in the middle of the nineteenth century cannot be stated with certainty but the evidence is that bread, either wheaten or mixed as mashlum, oatmeal and potatoes were predominant. There is no evidence as to how much food was obtained from charitable sources or how much shopkeepers gave to the poor, most often food that could not be sold, nor is there any evidence as to how much surplus food came from the houses of the better off. Another question that

44 Drummond and Wilbraham, The Englishman's Food, pp.301 & 303.
45 J.Pereira, A Treatise on Food and Diet, (London, 1843), pp.147–149.
cannot be answered is to what extent alcohol contributed to energy intake, the number of spirit dealers in the city suggesting that the amount may have been considerable. Further evidence will be obtained from the dietaries recorded by Christison in 1854, which include those of Perth prison (1844), Edinburgh Infirmary full diet (1843), and Edinburgh Charity Workers’ Diet (1847). All these diets have oatmeal and bread in roughly equal quantities as part of the daily ration, but the type of bread is not specified. For example, the prison diet had twelve ounces of bread and fourteen of oatmeal; the hospital diet four and a half ounces of oatmeal, three ounces of bread; the charity workers’ had eight ounces of oatmeal and eight ounces of bread, but again the kind of bread is not made clear. It is significant that potatoes did not appear in either the prison or the charity workers’ diet but that of the infirmary included sixteen ounces of potatoes with dinner and a further sixteen ounces with supper each day. The inclusion of two pounds of potatoes daily in the hospital diet and its absence from the prison and workhouse diet suggests that the potato was seen as a valuable part of a sick person’s diet. Whether oats or wheat predominated as the staple cereal is uncertain but there is little doubt that the potato occupied an important place in the diet of the new urban labour force and that the potato famine in the middle years of the decade had a considerable impact on the poor of urban Scotland.

The Potato Blight

The potato blight reached Scotland in 1845, infecting the potato plant in the south of the country to a very great extent but leaving the crop in the highlands and islands untouched. The origin of the disease, according to nineteenth century theorists, was the northern Andes region in South America when the newly independent South American states began to trade with Europe. This explanation is supported by Bourke who states that ‘among the newly introduced varieties which went down in the first attack in Belgium in 1845 were three which bore the significant names “Lima”, “Péruviennes” and “Cordillières.”’ The disease was first reported in Europe from the Courtrai area of Belgium in June 1845 and its spread to neighbouring countries,

47 Christison Papers, Tables of Nutriment of Various Dietaries.
including Britain and Ireland, was rapid. Theories as to the cause were many and equally numerous were the proposed cures; an editorial in *The Gardeners' Chronicle and Horticultural Gazette* claimed: '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.' The calamity in Scotland affected the western highlands and islands to a greater degree and for a longer period than other parts but research proves that the rest of Scotland did not escape.

It is now known that the cause of the potato blight is a fungal infection, *Phytophthora infestans*, the spores spreading rapidly in wet, windy weather, conditions that prevailed in many parts of the country in 1845 and 1846. The first reports of the blight in Britain were from the Isle of Wight in August 1845 and by the middle of September most counties of England, the eastern counties of Ireland, and the southern parts of Scotland were affected. The potato crop in the highlands and islands was unaffected in 1845 but the following year infected seed was planted and prolonged wet weather encouraged the spread of the fungal spores. A disastrous failure of the potato crop resulted 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 became apparent in the last quarter of 1846 and in the highlands, including the lowland areas on the east of the country, the loss of the crop was disastrous. Devine estimates that all the crofting districts were affected, 90 per cent losing their entire crop and the farming districts were not spared with 80 per cent badly hit by the blight. It was ironic that a greater acreage than normal had been planted in the hope of a repeat of the good prices of 1845 and early 1846, when potatoes were exported from the highlands at extremely favourable rates. It was reported in April 1846 that five thousand barrels of potatoes were exported from North Uist to Glasgow and sold at almost twice the going rate.

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In November 1845, James Johnston (1796–1855), a Glasgow University graduate and lecturer in chemistry at Durham, was asked by the Highland and Agricultural Society of Scotland to collect and analyse reports on the failure of the potato crop in Scotland. The society also offered ‘a premium’ for analyses of diseased potatoes and instituted an inquiry, which was to be ‘Chemical, Botanical, Entomological and Practical’ in scope. A questionnaire was circulated and the report, which was published in pamphlet form, included replies from farmers in the lowlands, which suggest that the disease may have reached the south of Scotland earlier than is generally thought. A farmer from Maryholm in Dumfries-shire wrote of the sudden and unexpected appearance of the blight in the first week of September and Mr Girdwood of Featherhall, Corstorphine described the destruction of 80 per cent of his potato crop by the disease, which he had also first noted in early September. The replies from Ayrshire, Lanarkshire, Angus, Fife and Clackmannan were equally dismal; a proprietor from the latter county complained that there was not a sound field in the district and agreed with his foreman who had said ‘they may crack on 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’; he thought this was ‘one of the truest and most sensible remarks that I have yet fallen in with about the matter.’ Such resignation to the will of the almighty is also found in responses to epidemic disease and other apparent manifestations of divine displeasure; in 1842, at a time of fever in Scotland, the Moderator of the General Assembly of the Church of Scotland in a pastoral address spoke of ‘the alarming indications of the divine displeasure’ and in 1848 the General Assembly ‘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…’ were moved to ‘bear our willing testimony to the exemplary patience with which it [distress] has been borne by the poorer classes.’

The widespread distress to which the Moderator referred included the hardship caused to many by the lack of a dietary staple as a consequence of the potato blight. There were shortages in many parts of the country with, as will be described later in the chapter, food riots breaking out in towns and cities. In March 1847, the Edinburgh committee of the Central Board of Management for Highland Relief expressed its concern at the disturbances in the parts of the country under the Board’s supervision and concluded that ‘these disturbances have originated in attempts to prevent the export of grain under the mistaken notion that this would lower the price of provisions in these districts.’ 55 This was probably an accurate assessment of the position at this time—Devine concludes that the loss of the potato crop did not significantly affect the price of grain except briefly in late 1846 and early 1847.56 However, there were sharp movements in the price of basic commodities with the cost of the coarse four pound loaf rising from seven pence to nine pence between August and October 1846, by January 1847 the price was eight pence and the eight pence fine loaf of August 1846 cost eleven pence in January 1847. The difference between the rise in price of the coarse loaf and the fine loaf arose from the extra wastage involved in milling the finer flour used for the more expensive loaf. These increases were short-lived and confirm Devine’s conclusion that the price of grain was hardly affected by the potato blight but for a brief period these changes did cause alarm among the working class up and down the country, an alarm that was manifested in civil unrest.

Food Riots in Scotland

People living in the farming districts of the highlands, in the rural lowlands and in urban Scotland did not suffer so severely from the loss of the potato as many in the crofting districts of the north, nevertheless, one estimate suggests that by the end of March 1847 nearly 50 percent of families in the farming districts were likely to have consumed their grain crops and by the end of April the figure was closer to 75

55 Glasgow Herald, 12 March 1847; the Central Board was established in 1847 combining three relief committees: the Free Church relief committee, the Edinburgh committee and the Glasgow committee.
56 Devine, The Great Highland Famine, p.113.
per cent. Little grain or potato was available for export to other parts of Scotland and any suspicion that grain was being exported triggered violent attempts to stop the trade. The first such disturbance took place in Inverness early in 1846 and these ‘Inverness potato riots’ for a time prevented the export of local potatoes to London, the poor of the town having been successful in persuading the carriers not to deliver potatoes to the ships. Civil disorder was widespread in the north by January 1847, first in MacDuff but rioting spread rapidly to many coastal towns along the Moray and Cromarty Firths and north into Caithness. It was reported by the Glasgow Herald that in Macduff a mob had tried to stop the shipment of meal and grain; in the same edition the price of meal was said to have risen from 18 to 30 shillings per boll. ‘Serious Rioting in Caithness’ was the headline reporting a riot in Wick, ‘the excitement so general throughout the north with respect to the shipment of grain has, in Wick, resulted in a collision between the populace and the military.’ Two companies of infantry were deployed to guard a cargo ship loading grain but their presence did not deter the mob who attacked them with stones and other missiles. The Riot Act was read and the crowd forced back at bayonet point but when those arrested were being escorted to prison the soldiers were once more attacked, several of the men were hurt and both the Sheriff and the Provost were struck. After a warning had been issued, the order to fire was given and two persons were wounded. The vessel sailed from Wick three days after the riot whereupon the detachment of soldiers left for Thurso to deal with similar disturbances that were causing official concern. By March, there were reports of food riots in Ross-shire, at Invergordon and Portmahomack and at Grantown in Inverness-shire. As Richards records, there were some inland towns affected also, mainly in the north east at Elgin, Fochabers, Keith, Huntly and further inland at Grantown on Spey. The fear of disorder lingered for several months and there was very little official confidence in the

57 Devine, The Great Highland Famine, pp.41 & 42, the source of the figures being the Destitution Papers, Second Statement of the Destitution Committee of the Free Church, 6–13.
58 Glasgow Herald, 25 January 1847.
59 Inverness Courier, 10 March 1847 & 17 March 1847.
60 Disturbances took place in the following localities: Peterhead, Portessie, Findhorn, Cromarty, Fraserburgh, Buckie, Nairn, Dingwall, Gardenstown, Portgordon, Fort George, Foulis Point, Macduff, Kingston, Inverness, Evanton, Banff, Garmouth, Beauly, Rosskeen, Portsoy, Lossiemouth, Avoch, Tain, Cullen, Cummingstown, Fortrose, Invergordon, Portknockie, Hopeman, Chanonry Point, Balmoral, Findochty, Burghead, Rosemarkie, and Portmahomack.
stability of the situation in the north of Scotland. Military supervision of the area was to continue; the warship Birkenhead which had been in the Moray Firth for two months was replaced by another warship, the Bloodhound, and it was May before the authorities felt able to sanction the withdrawal of troops from Cullen, one of the north east coastal towns affected by rioting.62

Riots were not confined to the north with towns and cities of the central belt also experiencing unrest. An Edinburgh doctor wrote in March 1848 to his brother in Paris describing ‘the revolution on a small scale last night in Glasgow.’ He heard of the affair from a watchman who told him that the cause of the riot was the poor Irish who did not have enough to eat and ‘therefore they had to rise.’ Troops from Edinburgh, Hamilton and Stirling had been transferred to Glasgow to quell the rioting. The letter writing was interrupted to tell of a similar disturbance in Edinburgh:

As I write Mr. MacGregor comes in and tells me there is a riot in the High Street and Cowgate consisting of about 5,000 people shouting through the streets, breaking lamps, windows, etc., and seeming in high spirit for a row... I rose to look out but before I could hardly get a sight every lamp in the street has smashed and it's not a short one (about the length of the High Street in Belfast). It is now 10 o'clock and as there are no military comparatively speaking in Edinburgh we expect to hear some fun before morning.

By midnight the cavalry from Piershill barracks were on West Nicolson Street where the writer lodged and the Pensioners were under arms guarding the Castle.63 Perhaps the writer hoped to impress his brother who would have been aware first hand of revolutionary events in Paris, but nonetheless, there remains a very clear impression of disorder.

There were other towns in the central district similarly affected with riots in Kilmarnock and strikes in the mining districts of Glasgow, Airdrie and Holytown. The riots in Glasgow were the most alarming and were said to have originated on the Green at a meeting of the unemployed who were demanding work ‘that they might

62 Inverness Courier, 18 May 1847.
support themselves and their families.’ A large crowd of men, boys and women were
gathered on the Green to hear a speech ‘calling on the multitude to exert their rights
— “to do a deed worthy of the name of France.” ’ The infantry, the cavalry and ‘a
body of respectable citizens who had been sworn in as special constables’ were
called out; by nightfall sixty rioters were in custody and the town was quiet.
Disturbances continued and next day in Bridgeton the military opened fire on the
mob, wounding five men, one mortally and three others so severely that they died of
their wounds.64 The Glasgow Herald reported that of the sixty-four males in prison,
the following statistics show. British grain imports during the 1840s, measured in thousands of hundredweight’s,
show an increase in wheat imports from 3,777 in 1845 to 16,663 in 1849 while
imports of maize rose from 242 to 15,464. The average price of corn per Imperial
quarter rose from 50s 10d in 1843 to 69s 9d in 1847. During the same period the
Bank Rate rose from 3½ per cent in January 1847 to 8 per cent in November 1847,
only returning to its previous level in June 1848.65 The loss of the potato crop in the
years from 1845 is reflected in the increased amount of grain imported and although
Alison emphasised the seriousness of the problem in the western highlands and
islands, explaining that ‘it is only in certain districts that the people have been
absolutely dependent on the potato and reduced to absolute destitution by its failure.’

The predominance of employed men amongst those imprisoned and the references to
the rights of the people suggest that food may not have been the chief cause for the
unrest and that political agitation was a factor. Nevertheless, food shortages and
rising prices were troubling all classes of society as the following statistics show.

63 Edinburgh University Library, Special Collections, unpublished letter from 25, Nicolson Street
dated 7th March 1848 written by Dr (?) Cunningham to his brother in Paris.
64 Scotsman, 8 March 1848, 11 March 1848 and Glasgow Herald, 10 March 1848.
65 Glasgow Herald, 17 March 1847.
been totally dependent on the potato but for whom the potato was an important part of their diet. For them the scarcity of potatoes and the increase in price of bread and meal at a time of economic depression and unemployment may not have been as critical but, as will be shown in a later chapter, there was increased morbidity as a result of malnutrition. The problem of the starving urban poor was not new and ideas as to how the issue might be resolved were still being influenced by the theories of men like Ricardo and Malthus writing earlier in the century but whose ideas were still current. Mokyr suggests that 'the fear of food scarcity' was the single motivating factor that led Ricardo and Malthus to their pessimistic conclusions in their works on political economy. Food scarcity was not a matter that concerned many physicians in the middle of the nineteenth century, (Alison was a notable exception), but with developments in chemistry and physiology, however, the importance of food to the health of the individual was now accepted, as will be demonstrated in the section which follows.

Attitudes to Nutrition

This section will consider medical, official and charitable reactions to food shortages and the food supply and at the same time will examine the hypothesis that a residual belief in the 'positive check' as a solution to population pressure affected the response to food crises. One Scottish physician aware of the importance of nutrition was Dr. R.D. Thomson, who in a paper read to the Philosophical Society of Glasgow in 1841 referred to Liebig's contention that unless a sufficient amount of carbon was provided by food it would have to be supplied "from the muscles and substance of the body; the latter becomes thinner and weaker, and, like an expiring taper, is extinguished by the influence of the most trivial causes." But physicians who thought sanitary improvement was the answer to epidemic disease greatly

outnumbered those who were alarmed about the effects of malnutrition on the poor. The sanitary improvers were disciples of Arnott and Kay, two physicians trained in Edinburgh but practising in England and highly influential in the movement for sanitary reform. Their contribution will be considered in a later chapter. On the other hand there were clergymen like Thomas Chalmers who believed that only evangelisation and parish control by the church would cleanse the slums of squalor, destitution and immorality. One Edinburgh physician, Andrew Combe (1797–1847), was the author of several treatises on digestion and health education and had republished Beaumont’s researches on Alexis St. Martin, described in an earlier chapter. Combe, a recognised authority on the subjects of digestion and nutrition, wrote in 1836:

...as a general fact, at least nine tenths of the lower orders suffer physically, morally and intellectually from being overworked and underfed .... It is true that very few persons die from actual want of food; but it is not less certain that thousands upon thousands are annually cut off, whose lives have been greatly shortened by excess of labour and deficiency of nourishment. ... during epidemics, too, the poor from their impaired stamina, almost invariably become victims in a proportion far exceeding that of the more wealthy classes.

The origins of nutrition science have been placed around 1840, ‘when a fair number of professional chemists and physiologists explicitly began to relate the chemistry of food to animal physiology’. Whether these new approaches to food and health influenced opinions on poor relief during the 1840s is debatable; Malthusian theory may have had a greater impact on attitudes to the nutrition of the poor. Thomas Malthus, writing in 1798 on the positive check to population, commented:

But I believe it has been very generally remarked by those who have attended to bills of mortality, that of the number of children who die annually, much too great a proportion belongs to those, who may be supposed unable to give their offspring proper food and attention; exposed as they are occasionally to severe distress, and confined,

perhaps, to unwholesome habitations and hard labour. This mortality
among the children of the poor has been constantly taken note of in all
towns. It certainly does not prevail in an equal degree in the country....

Malthus was well aware that the mortality in towns and cities was greater than that in
the country, especially in the case of children, and attributed this to a lack of proper
food. Livi-Bacci has shown that the calorie intake of historical populations can be
estimated and conclusions drawn as to the adequacy or otherwise of the energy
provision, but it is difficult, if not impossible, to calculate the nutritional status or the
presence or absence of essential nutrients in the diet of selected population groups.
However, deductions can made as to their diet using contemporary sources written
by professional men, usually medical, who had an interest in the physiology and
chemistry of nutrition. Information can be derived also from commentaries on diet
recorded by interested laymen and from records of wages and the amount of food
which could be purchased at periods during the 1840s in Edinburgh and in this
respect the manuscript lectures of Professor Robert Christison, later Sir Robert
Christison, are particularly valuable. McKeown’s contention that ‘the most
acceptable explanation of the large reduction of mortality and growth of population
which preceded advances in hygiene, is an improvement in nutrition due to greater
food supplies’ has stimulated debate. Whatever the merits of McKeown’s
argument, it has encouraged research into the effects of malnutrition but as yet there
has been no study of nutrition and the nutritional status of specific populations in
Scotland. As pointed out in the introduction, the nutrition of selected populations in
England and Wales has been studied and that of Ireland has been researched in
considerable detail as a result of interest in the potato famine. It is hoped that the
next chapter will deal with this comparative neglect of nutrition and nutritional
standards in Scotland by analysing several Edinburgh dietaries collected by Professor
Christison during the 1840s. The quality of these diets will furnish information both
as to the type of food in common use in the city and its nutritional content. This

75 D.J. Oddy, ‘Food in nineteenth century England: nutrition in the first urban society’, Proceedings of
the Nutritional Society, 27, (1970); Burnett, Plenty and Want, (London, 1979); E.M. Crawford, (ed.),
Famine: The Irish Experience 900–1900, (Edinburgh,1989); C. Kinealy, A Death–Dealing Famine,
analysis will not of course reveal the type, quantity or quality of the food eaten by the poor but will enable certain observations to be made on the relationship between malnutrition and epidemic disease.

Chapter Three

Analysis of Edinburgh Dietaries.

The Christison papers include the handwritten notes of lectures on nutrition given by Professor Christison to medical students from the 1830s to the 1860s and with these is a pamphlet dated 1854, printed in Edinburgh, entitled *Tables of Nutriment in Various Dietaries*, annotated in Christison’s hand.¹ On page one are tabulated the nutriment content of various dietaries ranging from that of the British Navy, Hessian Soldiers, Australian Shepherds and French Troops in the Crimea to the diets of Heriot’s Hospital Boys, Scotch General Prison, Edinburgh Charity Workhouse and the Infirmary full diet.² This table divides what is referred to as ‘real nutriment’ into their carboniferous and nitrogenous, (equivalent to carbohydrate and protein) elements, without specifying the types or quantities of food that make up these diets; it is only later in the document that a number of daily diets are broken down with accurate amounts of the daily provision recorded. The next page has a ‘Standard Table of Nutriment’ (1849) in which the carboniferous and nitrogenous percentage content of various foods is set out; for example oatmeal was said to contain 65.75 per cent carboniferous matter and 16.25 percent nitrogenous matter, the remaining 18 per cent was not considered to be of nutritional value; potatoes contained 24.5 per cent carboniferous, 2.5 per cent nitrogenous, the remaining 73 per cent was thought to be worthless.³

On succeeding pages of the pamphlet are listed particulars of nine diets, the first being the Hessian Soldiers Diet with the name Liebig in parentheses, followed by the Berwickshire Reapers Diet, private information in parentheses, and one diet based on detail from the Irish Poor Law Commissioners. The provenance of the other six diets

¹ Edinburgh University Special Collections, Christison Papers, *Tables of Nutriment in Various Dietaries*, (Edinburgh, 1854). The handwriting is the same as that in the reports of cholera cases treated by Christison filed in the Royal College of Physicians of Edinburgh Cholera Returns 1848–49.
² *Tables of Nutriment*, p.1.
recorded is not given but, as was stated in chapter one, Christison was one of the medical advisers to the Charity Workhouse, responsible with Alison for a diet prepared at the request of the Board of Supervision in 1846. In 1847 he wrote an account of the scurvy outbreak at the Perth General Prison and was therefore well placed to obtain details of the diets in these institutions. Christison was not only a consulting physician to the Edinburgh infirmary but also sat on the board of managers, and as an adviser to the hospital on dietary matters was involved in planning the diet he recorded.

<table>
<thead>
<tr>
<th>Dietary</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hessian Soldiers’ Diet (Liebig)</td>
<td></td>
</tr>
<tr>
<td>Berwickshire Reapers’ Diet (Private Information)</td>
<td></td>
</tr>
<tr>
<td>Yorkshire Farm Labourers’ Diet (Private Information)</td>
<td></td>
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<tr>
<td>Irish Labourers Diet (Poor Law Commissioners Report)</td>
<td></td>
</tr>
<tr>
<td>Edinburgh Infirmary Full Diet, 1843</td>
<td></td>
</tr>
<tr>
<td>Convalescent Diet (Personal Experiment)</td>
<td></td>
</tr>
<tr>
<td>Perth General Prison Highest Diet, 1844</td>
<td></td>
</tr>
<tr>
<td>Scottish Local Prisons, Lowest Diet, 1851</td>
<td></td>
</tr>
<tr>
<td>Edinburgh Charity Workhouse Workers’ Diet, 1847</td>
<td></td>
</tr>
</tbody>
</table>

Table 3.1. Dietaries recorded by R. Christison, 1854

When Alison and Christison revised the diets of the Edinburgh Charity Workhouse they prescribed, first, a diet for the healthy who did not work, secondly, a diet for the healthy who did work and lastly, a diet for the infirm and the sick. The diet recorded in Christison’s pamphlet was that fed to the inmates who worked and differed only slightly

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5 Edinburgh University Special Collections, Lothian Health Services Archive, Minutes of the Edinburgh Royal Infirmary, January 1839, pp.154 & 155 records the appointment of Christison as one of the infirmary manager’s; an entry in the Minutes of the Edinburgh Royal Infirmary, April 1843 records that thanks were due to Dr Christison who had helped prepare eight diet tables including one called the full diet.

6 *Tables of Nutriment*. 

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from that given to the healthy who did not work.

Christison’s collection of diets is very similar in concept, albeit on a very much smaller scale, to that produced by Dr Jonathan Pereira, a London physician and fellow of the Royal Society, who published a treatise (1843) in which, amongst others, he lists diets from a foundling hospital, the Royal Military Asylum in Chelsea, the Naval Asylum in Greenwich, diets for paupers, for prisoners and for the sick. Christison who had a special interest in nutrition and nutritional associated illness would have been aware of Pereira’s work and influenced by it. In a similar fashion to Pereira, Christison outlined the amounts of food given at each of the three main meals of the day but went further by calculating the carboniferous and nitrogenous content of each item, equating this to what he called the nutritive proximate principle, terms that have little meaning or relevance in current nutritional practice. However, the detail documented in these diets is such that it was considered that employing up to date methods of nutritional analysis would yield information of great significance.

Accordingly, in the absence of a department of nutrition in the University of Edinburgh the department of nutrition at Queen Margaret University College, Edinburgh was contacted to arrange dietary analyses. With the kind and enthusiastic co-operation of Michael Clapham, nutritionist and lecturer in the department, energy and nutrient analysis of four diets was carried out. The software programme, used by nutritionists in the assessment of diets, calculates the energy supplied by the food and measures the absolute amounts of macronutrients, minerals and vitamins present which are then compared to a series of recommended norms for each, thus obtaining a true measurement of the quality of the diet.

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8 The analyses were performed using the CompEat 5 nutritional software package, produced by Carlson Bengston Consultants Limited Nutrition Systems with the value of food content based on the McCance food tables.
The first diet to be analysed by this technique was the Irish labourers' diet, the details of which Christison says were acquired from the Irish Poor Law Commissioners' reports; Christison explained that 'this was found to be the general diet of labourers in Ireland, living at home in their own way.' The amount of potatoes eaten by the Irish working man appears extraordinary but this consumption has been confirmed by other authorities.10

<table>
<thead>
<tr>
<th>Potatoes</th>
<th>143.75 daily ounces</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skimmed Milk</td>
<td>46.0 daily ounces</td>
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</tbody>
</table>

Table 3.2. Irish Labourers' Diet from Christison's Table of Nutriments.11

The figures for potato intake, Christison explained, were based on 'mean consumption, the extreme daily intake being 128 to 176 ounces of potatoes and 32 to 64 ounces of skimmed milk.' Computer analysis of the labourers' diet shows that the energy supplied was 3364 calories per day, above the 3000 calories suggested as the minimum for someone carrying out heavy physical work according to Halstead's formula cited in chapter one, page eighteen. The energy provided by this diet would ensure that the labourer maintained a good nutritional status so long as he remained free from disease. However energy measurement is only one part of dietary analysis; there are other parameters which have to be assessed and this was done by first calculating the reference nutrient intake (RNI), a measurement of the quantity of the nutrient, protein, vitamin or mineral that is sufficient for the needs of about 97 per cent of people in a representative group. In the case of the Irish labourer the RNI was satisfactory for protein, minerals and all vitamins except for vitamin A where the recommended intake was 700μg but the actual intake was only 13.04 μg, an amount sufficient to satisfy the body's requirements of only 1.9 per cent of the population. The Irish labourer existing on this diet would have been markedly deficient in vitamin A and his ability to resist and fight infection would have been greatly compromised. It has been pointed out that had the milk been full cream rather than the less expensive skimmed variety the levels of

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10 R.E.Hughes, 'Scurvy' in K.F.Kiple and K.C.Ornelas, (eds), The Cambridge World History of Food, (Cambridge, 2000), p.991 says that the daily consumption of as much as 6.5 kilograms of potatoes has been recorded.
11 Table of Nutriments, p.3.
vitamin A would have been more nearly normal.\textsuperscript{12} The dangerously low intake of vitamin A is highly significant and will be discussed later in the chapter when deficiencies in other diets are also examined.

In view of the later discussion on the epidemic of scurvy in Edinburgh and neighbourhood during the potato famine it is interesting to look at the amount of vitamin C provided by this diet. The RNI for vitamin C is 40 µg daily, while the actual intake in this diet was 257.4 µg, an amount far in excess of daily requirements, confirming the potato’s importance as an anti scorbatic when eaten in large quantities.

The Edinburgh Infirmary Full Diet, 1843, (table 3.3), was the next of the recorded diets to be analysed. The Infirmary minutes record that the full diet was one of eight diets offered to infirmary patients during the 1840s; these included the following: the low diet; the rice diet; the steak diet; the steak diet with added bread; the common diet; the common diet with added bread; the full diet and the full diet with added bread. There is no indication given as to whether a particular diet was prescribed for patients with particular illnesses or whether one diet was thought to be more nutritious than another but it seems likely that the full diet must have been close to the top of the nutritional scale. The fact that so many diets were thought necessary implies that adequate and varied nutrition was considered to be an important part of treatment.

\textsuperscript{12} M. Clapham, Personal Communication.
BREAKFAST
oatmeal for porridge 4.5 ounces
buttermilk 20 ounces

DINNER
boiled meat 6 ounces
potatoes 16 ounces
Bread 3 ounces
Broth vegetables 0 ounces
barley 2 ounces
meat 1 ounces

SUPPER
potatoes 16 ounces
new milk 10 ounces

Table 3.3. Edinburgh Infirmary Full Diet, 1843.13

Computer analysis establishes that the energy derived from this diet was 2266 calories daily as against a recommended intake of 2850 calories, a shortfall of about 20 per cent, indicating that the nutritional status of someone suffering or recovering from serious illness or surgery would have been seriously compromised as would their ability to fight disease or recover from it. As was discussed earlier, (chapter one, page fifty-four), synergy between malnutrition and disease has a negative effect on nutritional status initiating a downward spiral because the body's demands on nutrient stores are very much greater when it has to deal with infection, chronic disease or trauma. During recovery from illness nutritional needs remain high to enable repair of tissue damage to be carried out. The energy provided by this diet is insufficient to meet these increased demands but it is possible that a substance omitted from the diet table, alcohol, may have increased the calorie intake of at least some patients to more acceptable levels. There is no reference to the use of alcohol as a dietary constituent or as a treatment in Christison's works, although hospital records reveal that it was commonly used at this time, particularly for fever patients who 'in the judgement of the medical men' often required large quantities of wine.14 The quantity of alcohol purchased was considerable; the minutes of a meeting of the managers in January 1839 record their dissatisfaction about the increased expenditure on wines for fever patients during 1838 when 436 dozen bottles of port, twenty five dozen bottles of sherry and 1,633 dozen bottles of ale and porter were purchased. If alcohol was given on a regular basis to patients suffering from

13 Table of Nutriments, p.3.
14 Minutes of the Edinburgh Royal Infirmary, January 1839, p.148.
fever and other infections the energy from this source might have been sufficient to ensure an adequate nutritional status. Discounting the possible contribution of alcohol, the less than adequate amount of energy provided by the infirmary diet is to some extent balanced by the high intake of protein, 121.9 grams daily as opposed to the recommended intake of 55.5 grams daily. There are two micronutrients, vitamin A and iodine, which the analysis shows are below the recommended levels, (table 3.4).

<table>
<thead>
<tr>
<th></th>
<th>RNI</th>
<th>Actual</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>700.0 µg</td>
<td>288.38 µg</td>
</tr>
<tr>
<td>Iodine</td>
<td>140.0 µg</td>
<td>95.24 µg</td>
</tr>
</tbody>
</table>

Table 3.4. Micronutrients in the Infirmary Full Diet, 1843.\(^{15}\)

The iodine intake would be sufficient for 68 per cent of the representative group but over a short period this shortfall would significantly affect thyroid function, the organ mainly affected by iodine lack. Vitamin A intake is calculated as being sufficient for only 41 per cent of the representative group so that nearly 60 per cent of patients would have received inadequate amounts of the vitamin. The small amount of vitamin A in this diet is a direct result of low dietary fat which in turn is caused by the need to minimise the cost of feeding patients — fat is always an expensive commodity. In a person whose stocks of the vitamin may already have been low from a poor diet the lack of new vitamin A in the hospital diet would have impaired the immune system’s ability to fight infection and would have compromised or delayed recovery.

The convalescent diet recorded by Christison was a personal experiment undertaken when he was forty seven years of age in 1844, recovering from a febrile illness. When he began to record his food intake he mentions that his appetite was good, he was taking moderate exercise and both his weight and strength were increasing. There are items that are conspicuous by their absence in the other diets analysed, an absence that was probably determined by expense. For example, eggs and cream appeared in Christison’s

\(^{15}\) CompEat 5 Nutritional Software, analysis carried out by M.Clapham, Department of Nutrition, Queen Margaret University College, Edinburgh, 2002.
diet but were absent from all other diets, as were expensive commodities such as tea and coffee.

<table>
<thead>
<tr>
<th>BREAKFAST</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>bread</td>
<td>8 ounces</td>
</tr>
<tr>
<td>egg</td>
<td>2 ounces</td>
</tr>
<tr>
<td>butter</td>
<td>1 ounces</td>
</tr>
<tr>
<td>sugar</td>
<td>0.5 ounces</td>
</tr>
<tr>
<td>cream</td>
<td>0.25 ounces</td>
</tr>
<tr>
<td>coffee</td>
<td>0.5 ounces</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DINNER</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>meat</td>
<td>8 ounces</td>
</tr>
<tr>
<td>bread</td>
<td>3 ounces</td>
</tr>
<tr>
<td>potatoes</td>
<td>6 ounces</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TEA</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>bread</td>
<td>6 ounces</td>
</tr>
<tr>
<td>butter</td>
<td>1 ounces</td>
</tr>
<tr>
<td>sugar</td>
<td>0.5 ounces</td>
</tr>
<tr>
<td>cream</td>
<td>0.25 ounces</td>
</tr>
<tr>
<td>tea</td>
<td>0.25 ounces</td>
</tr>
</tbody>
</table>

Table 3.5. Christison's Convalescent Diet.16

The analysis establishes that the energy obtained was 2,422 calories compared to the recommended level of 2,347 calories. The amount of vitamin E was low, 2.1 mg, and the RNI was 3.67 mg, a difference that is not critical. Vitamin A intake was 757.56 µg, against a recommended level of 700.0µg; vitamin C intake was low at 26.68 mg, an amount sufficient for only 66 per cent of the representative group, the RNI being 40.0mg. The low vitamin C intake is the result of a diet which had no fruit and the only vegetable was the potato.

16 Table of Nutriments, p.4.
The last dietary subjected to computer analysis was the Edinburgh Charity Workhouse Workers' Diet, 1847, (table 3.6), an establishment where the average age of the workhouse inmates carrying out work was sixty years, the records showing that those who did no work were given less food and at times beer was given instead of milk.17

<table>
<thead>
<tr>
<th>BREAKFAST</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>oatmeal for porridge</td>
<td>4 ounces</td>
</tr>
<tr>
<td>buttermilk</td>
<td>15 ounces</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DINNER</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>bread</td>
<td>8 ounces</td>
</tr>
<tr>
<td>broth: barley</td>
<td>2 ounces</td>
</tr>
<tr>
<td>pease</td>
<td>0.5 ounces</td>
</tr>
<tr>
<td>vegetables</td>
<td>1.5 ounces</td>
</tr>
<tr>
<td>ox head, hough or beef</td>
<td>2 ounces</td>
</tr>
<tr>
<td>broth in total</td>
<td>30 ounces</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SUPPER</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>oatmeal</td>
<td>4 ounces</td>
</tr>
<tr>
<td>skimmed milk</td>
<td>15 ounces</td>
</tr>
</tbody>
</table>

| Total daily ounces of nutritive proximate principles | 16.95 |

Table 3.6. Edinburgh Charity Workhouse Workers' Diet.18

The analysis shows that energy intake was 2087 calories, 11 per cent below the RNI of 2347 calories but the amount provided would be sufficient for 89 per cent of the representative group. The carbohydrate and protein amounts were both above those recommended but the fat consumed would have been sufficient for only 75 per cent. The dietary cannot be criticised because of these levels but there were significant and potentially dangerous shortfalls in other nutrients as table 3. 7 reveals.

<table>
<thead>
<tr>
<th>ACTUAL LEVEL</th>
<th>RNI</th>
</tr>
</thead>
</table>

17 Tables of Nutriment, p.4.
18 Tables of Nutriment, p.4.
Iodine | 85.33 µg | 140.0 µg  
Vitamin C | 14.03mg | 40.0mg  
Vitamin A | 217.95 µg | 700.0 µg  
Vitamin E | 3.59mg | 4.25mg  

Table 3.7. Nutrient and RNI levels in the Charity Workhouse Workers' Diet.\(^{19}\)

The two crucial deficiencies are the levels of vitamin C and vitamin A, which would have been sufficient for only 35 per cent and 31 per cent of the representative group respectively. The deficiency of vitamin C was not such as to produce frank scurvy since it has been estimated that 10 mg per day will prevent its development—although it is currently recommended that the daily intake should be between 30 and 60 mg daily.\(^{20}\)

However, the small amount of vitamin C would have resulted in a depletion of tissue stores and induced the features of ill health associated with so called sub clinical scurvy. Although vitamin C has an important role in many bodily functions the early signs of deficiency often go unrecognised and unreported, being relatively non specific. Fatigue, weakness, shortness of breath, loss of appetite, aching bones and joints, apathy, depression and emotional disturbances have all been blamed on deficiency of vitamin C but, as has been pointed out, diets which are so poor as to cause sub clinical scurvy are generally deficient in many other essential nutrients and it is sometimes difficult to identify the factor or factors mainly responsible.\(^{21}\)

This marginal state of vitamin C deficiency is seen most often in particular population groups, for example elderly people living alone and those living in institutions. It has been observed that the intake of vitamin C in institutional diets is compromised by ‘an additional loss of the vitamin in large scale institutional cooking’ and during the mid nineteenth this was a factor in the appearance of scurvy.\(^{22}\)

The actual amount of vitamin C, calculated as 14.03mg daily by computer analysis, available to the inmates, may well have been less because of losses.

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\(^{19}\) CompEat 5 Nutritional Software, analysis carried out by M.Clapham.  
\(^{22}\) Basu and Dickerson, Vitamins in Human Health and Disease, p.142.
during food preparation. If florid scurvy did not become widespread in the workhouse it is probable that sub clinical manifestations of vitamin C lack were common.

The analysis shows that the vitamin A level is considerably below the recommended norm and although there are variations in the levels suggested by different countries the lowest national recommendation, that of the National Institute of Health and Nutrition, Japan, is 600 µg for males and 540 µg for females, considerably higher than that provided by the workhouse diet. In the USA recommended levels are higher still at 1000 µg for males and 800 µg for females. The outcome of vitamin A deficiency in the Charity Workhouse diet would be the appearance of ophthalmic symptoms, alteration in the epithelial lining of the respiratory tract and the gut and general impairment of the immune response. The epithelial lining is 'the first step in the protective mechanism against infection' and loss of its integrity often leads to bacterial colonisation and infection. The increased susceptibility to infections of the lung and the gut because of the loss of the protective function of the epithelial cells is further enhanced by the diminished immune status and a decreased ability to fight infection once established. 'Vitamin A deficiency and infections can establish a vicious cycle that induces and perpetuates systemic disease.'

In all four dietaries analysis reveals that there were serious deficiencies in the provision of several essential nutrients which would have had serious effects on health if consumed for prolonged periods; scurvy either frank or sub clinical would have developed in all except the Irish labourers and possibly Christison himself. The body's immune response would have been compromised by the lack of vitamin C in the cells involved in this process but of greater importance in this respect is the deficiency of vitamin A evident in all diets except that of Christison. If these diets were eaten only for a short period of time the damage to health would have been temporary and recovery of normal function would be expected. This assumes that the individual surviving on such a

23 Basu and Dickerson, Vitamins in Human Health and Disease, p.169.
24 Basu and Dickerson, Vitamins in Human Health and Disease, p.168.
diet was not suffering from a chronic illness and did not develop an infectious disease while his or her defence mechanisms were impaired. A prolonged period on this kind of diet would have affected health with the appearance of sub clinical scurvy and the development of respiratory and gastro intestinal infections, illnesses which would have been more prolonged and more severe than in a person with normal resistance. Diseases like measles, scarlet fever and virus induced diarrhoea are also much more severe in the malnourished.26

The calculation of the carboniferous and nitrogenous proportions in the nutritive proximate principles, referred to earlier in the chapter, may have been the method in use at the time to estimate the nutritional content of a diet. In Christison’s personal convalescent diet the carboniferous element amounted to 14.28 ounces, the nitrogenous to 4.08 ounces, in total 18.36 ounces daily. The Infirmary full diet, according to these calculations, supplied 21.84 daily ounces and the Irish labourer consumed 43.4 daily ounces. If the nutritional value of a particular diet was calculated by reference to these standards it is not surprising that certain diets fell below what is now considered to be acceptable.

The nutritional deficiency revealed by the analysis of three Edinburgh diets is evidence only of defects in the nutrition of a certain number of people for a short period of time and is not proof of widespread and prolonged malnutrition in the city in the period being studied. The question that cannot be answered is whether the diet of patient or inmate would have improved or deteriorated after discharge from the Infirmary or the workhouse. Firstly, it is doubtful if greater quantities of good quality food were available in the community to the poor on a regular basis and secondly it is very likely that the type of food, if not the quantity, offered in the two institutions was similar to that

25 Basu and Dickerson, Vitamins in Human Health and Disease, pp.168 & 291.
normally eaten by the poor of the city.27 If these propositions are accurate the deficiency of essential nutrients identified in the diets analysed would have continued after discharge with the ill effects specified.

As pointed out earlier Professors Christison and Alison were medical advisers to the Edinburgh Charity Workhouse and responsible for changes in the quantity and type of food given to the inmates. They had revised the diets of the Charity Workhouse in 1846 at the request of the Board of Supervision who were continuing the interest in workhouse diets shown by the Poor Law Commissioners earlier in the decade. The commissioners had questioned Mr Small, the treasurer of the Charity Workhouse, on the food allowed to the paupers in the city’s workhouses and were told that the diet had been considerably increased within the previous year as a result of the surgeon’s report and that ‘the dinners were fully doubled.’ Small explained that the reason for the improved diet was to prevent deaths which had risen from sixty each year when the diet was adequate to eighty four per annum when the diet was reduced. It appears that the motivation for dietary improvement in the 1830s was the cholera epidemic of 1832 when an increase in the diet ‘was found a necessary thing to prevent the introduction of that disease.’ Small told the commission that the diet had been reduced some years after the cholera outbreak until in November 1841 it had been increased to its present state.28 Tables of diets for the Canongate Charity Workhouse and the Edinburgh House of Refuge were given to the Poor Law Commissioners and the diets of outdoor paupers were also discussed. Dr John Smith, physician to the Edinburgh Charity Workhouse, told the commission in March 1843 that the diet had been increased a year previously because he thought that the health of the inmates had been affected by the previous diet which was too poor; health was now better with less diarrhoea and dysentery.29 The House Governor of the Canongate Charity Workhouse, Peter Cummings and William

27 M. Clapham, Personal communication in which he confirms that institutional diets customarily reflect the food eaten in the community.
28 P.P. Report from the Commissioners appointed for inquiring into the Administration and Practical Operation of the Poor Laws in Scotland, 1844, xx, pp.2–10.
Grey, the House Governor of the West Kirk, (St Cuthbert’s) Charity Workhouse handed over to the commission the diet tables of their respective institutions and these are reproduced along with the Charity Workhouse diet in table 3.8.

<table>
<thead>
<tr>
<th>Canongate Charity Workhouse</th>
<th>West Kirk Charity Workhouse</th>
<th>Edinburgh Charity Workhouse</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BREAKFAST</strong></td>
<td><strong>BREAKFAST</strong></td>
<td><strong>BREAKFAST</strong></td>
</tr>
<tr>
<td>oatmeal</td>
<td>oatmeal</td>
<td>oatmeal</td>
</tr>
<tr>
<td>4 ounces</td>
<td>4 ounces</td>
<td>4 ounces</td>
</tr>
<tr>
<td>churned milk</td>
<td>buttermilk</td>
<td>buttermilk</td>
</tr>
<tr>
<td>5 ounces</td>
<td>10 ounces</td>
<td>15 ounces</td>
</tr>
<tr>
<td><strong>DINNER</strong></td>
<td><strong>DINNER</strong></td>
<td><strong>DINNER</strong></td>
</tr>
<tr>
<td>bread</td>
<td>bread</td>
<td>bread</td>
</tr>
<tr>
<td>5 ounces</td>
<td>6.5 ounces</td>
<td>8 ounces</td>
</tr>
<tr>
<td>broth: barley</td>
<td>broth: barley</td>
<td>broth: barley</td>
</tr>
<tr>
<td>1.5 ounces</td>
<td>1.5 ounces</td>
<td>2 ounces</td>
</tr>
<tr>
<td>vegetables</td>
<td>vegetables</td>
<td>Pease vegetables</td>
</tr>
<tr>
<td>not stated</td>
<td>Not stated</td>
<td>0.5 ounces</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.5 ounces</td>
</tr>
<tr>
<td>meat</td>
<td>meat</td>
<td>meat</td>
</tr>
<tr>
<td>not stated</td>
<td>2 ounces</td>
<td>3.5 ounces</td>
</tr>
<tr>
<td><strong>SUPPER</strong></td>
<td><strong>SUPPER</strong></td>
<td><strong>SUPPER</strong></td>
</tr>
<tr>
<td>oatmeal</td>
<td>oatmeal</td>
<td>oatmeal</td>
</tr>
<tr>
<td>3.5 ounces</td>
<td>4 ounces</td>
<td>4 ounces</td>
</tr>
<tr>
<td>milk</td>
<td>milk</td>
<td>skimmed milk</td>
</tr>
<tr>
<td>5 ounces</td>
<td>10 ounces</td>
<td>15 ounces</td>
</tr>
</tbody>
</table>

Table 3.8. Comparison of the West Kirk and the Canongate Workhouse Diets with the Edinburgh Charity Workhouse Workers’ Diet.30

Notes: 1) The Canongate workhouse diet has churned milk given at breakfast. I assume that this is skimmed milk, milk obtained after making butter.

2) Children in the West Kirk Workhouse, in addition, were given 1 gill of milk, type not specified, an extra that was recently dispensed with. and were given 3.25 ounces of bread at 4 pm.

3) The Canongate Workhouse diet does not specify the type of milk given for supper.

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When the diets are compared it is plain that the Edinburgh Workhouse diet is superior to either of the other two and although these two diets have not been subjected to computer analysis, the data presented in table 8 permit certain conclusions to be made. Computer analysis showed that the Edinburgh workhouse diet was deficient in both vitamin C and vitamin A; it follows that the Canongate and the West Kirk workhouse diets contained even smaller amounts of these two essential nutrients.

The number of people in Edinburgh and the associated burgh of the Canongate receiving help in 1843 from the three workhouses is shown in table 3.9.

<table>
<thead>
<tr>
<th></th>
<th>Indoor</th>
<th>Outdoor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edinburgh Workhouse</td>
<td>420</td>
<td>629</td>
</tr>
<tr>
<td>West Kirk Workhouse</td>
<td>500</td>
<td>1195</td>
</tr>
<tr>
<td>Canongate Workhouse</td>
<td>107</td>
<td>370</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1027</td>
<td>2194</td>
</tr>
</tbody>
</table>

Table 3.9. Number of Indoor and Outdoor Paupers on the Workhouse Rolls in 1843. 31

Notes: 1) The Edinburgh Workhouse also had 260 children as inmates, not included in the figure of 420.

In addition to those who were helped by the three workhouses, in March 1843 there were 422 people in the House of Refuge and, according to one of the managers, the number who were applying for admission was increasing. He commented ‘it is life to them, if they did not come to us they must beg or steal, or die of starvation.' 32 There were other means whereby the starving were able to obtain some food; David Ridpath, a manager of the Edinburgh Charity Workhouse, explained that many old people got food

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31 P.P. Report into the Administration and Practical Operation of the Poor Laws in Scotland, 1844, xx, pp.10 & 64 and volume xxv, Appendix, Analysis of the Poor, pp.ix.
from families where they had once been servants and he also knew of families 'that have their regular decent paupers, who come every day to get something.'

The professional and domestic limitation in Christison’s dietary advice is clear when his convalescent diet is examined. This had no fruit or vegetables, suggesting that these were not thought of value by Christison or the more prosperous, but were considered to be the food of the lower classes. The poor could buy vegetables such as kale and turnips very cheaply and, as Davidson and his colleagues point out, 'a single helping of vegetables (90 grams) daily, even if it has been badly treated by the cook, will usually provide at least 10 milligrams of ascorbic acid, an amount known to prevent scurvy.'

There is no description of vegetable markets in Edinburgh during this period but Barker recalled a London street market of 1866, 'a poor man’s market' where 'one of the first ideas which strikes the stranger is that the London poor must be desperately fond of vegetables. The barrows are heaped with bunches of greens and Savoy cabbages.'

The productive farms of the Lothians, in close proximity to Edinburgh, would have provided the city’s markets with kale, cabbage and turnips, the food of the poor. Campbell also makes the point that the Scottish diet in the eighteenth and nineteenth centuries was nutritionally adequate only because of the presence of kale or cabbage, often taken as broth. Sir William MacArthur in his medical history of the Irish potato famine quoted Dr Curran describing events in Waterford where "scurvy was mainly confined to the class above what could be called poor" while the destitute were exempt, due in Curran’s opinion "to nothing else but the soup given them by the Quakers, which was well seasoned with vegetables.” This availability of kale or cabbage cheaply and the free soup handed out by various charities may explain the apparent absence of

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33 P.P. Report into the Administration and Practical Operation of the Poor Laws in Scotland, 1844, xx, p.38.
scurvy among the most destitute while the disease appeared among the better off in the city who disdained to eat the food of the poor.

These figures prove that there were several thousand people in the city who were unable to provide for themselves and for periods of time were forced to seek help from various charitable organisations. The food they obtained in these institutions may have prevented starvation but analysis of the various dietaries provides hard evidence of malnutrition. The nutritional status of the poor who did not have to rely on institutional charity but who resorted to begging and stealing is unlikely to have been any better than that of the people in the workhouse.

These analyses of institutional dietaries of the 1840s have demonstrated first of all that the knowledge of nutritional science in Edinburgh at this time was not sufficiently advanced to prevent serious shortcomings in the diets prescribed. Secondly, they have highlighted dietary deficiencies, particularly of vitamins A and C, that would have caused illness and affected individual response to infectious diseases. The question as to whether these diets can be used as evidence of widespread poor nutrition amongst certain of the population of Edinburgh has to be addressed. The workhouse diet given to workers was the best diet of that institution and yet it was deficient in vital constituents. The full diet of the infirmary was prescribed by physicians and was designed to aid convalescence from disease or trauma but analysis proves that it was lacking in the provision of energy and vitamin A. Christison’s personal convalescent diet was barely adequate with the amount of vitamin C insufficient.

There is no proof that the food eaten in the workhouse was better than that eaten daily by the poorest of the city but the workhouse managers could not afford to be over generous with food, a fact that Wilbraham emphasises in a chapter on diets in nineteenth century institutions, referring to the recommendation of the English Poor Law Commissioners

that 'the paupers should be housed in return for work done but they were by no means disposed to be generous in respect of food...' There is no reason to believe that Scottish workhouse managers were any more liberal since the cost of food was a significant item in the annual budget of an organisation with limited funds and faced with applications for entrance that could not always be accepted. Secondly, over-generous food provision might have encouraged even more requests for admission and unnecessarily prolonged stays in the establishment. The city’s workhouses were under great pressure from the mass of unemployed paupers, as Alison told the Poor Law Commission, explaining that he had spent three days in the summer of 1842 ‘to go through Manchester with the relieving and dispensary officers’ and had concluded that the ‘evidence of redundant population was not so strong there as in Edinburgh or Glasgow.’ Edinburgh’s ‘redundant population’ had to be fed and after 1845 the food supply in the city worsened with the loss of the potato as a consequence of the blight. This nutritional crisis coincided with a fever mortality crisis but it is with the epidemic of nutritional deficiency disease, scurvy, that the next chapter is concerned.

Chapter Four

Scurvy

This chapter is concerned with a number of questions relating to the appearance of scurvy in Edinburgh in 1846. Scurvy, once a common sickness, had become rare since the potato had become the staple carbohydrate, a theory substantiated in a report written in 1847 on the state of the public health which observed: ‘it is certain that scurvy, which was formerly common, has almost disappeared since the potato entered largely into the food of the population.’\(^1\) This development was not confined to Edinburgh and the surrounding country but was reported from many parts of Britain; in 1847, the *Lancet* reported on the prevalence of scurvy ‘both in London and the provinces’ and attributed the outbreak to the absence of the potato.\(^2\) The emergence of the disorder in Edinburgh was therefore not an isolated event but it is important in the context of this thesis since scurvy is the only nutritional deficiency disease that can be identified with certainty in the city at this time. The significance of the occurrence will be analysed and comparisons made with outbreaks of the disorder in other places and at other times. Scurvy was first noticed in Edinburgh in September 1846 and many of the cases are recorded in contemporary hospital records and medical journals in which there are detailed descriptions of the disorder with comments, analysis, speculation as to the cause of the disease and suggested treatment. These sources will be reviewed and their contents compared with earlier theories about scurvy and its management. However it is important first to summarize current ideas about the aetiology and patho-physiology of the condition.

Scurvy is a nutritional deficiency disease caused by prolonged subsistence on a diet practically devoid of fresh fruit and vegetables. Since vitamin C is found almost

\(^1\) Anon, ‘State of the Public Health in the Last Quarter of the Year 1846’, *Journal of the Statistical Society of London*, 10, (1847), p.87.
\(^2\) *The Lancet*, 26 June 1847, pp.676 & 677.
exclusively in foods of plant origin, such a diet does not provide the body with sufficient ascorbic acid (vitamin C) and the lack of this essential constituent disrupts many of the body’s biological systems. The vitamin is important in the function of the immune system and in the normal development of the connective tissues which hold together the cells in all organs of the body. Collagen provides connective tissue with its strength and the failure to synthesize this material causes fragility; the supporting tissue in the blood vessels is weakened and easily damaged, giving rise to the haemorrhages that are diagnostic; bleeding from the gums and spontaneous bruising are frequent early manifestations. There is also a malfunction of the tissue responsible for the growth and repair of bones and bony deformities appear in children deprived of vitamin C. Scurvy is now rare in developed countries but is found occasionally in patients whose malnutrition is the result of alcoholism or drug abuse or in elderly men, living alone existing on a diet of tea and bread. In the past, however, scurvy was so well known that the frequency of its occurrence and the associated mortality were such that it has been described thus: ‘among specific nutritional deficiency diseases, scurvy has ranked with the highest in its toll of human suffering and death.’

The earliest symptoms of sub clinical deficiency in the adult may be relatively non-specific: a feeling of weakness, depression, lassitude, and loss of appetite followed by shortness of breath and aching bones, joints and muscles. These are followed by more specific symptoms: transient subcutaneous haemorrhages, lasting only a day or two and appearing after the most trivial injury. The true nature of the disease is often first recognised by the appearance of the gums which become swollen, inflamed and spongy.

4 Basu and Dickerson, *Vitamins in Human Health and Disease*, p.139.
6 Davidson, Passmore, Brock, Truswell, (eds.), *Human Nutrition and Dietetics*, p.328.
bleeding at the slightest touch, leading to eventual tooth loss. This scurbutic gingivitis is pathognomonic and is the result of impaired collagen synthesis. Later in the disease, haemorrhage into the muscles and joints may produce a hard painful swelling of the leg, known as ‘woody leg’ with discoloration of the overlying skin. Old wounds may reopen, recent ones fail to heal and sudden death can occur when bleeding into vital organs occurs. Infantile scurvy appears usually during months six to twelve with a period of fretfulness, loss of appetite and pallor. Tenderness and swelling of the joints, particularly at the knee and the ankle are early signs and are followed by changes in the bones, most marked at the ends of the ribs. Bleeding may occur at any site and many of the signs found in the adult are also present in the infant.

The disease has been known since the thirteenth century but the complete syndrome became recognised only when long sea voyages of exploration became common, hence 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. It was the frequency with which scurvy appeared at sea that persuaded many observers that there was a specific association between certain factors peculiar to life afloat and the condition. This belief was widespread; for example, Hosack’s System of Practical Nosology, (1821), 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.

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9 Basu and Dickerson, Vitamins in Human Health and Disease, p.139.
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.

The critical dietary lack in scurvy is vitamin C but it has been pointed out that this is unlikely to be the only nutrient deficient in a diet so poor that it produces scurvy. Such diets are likely to lack other nutrients such as iron, folic acid, vitamin A and protein; accordingly, some of the features of the disease are not from lack of vitamin C alone.\(^{14}\)

Long before it became clear that there were certain essential but as yet unknown factors in the diet, the condition was shown to be caused by a diet poor in fresh fruit and vegetables by James Lind (1716–91), the son of an Edinburgh merchant and burgess, a graduate of the university and Royal Naval surgeon, who published his treatise on the disorder during the ten years he was in practice in the city.\(^{15}\) One of the reasons for Lind's interest was the catastrophe that befell Admiral Anson's circumnavigation of the globe in 1740–44. Anson set out with six ships, of which only one returned and over one thousand men are reputed to have died, three hundred from fever and dysentery and over nine hundred from scurvy.\(^{16}\) In his classic account, *A Treatise of the Scurvy*, (1753), Lind demonstrated that scurvy could be cured and, more importantly, it could be prevented.\(^{17}\) In 1747, he carried out what has been described as the world's first controlled clinical trial when he treated twelve sailors with scurvy and showed that oranges and lemons could cure the disease. His account of the experiment, its protocol and the outcome tells of how in May 1747 he took 'twelve patients in the scurvy', placed them in six treatment groups and found 'that the most sudden and visible good effects were perceived from the use of the oranges and lemons; one of those who had taken

\(^{14}\) Davidson, Passmore, Brock, Truswell, (eds.), *Human Nutrition and Dietetics*, p.330.


them, being at the end of six days fit for duty...’\textsuperscript{18} Lind demolished the theory then prevalent that there were two types of scurvy, land scurvy and sea scurvy, citing evidence from physicians who had treated scurvy not only on land but also at sea.

Forty years passed before the Royal Navy adopted the practice of giving sailors daily amounts of lemon or lime juice when Sir Gilbert Blane (1749–1834), personal physician to Lord Rodney, the naval commander, distributed to all captains a\textit{Short Account of the most Effectual Means of Preserving the Health of Seamen} in which he drew heavily on the work of James Lind. The navy was not alone in being slow to implement the new cure, the military authorities in Europe and America alike remained convinced that scurvy was peculiar to life at sea and so for many years did not take steps to prevent its occurrence. As late as 1903, a popular manual of medicine for India warned Europeans that a predisposing cause of scurvy in India was ‘the darkened dwellings in which so many persons exist during half the year.’\textsuperscript{19} The survival of these ideas is to a certain degree understandable; Stewart suggests that the continued prevalence of scurvy in the nineteenth century was in part unavoidable because nobody was aware of the instability of the anti scorbutic factor in citrus juice or of the fact that lemon juice deteriorated with time. These two unwitting errors were important in that they lent credence to the belief that other factors must be involved. Stewart suggests also that it was partly the result of a ‘peculiar obtuseness’ and inability to shake off the old belief that sea air, or salted food or infection was responsible.\textsuperscript{20} As recounted in chapter one, it was in 1932 that Waugh and King, isolated pure vitamin C from lemon juice proving its efficacy and finally confirming the pioneering work of Lind.\textsuperscript{21}

Civilian doctors were also slow to accept Lind’s work, perhaps because there was little interest in scurvy or its treatment in the first half of the nineteenth century. Scurvy had


become uncommon, and many thought that it had been eradicated; medical opinion was divided as to whether conditions at sea had improved to such an extent that the disease no longer appeared or whether the scurvy described by earlier writers was in fact something entirely different.\textsuperscript{22} In many parts of Scotland and Norway, where scurvy had previously been endemic, it had disappeared by 1800, at about the time the potato became an important item in the diet.\textsuperscript{23} Perhaps it is not surprising that the medical profession failed to diagnose scurvy in the 1840s as only the oldest practitioners or those who had served in the Navy had any experience of the disease. Whatever the prevailing medical orthodoxy, the conviction persisted 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 ...'\textsuperscript{24} 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.'\textsuperscript{25}

As described in chapter two, the potato blight in 1845 destroyed most of the crop in those parts of Scotland south of the Highland line but in 1846 the Highlands and Islands were also affected and suffered an almost total loss of the year's crop.\textsuperscript{26} The failure of the potato for two successive years and the consequent rise in the cost of all provisions caused hardship in town and country alike. Mitchell records that the price of oats per Imperial quarter was 20s 7d in 1844, in 1847 the price was 28s 8d, a rise of 40 per cent in two years.\textsuperscript{27} This increase at a time when a dietary staple had become scarce was the underlying cause of the malnutrition which led to scurvy.

\textsuperscript{22} Carpenter, \textit{The History of Scurvy and Vitamin C}, p.98.
\textsuperscript{23} Carpenter, \textit{The History of Scurvy and Vitamin C}, p.101.
\textsuperscript{24} Lind, \textit{A Treatise of the Scurvy}, p.66.
\textsuperscript{25} Davidson, Passmore, Brock, & Truswell, (eds.), \textit{Human Nutrition and Dietetics}, p.328.
The cultivation of the potato in Scotland, which began in the 1720s and was widespread by the end of the century, had largely eradicated the disease from Scotland so that an eminent physician, Professor Christison, could claim that during the thirty years in the Royal Infirmary he had not seen a single case of scurvy until February 1847. He 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 even so lately as last autumn.

Robert Christison (1797–1882), was professor of materia medica at Edinburgh and the extract is from a paper he read to the Medico–Chirurgical Society of Edinburgh in 1847 in which he observed 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.’ He admitted that the first case he had been called to see, that of a master upholsterer, had puzzled him and it was not until he had seen the scurvy epidemic at Perth prison that he was able to make the diagnosis. By the end of the year he had seen two other cases, also ‘in the middle ranks of society’, who had been living for months on bread and meat, coffee and tea with no fresh vegetables, milk or malt-liquor. By May, thirty-five patients with scurvy, thirty two males and three females, had been admitted to the Infirmary, not including railway labourers with the disease. These included a master upholsterer, a blacksmith, two shoemakers, a tailor, a tanner, a porter and a barber, all male. Although Christison took a detailed dietary history from all these patients there is no reference to the absence of potatoes, instead, he concentrated on the lack of milk in their diet. Milk was scarce at this time following an epidemic of pneumonia in milch-cows, an episode mentioned by Christison when considering the effect of “the epidemic constitution” on humans and animals alike. In attributing the

28 Anon, On the Potato, (London, 1795), p.317. This paper appeared originally in communications to the Board of Agriculture and was reprinted in the Report of the Committee appointed by the Board of Agriculture to extract information from the County Reports concerning the Culture and Use of Potatoes, (London, 1795).
increase in illness in the city to the “epidemic constitution”, Christison spoke of the appearance of ‘enteric typhus, or dothinenteritis, in general a rare disease here, now by no means uncommon’ and the gradual increase in ‘ordinary continued fevers,’ but he thought ‘by far the most remarkable circumstance connected with the health of the community has been the appearance of scurvy.’

Carpenter in his analysis of the Edinburgh outbreak, based on Christison’s account, wrote: ‘the physicians admitted that they had at first failed to diagnose the condition correctly, because it was something not previously encountered; some cases were at first classified as purpura.’

Christison explained how the failure of the potato crop, ‘involving dearness of all other provisions,’ affected the diet of the working-classes, causing intestinal disorders to become more common than usual during the winter. From his own observations and those of colleagues, he concluded that scurvy appeared in September or October, 1846, and involved the general population of the city, becoming prevalent among the working classes in March 1847 with the numbers admitted to hospital steadily increasing until May. Christison described in detail the case of a thirty-six year old shoemaker admitted to the infirmary on 17 February 1847. The surviving ward journals do not have this man’s records but those of a similar case in the male clinical medical ward 1, are available. It is of interest that the ward clerk, a senior medical student responsible for maintaining the journal, was H. D. Littlejohn, later Sir Henry Littlejohn, who in 1862 was appointed Edinburgh’s first medical officer of health. The patient whose details Littlejohn recorded was a 50-year-old tailor, Donald Baillie, admitted for treatment on 23 April 1847. Baillie explained that ‘he had been much exposed to privation during the last few months from want of employment and that his food consisted entirely of

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Edinburgh University Special Collections, Lothian Health Services Archive, Journal of Clinical Medical Ward 1, Male, 21 January 1847 to 6 October 1847.
Journal of Ward 1, p.111.
bread and coffee which he had frequently only once a day and never more than twice. He never tasted animal food or milk.' The last remark perhaps denoted his inability to pay for meat or dairy products during this time rather than implying a dietary choice. The Ward Book does not record the treatment or the diet prescribed but analysis of the infirmary full diet, (chapter 3), proved that this diet had sufficient vitamin C to correct the deficiency and he was discharged home cured on 10 May 1847.

There were seventeen men with scurvy in ward 1 at this time, eleven being railway labourers and six local working men.37 Baillie, the tailor, explained that he had been out of work for some time and 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.38 Among the remaining men was another weaver who had 'lately been working on the railway', a collier who was unable to work through injury and had to support 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.39 Of the eleven railway labourers, four were employed on the Hawick line, two on the Caledonian line, three on the Fife line and one at St Boswell's Green. Four of these said that they 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.' The fraud enabled the employer to avoid paying the full wage and forced the employee to buy inferior goods, often at inflated prices. Smith comments that 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.'40 The result was that the system was allowed to continue with little restraint on unscrupulous employers or legal sanctions. The eleven railway workers and the three

37 Journal of Ward 1, p.111.
38 Journal of Ward 1, p.69.
39 Journal of Ward 1, p.74.
Many of the railway labourers with scurvy were Irish but the first railway worker admitted to the infirmary was an Englishman called Buckland who told the clinical clerk that many of his fellow labourers were similarly affected. Scurvy appeared among the railway workers between the middle and end of January, of those one was English, two or three were Scots and the rest were Irish. Christison describes Buckland’s illness and the circumstances leading to it in his paper on scurvy. Buckland was fifty years of age and had previously been a foreman in an iron foundry but began work on the railway in June 1846, being compelled to buy all his provisions at the contractor’s store or “tommy shop” although there were shops in the village close by. He was paid his wages at the end of each month when the cost of purchases at the company store was deducted, leaving only sufficient money for his lodgings and clothing with nothing left to buy food elsewhere. Bread, salt pork, salt butter, cheese, coffee, tea and sugar were available at the store and Buckland told how he had complained repeatedly about their poor quality. Christison expressed some doubt as to whether the quality of the food at the stores was quite as bad as the men suggested but, nevertheless, he was sufficiently impressed by their claims to make representations on their behalf to the legal authorities. As a result of Christison’s disquiet, the county Sheriff ordered an inspection of the store most strongly criticised and this check was repeated at the request of the directors of the railway. No

41 Journal of Ward 1, 10 April 1847 to 25 February 1848.
42 Christison, ‘On Scurvy’ p.5.
43 Journal of Ward 1, p.64.
fault was found with the store or the arrangement and the illegality of the truck system did not seem to have troubled the Sheriff who ordered the inspection.  

In analysing the food eaten by one of the Irish labourers with scurvy, by name Sherry, Christison emphasised that his diet was essentially different to that to which he had eaten in Ireland, inasmuch as he had no milk. Christison was well aware that Sherry no longer had potatoes but does not deduce that this might be responsible for the scurvy. It appears from the passage quoted below as if Christison was manipulating 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 comments:

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.

Christison concluded therefore that lack of milk was the cause, ignoring evidence which suggested otherwise. He cited the parliamentary reports of the English Poor Law Commissioners for 1840 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. Christison thought that several factors were responsible for the scurvy among the railway labourers: the failure of the potato crop, the epidemic among milk cows, the cost of all provisions and the distance from the place of work to their lodgings. However, he remained convinced that milk was a powerful anti scorbutic and that there was a ‘tendency of a saccharo–farinaceous diet to engender scurvy.’ In his lectures on nutrition he was at pains to emphasise what he referred to as ‘an interesting

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45 Journal of Ward 1, in which Felix Sherry, a labourer on the Hawick line is listed, p.79.
point of doctrine, the nutritive proximate principles of the food."\(^{49}\) Many of these terms used by Christison are now meaningless and in the 1840s, in the infancy of nutritional science, they meant little to most chemists, physiologists and medical men alike.

Colleagues from neighbouring districts gave Christison information about patients with scurvy. Dr Brotherston of Alloa had seen twenty-six cases of the disease among ironworkers and labourers and Dr Hope of Polmont had several patients with scurvy. Brotherston found that his patients’ diet had deteriorated and ‘that the food had consisted for some time of bread, oatmeal porridge, and tea, without any meat or milk.’\(^{50}\) Christison used these examples of faulty diet to support his theory that lack of milk and meat were the main causes of the disease, the other reason he thought was ‘a diet too purely farinaceous, saccharo-farinaceous, or saccharo-farinaceous and fatty.’ He then combined his two theories by insisting that the faulty diets could be counteracted by the addition of milk and goes on to compound his errors by criticising the work of Dr Budd, an English physician, who had written “it is probable that anti scorbatic properties are possessed exclusively by substances of vegetable origin.”\(^{51}\) Another English doctor, Baly, had concluded that the potato had anti scorbatic properties when he investigated an epidemic of scurvy among military prisoners in the Milbank Penitentiary in 1840 and 1841. The men had been well on a diet that included potatoes but had developed the disease when potatoes had been withdrawn.\(^{52}\) Christison, while admitting the possibility that potatoes may have prevented scurvy, was equally convinced that animal nitrogenous nutriment had good anti scorbatic properties.

Terms like farinaceous and saccharo–farinaceous, nowadays fairly meaningless, were coming into use in the 1840s as the science of nutrition developed. Jonathan Pereira (1804–1853), the London physician whose treatise on nutrition, cited in chapter three, was the first work in English to analyse institutional diets, listed classes of alimentary

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\(^{49}\) Christison Papers, Lectures on Nutrition, p.154.

\(^{50}\) Christison, ‘On Scurvy,’ p.17.

\(^{51}\) Christison, ‘On Scurvy,’ p.18 quoting Dr Budd who had written on scurvy in the *Library of Medicine*.

\(^{52}\) Christison, ‘On Scurvy,’ p.16 quoting Dr Baly’s paper on the occurrence of scurvy in the *London Medical Gazette*. 
principles including the saccharine and the amylaceous, alternatively called the farinaceous. Christison, at the time one of Scotland’s most distinguished physicians, had become increasingly interested in the subject and included advice on diet in a series of lectures on nutrition at the university. The Monthly Journal of Medical Science, commenting on the ‘epidemic of scurvy in Edinburgh’, intimated that an able historian had been found in Professor Christison who ‘had read an elaborate memoir at the last meeting of the Medico-Chirurgical Society, a memoir abounding in novel views of the aetiology and mode of treatment of the disease.’ Christison in this paper, again expounded his theory of ‘the tendency of a saccharo-farinaceous diet to engender scurvy, 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 concluded by postulating some other cause, which, in conjunction with a faulty diet, had been responsible for the numerous cases of scurvy seen in Edinburgh and its neighbourhood. He believed that having considered the question, ‘no other mode will appear adequate to account for the facts, except the assumption of some particular “epidemic constitution.”’

A meeting of the Medico-Chirurgical Society of Edinburgh in 1847 discussed scurvy in the city; Mr Spence said that he had seen scurvy in several persons in middling ranks of society, while Dr Hughes Bennett reported people with scurvy at the Royal Dispensary and two unnamed physicians had also seen several cases. The scarcity of potatoes as a result of the blight was very much in their minds; Bennett had been able ‘to trace the

54 In 1847 Christison was President of the Royal College of Physicians of Edinburgh, Professor of Materia Medica in the University of Edinburgh, Vice President of the Royal Society of Edinburgh and Physician to the Queen in Scotland.
56 Christison, ‘On Scurvy,’ p.16.
disease in every case to a want of fresh vegetables, more especially potatoes, or to an insufficient diet.' The confusion is plain when Bennett’s opinion is compared with that of Paterson and Spence, both of whom were certain that the scarcity of potatoes had nothing to do with the appearance of the disorder. The fourth doctor was convinced that the lack of ‘wholesome animal nourishment’ was the problem. Despite Professor Christison’s position and reputation few of his colleagues appear to have been convinced by his “novel views”. Dr James Stark in his report on the mortality of Edinburgh for 1847 wrote:

this scurbutic tendency was not confined to the poor alone though it was only among them that the severer forms of scurvy were remarked but was observable to a small extent in all classes, among many of whom it could not be traced to any deficiency of proper nutritious food, unless it arose from the simple want of the potato, but appeared alone attributable to epidemic influence.

These reports indicate that there were many who had scurvy and recovered, but the small number of deaths is surprising and poses several questions. If the labouring classes and the poor were so dependent on the potato it might have been expected that the loss of their main source of vitamin C would have resulted in many more cases of scurvy and an associated greater mortality than seems to have been the case. In his 1847 report Stark lists seven deaths from purpura and scurvy but in his analysis of the year does not link malnutrition and increased disease. He attributes the increased mortality from all causes and the failure of the crops to a common cause, citing the work of Noah Webster who described what he saw as the connections between increased mortality, pestilential periods, bad seasons, failure of the crops, cattle disease and earthquakes.

The potato crop failure was the cause of the scurvy epidemic and there is a ready explanation for its early and frequent appearance among the Irish labourers working on railway construction. The rapidity with which symptoms of scurvy appeared in this group has been explained by research which demonstrated that people accustomed to very high levels of vitamin C, when deprived of the source of the vitamin, lose their stores much more rapidly than those accustomed to lower levels and as a result they develop quickly the symptoms and signs of scurvy. The potato is not rich in vitamin C, but the huge daily consumption by Irish workers in particular provided far in excess of the daily requirement of thirty to sixty milligrams per day. The loss of the potato removed their main source of the vitamin and during the winter and early spring when no alternative was available they developed scurvy.60

Christison and many of his colleagues in Edinburgh were unable to abandon old ideas. In his lectures he declared that in the 1840s, scurvy, the terrible scourge of fleets, armies, and society at large, was favoured by confinement, want of exercise and by a moist, cold atmosphere. He considered that the main cause was a combination of damp air and inactivity, but that faulty food, particularly lack of milk, did play a part in the condition also.61 Christison was not the only physician in Scotland writing about the sudden, and at the time inexplicable, appearance of scurvy. Dr Ritchie, a physician at the Glasgow Royal Infirmary, published a series of articles in May 1847 on '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.' There had been admitted to the medical wards of the infirmary, by the end of May, eighty-three cases, seventy being male and thirteen, female. At the Dispensary, thirty-nine cases, thirty-three males and six females had been treated and there were other instances of the condition, which 'some may be disposed to call purpura'. In Glasgow at that time purpura was common but it is probable that the correct diagnosis was scurvy,

61 Edinburgh University Special Collections, Christison Papers, Dietetics II, Faulty Food and its Effects, p.110.
perhaps still at an early stage.\textsuperscript{62} Ritchie reviewed all the cases of purpura treated from November 1846 and concluded that several patients diagnosed as purpura were in fact suffering from scurvy. As in Edinburgh, it was not until February 1847 that the actual extent of the outbreak became clear. Ritchie described the diets of the unfortunate sufferers in considerable detail and concluded that 'the general fact in regard to the food of all was, \textit{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, \textit{the patients had been exposed for months to a total deprivation of fresh succulent vegetables.' [Original italics].\textsuperscript{63} Dr Ritchie, in the second of his papers on scurvy, wrote that:

It is familiar to every one 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. From the same cause the value of money rose in the same period little short of a half; and in consequence, the workmen in every branch of home trade were thrown more or less out of employment...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.\textsuperscript{64}

He repeated his belief that the lack of proper vegetable food was the cause and the rest of the paper was taken up by analysing the various types of treatment prescribed, including 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.\textsuperscript{65}

Another detailed analysis of the Glasgow outbreak was that of Dr John Steele, Surgeon and Superintendent of the Royal Infirmary in which he commented on the increased mortality in the city due 'to the extremely debilitated condition of a large proportion of the cases under treatment.' Furthermore, it was his opinion that 'the want of

\textsuperscript{62}C.Ritchie, 'Contributions to the Pathology and Treatment of the Scorbatus, which is at present prevalent in various parts of Scotland', \textit{Monthly Journal of Medical Science}, 8, (1847), pp.38–49 and 76–87.
\textsuperscript{63}Ritchie, 'Contributions to the Pathology and Treatment of the Scorbatus', p.41.
\textsuperscript{64}Ritchie, 'Contributions to the Pathology and Treatment of the Scorbatus', pp.77 & 78.
\textsuperscript{65}Ritchie, 'Contributions to the Pathology and Treatment of the Scorbatus', pp.84 & 85.
employment and the high price of provisions, to which the lower orders were subjected during the bygone year, had a corresponding effect in increasing their liability to disease.' These 'irregularities' were also the primary cause of the outbreak of scurbutus which 'appeared almost simultaneously in an endemic form in various parts of Scotland.' The number treated in the infirmary was one hundred and one, eighty-two males and nineteen females. Of the males, twenty-seven were railway labourers, twelve weavers, five shoemakers, five quarriers, four in factory employment, three sailors and the remaining twenty-five 'pursued miscellaneous mechanical pursuits.' Of the females, sixteen worked in factories and the remaining six 'followed sedentary occupations.' There was only one fatality, an elderly male and the average length of stay in hospital was twenty-seven days—a figure that does not compare well with the six days described by Lind.66

Henry Lonsdale, 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 Carlisle area they were mostly weavers and their families, whereas in the most southern part of the county of Dumfries agricultural workers were the group most affected after January 1847. Lonsdale confirmed, with the help of Mr Carruthers, surgeon, of Dumfries, that potatoes had always been a staple article of food for these farm workers. '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 records a conversation with Dr Browne of the Crichton Institution, Dumfries, who said that every spring he had scorbatic cases and that now (1847) the disease was more common. The consensus among these practitioners was that the lack of potatoes was the chief cause of

66 J.C.Steele, 'View of the Sickness and Mortality in the Royal Infirmary of Glasgow during the Year 1847: illustrated by Tables of different Diseases', *Edinburgh Medical and Surgical Journal*, 70, (1848), pp.149–169.
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 (July 9th).’ 67

These three contributors of papers on scurvy to the *Monthly Journal of Medical Science* recorded approximately 400 cases known to them in just three parts of Scotland — Edinburgh, Glasgow and Dumfriesshire. It is probable that these were not the only cases in Scotland, since it would be surprising if the only areas in which people suffered from scurvy were the rural southwest, the railway developments and the cities of Glasgow and Edinburgh.

There are several questions which remain unanswered. First, it is surprising that more cases were not recorded in Edinburgh. There are several possible explanations; perhaps the poorest were reluctant to visit the free dispensaries so that their scurvy remained undiagnosed and unrecorded; possibly there remained amongst people recently come from the country a memory of how to treat scurvy using readily available plants. In the cities and in the encampments of railway workers it is less likely that such knowledge existed and in urban areas even if people knew of plants with anti scorbutic properties obtaining them must have been difficult. Scurvy grass, *cochlearia officinalis*, which is found in dry salt marshes and banks between April and August, was recognised as a good source of vitamin C, yielding 200 mg. from 100 gm. of leaves and buds, comparing well with the 45–60mg obtainable in 100 gm. of cabbage. 68 Scurvy grass was certainly known to the people of the Highlands being called in Gaelic by several names, *am maraiche*, the seasider or *an carran*, the thing for scurvy. 69 Mary Beith says that this plant was used as an anti scorbutic well into the nineteenth century and was cultivated for this reason and that the common dock, *Rumex obtusifolius*, taken as a decoction, was

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good for scurvy. Scurvy grass and the common dock were not the only anti scorbutic plants growing in the Scottish countryside, many of which may have been known to people not long removed from the country to the city. Lightfoot, who accompanied Thomas Pennant on his tour of Scotland in 1772 at the latter’s request, wrote *Flora Scotica*, which he described as ‘a systematic arrangement in the Linnaean method of the native plants of Scotland and the Hebrides.’ In this detailed botanical record there are numerous descriptions of plants which are ‘esteemed as an antiscorbutic’; among these he mentions pepperwort, common scurvy grass, wood sorrel, roebuck berries, common brooklime, sea bindweed and tormentil. There are many other plants which he says were used by the natives as greens, in salad, or boiled, for example, sea beet whose ‘young leaves boiled are wholesome and good greens.’ The frequent references to the use of these common plants as greens in springtime suggests that at the time of the year, early spring, when the first symptoms of scurvy began to show (at least before the potato came into general use) people knew of readily available plants which could be used as an anti scorbutic.

What is also striking is the preponderance of males in the reports from both Glasgow and Edinburgh. This may be a reflection of the habit of allowing the male provider to have a larger share of whatever food was available, implying a greater consumption of potatoes and therefore a greater fall in vitamin C levels when the potato was no longer available. Alternatively, women made up their diet by consuming more green vegetables which may have been looked on with disfavour by the men. There is no contemporary evidence to support these theories but the presence of scurvy confirms the existence of specific nutritional deficiency, more common amongst men, in Edinburgh during 1846–47. In chapter three the results of computer analysis of the Edinburgh Charity Workhouse workers’ diet and Professor Christison’s own convalescent diet were

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71 J. Lightfoot, *Flora Scotica*, (London, 1792). The introduction including a pen portrait of the author was written by Thomas Pennant and is dated 1788.
described, revealing the inadequate amounts of vitamin C in both diets, findings that bear out the critical importance of the potato as an anti scorbutic.73

This outline of the scurvy outbreak in the city has depended largely on contemporary published sources together with certain items from the Christison papers. According to Stark, there were seven deaths from purpura and scurvy in 1847, the only published statistical record of scurvy mortality in Edinburgh. There were papers on the topic in the learned journals with discussion of numbers affected but no effort was made to investigate the localities in which the sufferers lived, to record their habits or to estimate the mortality rate from the disease. This lack of interest in statistical information is unusual at a time when the gathering of statistical information had developed from its beginnings in the late eighteenth century when Sir John Sinclair had instituted the collecting of facts and figures for his Statistical Account of Scotland, (1791–99). The next chapter will therefore examine the history of mortality bills and will focus on the absence of statistical records of morbidity and mortality in Edinburgh during the 1840s, contrasting the city’s indifference with the attention paid to such matters as mortality bills elsewhere.

Chapter Five

Statistics

This chapter, although primarily concerned with statistics and the development of this new science, also raises a number of questions about the lack of an official record of mortality for Edinburgh during most of the first half of the nineteenth century. This deficiency is striking. Edinburgh was a capital city with a university and medical school, two medical Royal Colleges and a Royal Society but the Town Council, the Police Commission and the medical profession alike largely ignored statistics. Edinburgh was one of the few cities in the United Kingdom with no official mortality figures and, apparently, no interest in their collection until 1846. It is perhaps not entirely unexpected that the municipal authorities should not be aware of the usefulness of mortality bills but that the medical profession remained indifferent is remarkable and worthy of scrutiny. In Britain during the 1830s statistical investigations were seen as a means towards the alleviation of many of the ills affecting society but not, it appears, in Edinburgh. In order to investigate this omission and to compare the situation in Edinburgh with that in other cities, the development of statistics and of statutory registration in the United Kingdom will be reviewed and the history of mortality bills examined. The lack of interest in mortality figures was equalled only by the disregard for records of morbidity, the outcome being that the registers maintained by dispensaries and hospitals for annual publication to encourage charitable giving are the only source of information on morbidity during the 1840s and what survives is not comprehensive. The aim in this chapter will be to link the failure to pass legislation for Scottish civil registration with the evident lack of interest in statistics in Edinburgh, a capital city which retained considerable legal and ecclesiastical power and substantial influence.

The absence of any record of sickness and incapacity throughout the 1840s, the lack of mortality bills between 1840 and 1845 and the failure to implement national statutory registration of death in Scotland make a quantitative analysis of disease in Edinburgh at
this period impossible but examination of the records that survive does permit certain observations to be made. The diagnostic term for the cause of death recorded at the cemetery can be evaluated and this exercise in its turn may provide evidence as to the accuracy of the statistics collected when eventually mortality bills for the city were sanctioned. Burial records were the only sources from which figures could be obtained but the recorded cause of death was not always accurate or informative, factors which do not permit a quantitative review but examination of the burial records can provide information regarding nosological usage, age at death and the incidence of infectious disease.

Glass in his book, *Numbering the People*, an examination of population, census development and vital statistics, suggests that an important factor in the acceptance of civil registration in England was 'the interest of the medical profession and of a number of statisticians in the use of existing data and in the provision of better data on mortality.' The term 'medical statistics' was relatively new in the first half of the nineteenth century; the first book in English on the subject, *Elements of Medical Statistics*, an expanded version of his Gulstonian lectures at the Royal College of Physicians of London, was written by F.Bisset Hawkins, a London physician, in 1829. Hawkins defined medical statistics as 'the application of numbers to illustrate the natural history of man in health and disease.' and in his review he included the mortality bills of London. He says that these were first recorded in 1603 at a time of plague and were published weekly with occasional interruptions and variations into the nineteenth century. Hawkins believed that the bills and registers were of value and did 'afford some approximation to the truth' but he accepted that there was need for improvement in recording the cause of death. Cullen in his work on the statistical movement in Britain states that the London bills of mortality first appeared in 1519 as an early warning of plague and intermittently until 1603 with other causes of death added after 1629.

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In Scotland there was much less interest in bills of mortality, apart from that shown by James Cleland of Glasgow whose historical account of bills of mortality (1836) dealt with the subject at length. Cleland gives an account of the Glasgow bills, which he collected and for which he was responsible, publishing them first in 1823, and continuing the work through the 1830s. Hawkins said that these were 'more accurate, probably, than any others relating to our population, from the zeal which the authorities of Glasgow displayed in collecting them.' Hawkins commented that as far as the rest of Scotland was concerned 'from the acknowledged omissions in the registers of deaths in most part of Scotland, few just inferences can be drawn.'

There is less known about the history of Edinburgh's bills of mortality. Stark in his first report on Edinburgh's mortality in 1846 wrote: 'it is not a little strange that in the matter of mortality bills we have fallen greatly behind our predecessors of the last century' claiming that they were certainly in operation in 1739 and that monthly tables were published in the Scots Magazine until 1794.

Cleland was in the vanguard in Scotland in his interest in vital statistics. There were earlier works on statistics but these dealt with a variety of topics as, for example, the detailed account of Scottish parishes collected by parish ministers for Sir John Sinclair between 1791 and 1799. Cleland and his fellow enthusiasts for statistics were more interested in numbers; numbers born, numbers marrying, the size of families and age at death. The vital statisticians or 'statists' as they were called originally, belonged to an increasingly large group interested in the new science. By the 1830s, Government was taking an interest and in 1832 the Statistical Department of the Board of Trade was established to collect information on trade and manufacturing; in 1836, the act for the registration of births, marriages and deaths came into force in England and Wales. A separate bill for Scotland did not reach the statute book and subsequent efforts were

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equally unsuccessful until 1855 when civil registration in Scotland became a reality. The reasons for the failure are many and complex and merit further detailed study.

It is surprising that at this time no interest was shown in Edinburgh in any kind of statistical record when statistical information on a variety of subjects was being gathered enthusiastically and the new science was gaining adherents in Britain and in Europe. The 1830s in Britain saw the development of statistics as a tool of government departments but these were not the only bodies with an interest in the subject; the British Association for the Advancement of Science, founded in 1831, had a section of Statistics from 1833, until in 1856 it was renamed the section of Economics and Statistics. The Cambridge meeting of 1833, attended by such luminaries as Adolphe Quetelet, the Belgian astronomer and statistician whose work on the statistics of crime and of suicide were well known and Thomas Malthus, the English clergyman and author of *An Essay on the Principle of Population* (1798), was presented with a fait accompli by Charles Babbage, the mathematician and inventor of calculating machines, when he instigated the formation of a statistical section. The Association did not receive the new section with unqualified approval but its formation was ‘the signal for a rash of statistical societies’ of which the earliest was the Manchester Statistical Society founded in 1833, followed by the Statistical Society of London in 1834. The interest in and importance of social science is evident in the prospectus of the London society which says that it was established ‘for the purposes of procuring, arranging, and publishing “Facts to illustrate the Condition and Prospects of Society.”’ The society resolved that there were four great classes of statistics, namely, economical, political, medical, moral and intellectual and recognising the importance of accurate figures for births and deaths, a Vital Statistics committee was formed in 1838; two years later a committee was appointed to consider how to extend the registration of births, marriages and deaths to Scotland. In 1841 a

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hospital statistics committee was set up and issued forms to all the hospitals in London 'for the registration and annual collection of their experience.'  

In a short space of time societies were started in Aberdeen, Belfast, Leeds, Liverpool, Bristol, Nottingham, Newcastle and Birmingham. In 1836 two were formed in Glasgow, the Statistical Society of Glasgow and the Glasgow and Clydesdale Statistical Society and Cullen comments that 'the earliest significant society was that set up in Glasgow. Or, more correctly, the societies set up in Glasgow.' The former was largely the brainchild of Charles Baird and Robert Cowan; the first president of the Glasgow and Clydesdale was James Cleland and included in the list of ordinary councillors was Cowan who was therefore involved in both societies.  

Cowan was a young Glasgow physician who after graduating in 1834 was professor of Medical police and forensic medicine from 1839 until his death in 1841, writing on fever in the city from his experience as a physician in the fever hospital.

The eagerness with which towns and cities across the kingdom formed statistical societies is understandable. It was a time of reform, of committees of inquiry and of parliamentary reports and it is surprising therefore that Edinburgh showed no interest in forming a statistical society, particularly since the meeting of the British Association following that in Cambridge, was in Edinburgh in 1834. A reference by the council of the London society to the possible formation of a society in Edinburgh suggests that there was at least some interest in the subject. Unfortunately, the society’s minutes and letter books have no record of correspondence on the matter; the only reference to Edinburgh is in a brief exchange of letters concerning the proceedings of the London

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12 M.J.Cullen, The Statistical Movement in Early Victorian Britain, (New York), pp.77, 105 & 119; Anon, Constitution and Regulations of the Glasgow and Clydesdale Statistical Society, (Glasgow, 1837), p.1. It is probable that Cleland was the author of this impressively bound volume which was presented to the Royal College of Physicians of Edinburgh. C.R.Baird and A.Fullarton wrote jointly Remarks on the Evils at Present Affecting the Highlands and Islands of Scotland, (Glasgow, 1838) and both were members of famine relief committees in 1836–37 and again in 1847.  
13 Morrell and Thackray, Gentlemen of Science, p.548.  
14 Cullen, The Statistical Movement in Early Victorian Britain, p.119.
society between the secretary and Charles Oliphant, a retired Edinburgh lawyer, a fellow of the London society but who was not prominent in Edinburgh intellectual circles.\footnote{15 Minutes and Letter Books of the Statistical Society of London, 11 June 1836 & 11 January 1837; Post Office Annual Directory 1831–2, (Edinburgh, 1832).}

Alison used data from mortality bills, the New Town Dispensary and the Royal Infirmary in his writings on fever and in his campaign for a review of the Poor Law. In his paper on the fever of 1837–39 he referred to the number of men employed on charity work in the Meadows, a public park in the centre of the city, and the numbers of fever patients in the infirmary to show that they were related inversely.\footnote{16 W.P.Alison, \textit{Observations on the Epidemic Fever of 1843 in Scotland and its Connection with the Destitute Condition of the Poor}, (Edinburgh, 1844), p.6.} The Edinburgh Fever Board of which Alison was a member published a pamphlet in 1844 in which fever statistics from Edinburgh and other cities were used to illustrate the threat the city faced.\footnote{17 R.Deuchar, \textit{Observations on the Prevalence of Epidemic Fever in Edinburgh and Glasgow}, (Edinburgh, 1844).} The Royal College of Physicians of Edinburgh convened committees in 1840 and 1846 ‘to consider and recommend an effective and uniform system of Registration in Scotland of all Births and Deaths’. The 1840 decision to press for a registration was made by the college in response to a resolution passed at the Glasgow meeting of the British Association in the same year.

An important element in the early success of statistical movement was the drive for the registration of births, marriages and deaths, an objective that was rapidly achieved in England and Wales. There may have been particular reasons for this success, not least the attitude of English dissenters arising from the requirement for all persons to have births, marriages and deaths recorded in the established church, the Church of England, whose jurisdiction and validity they did not recognise. Their objections were significant factors in the successful introduction of a Registration Bill to parliament, the bill’s sponsors hoping to win the support of the English and Welsh dissenting factions in forthcoming elections. The bill for England and Wales was passed and became law in 1837, but a law for Scotland was not approved until 1855; Bills did reach the committee
stage in 1834, 1835, 1837 and again in 1847 but for one reason or another none of them progressed further.¹⁸

Before considering these aspects of statistics the next section will concentrate on the surviving parochial records of burials in Edinburgh. It is not intended to carry out a numerical analysis of these records but to record those that exist and to outline their contents. Prior to the Registration of Births, Deaths and Marriages (Scotland) Act, 1854, which took effect from 1 January 1855, parish registers contained the only formal records of these events. Death records, such as existed, were in effect records of burial and varied greatly in the quality and accuracy of the information, depending on the abilities and the literacy of the recorder. This duty was frequently handed over in country districts to the gravedigger whose literacy was often faulty or non-existent. In Edinburgh, where the number of burials was always considerable and therefore worth money to the kirk, it was important to have a recorder who was both numerate and literate. The surviving burial records from the city's graveyards are generally legible although the detail is very variable.

The system had not always been so haphazard. In the seventeenth century, every birth, marriage and death had to be entered in the parish register, kept by the established church, but it is not surprising that many events went unrecorded. In 1783 the imposition of stamp duty on registration of deaths resulted in a sharp drop in the number of burials recorded, including almost all dissenter burials and even many who belonged to the established church. Glass argues that the effects of the Act may not have been uniform throughout the United Kingdom and quotes evidence from an analysis of Shropshire

¹⁸ General Index to Bills 1801–1852 and Hansard's Parliamentary Debates 1829–1891 record the Bills and the debates which took place. The first was Bills Public 1834 (III), continuing with Bills Public 1835 (IV), Bills Public 1837 (I), Bills Public 1847 (III), Bills Public 1847–48 (VI), Bills Public (VI), Bills Public 1849 (VI). The changes in detail in these Bills give some indication as to the source of the objections that prevented the legislation from passing through Parliament. The Bill of 1835 was debated briefly in the Commons and that of 1847, which concerned only births and marriages, had both debating time allowed and reached the committee stage.
registers which show that no deterioration occurred.\(^{19}\) Scottish parish registers have not been studied but it is likely that there was a decrease in registration. The tax was rescinded in 1794 but the habit of registration was lost and many did not return to recording these events. Flinn points out that the Church Disruption of 1843 was also likely to have resulted in considerable harm to the process of parochial registration as had happened with eighteenth church schisms.\(^{20}\) Surviving burial records, therefore, are almost certainly incomplete and cannot be relied upon to give an accurate picture of mortality rates.

Details of burials gleaned from parish registers were used to produce bills of mortality in England from 1592, when, at a time of plague, bills were produced regularly in London. Originally these bills did not list the cause of death, they were simply a record of numbers, designed to inform the public and warn the authorities of the approach and severity of an epidemic. In Scotland the importance of maintaining such a register was first acknowledged when the Edinburgh commissioners to the General Assembly of the Church of Scotland in 1565 asked that every parish record all deaths but despite this early interest Scottish parish records were far from being uniform or complete. In England parish death records varied in quality but were good enough to be used as the basis of the first statistical work on the subject of mortality by John Graunt.\(^{21}\) Glasgow kept a register of deaths from 1670 but the earliest detailed figures are from 1699 and Edinburgh Town Council in 1695 agreed to have “a bill of mortality printed from time to time” but as Flinn points out those that survive are very variable in the years and parishes covered.\(^{22}\)

The only complete set of Edinburgh burial ground records that survive for the period 1840–1850 are those of St Cuthbert’s churchyard. These are essentially accounting


\(^{21}\) Cleland, *A Historical Account of Bills of Mortality*, p.3.

\(^{22}\) Flinn, *Scottish Population History*, p.74.
records, the register carrying the superscription "Cash Book", in which are detailed the charges incurred from the hire of the mortcloth, the ribbons and the spokes, the cost of digging the grave (to a greater depth if required), the hiring of beadles, ushers, batonmen and bearers, watching, money for the poor, warrants and the recorders' dues. The register records the name of the deceased, the address, occupation (and occupation of husband or father if the deceased was a married woman or a child) and date of death. The cause of death and the type of funeral, whether by hearse, spokes, shoulders or arms are both recorded in the same column, confirming the assumption that the book is not a record of mortality but of burials; the cause of death was not the most important fact recorded and was secondary to details concerning the type of funeral, and the number of men employed, all essential for accounting purposes. The stated cause of death before 1846, the year when Stark began to collect his statistics, supplying recorders with official forms, must be viewed with caution and should not be taken as an accurate record of the causes of mortality during this period. It has to be borne in mind also that for many years after statutory registration in England and in Scotland, diagnoses of cause of death were equally suspect as to their accuracy.

The geographical location of the homes of those buried in St Cuthbert's graveyard might have been expected to yield some useful epidemiological information but there are too few burials and the distribution of addresses is so widespread, including Newhaven, Stockbridge, Grassmarket, New Town, Cowgate, High Street and the village of Water of Leith (now the Dean Village), that it is not possible to distinguish any pattern of disease spread. The cost of the mortcloth might yield information as to the social status of the deceased, the inference being that those buried without a mortcloth were from the very poorest of society. Mortcloth charges ranged from nine pence to one guinea with fifteen intermediate prices. The percentage buried without a mortcloth between 1841 and 1849 is shown in table 5.1, decreasing from a maximum of 49 per cent in 1841 to a low of 25 per cent in 1848 indicating that a smaller percentage of the very poorest were

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24 St Cuthbert's Churchyard Cash Book, 1840–46.
being buried in St Cuthbert’s towards the end of the decade. The explanation may be that new cemeteries buried the poor, by arrangement with the council to reassure the public who were increasingly troubled by what they saw as the risks to health from burials near residential areas.

<table>
<thead>
<tr>
<th>Year</th>
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<td>1848</td>
<td>25%</td>
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<tr>
<td>1849</td>
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</tbody>
</table>

Table 5.1. Burials without Mortcloth, St Cuthbert’s 1841–1849.25

During the 1840s several new burial grounds were opened on the outskirts of the city, partly because an expanding city required more cemeteries and partly because of fears that burying in the centre of cities was dangerous and might be the source of fever. Cemeteries were opened at Warriston in 1843, Dean, Dalry, Grange, Newington, North Merchiston and Rosebank cemeteries opened in 1846.26 Burials continued in the old cemeteries of Canongate, Greyfriars’, St Cuthbert’s, St John’s, and Buccleuch (incorporating East Preston Street) but the householders in the vicinity were frequently concerned at some of the burial practices they witnessed. In 1844, at the height of the fever epidemic, the householders of Candlemaker Row, whose houses formed the boundary wall of the cemetery, complained to the council that three or four bodies were put in one grave which was then left open for days to wait for more bodies, a practice

26 Details of cemeteries were provided by the Recorder of District Burial Grounds and Crematorium, Environmental and Consumer Services, City of Edinburgh Council.
they felt was ‘in no small degree endangering the neighbourhood.’ The council investigated and found that the bodies in question were from the university department of anatomy. It was agreed that to prevent this abuse bodies after anatomical dissection would be buried on Tuesdays and Fridays and that graves would be closed on the days they were opened. The problem was not insignificant for the number of bodies from the anatomy class was considerable amounting to 117 in 1841. There was widespread concern about the supposed harmful effects to health from graveyards, a concern that increased when in 1843 Edwin Chadwick published a supplement to his sanitary report in which he criticised the consequences to health from the overloading of urban burial grounds. The uncertainty about the risks to health from burial practices was not confined to laymen, in 1848 a committee of the Royal College of Physicians of Edinburgh ‘appointed to consider any bills that may be brought into Parliament for the Improvement of the Health of Towns and the Applicability of such Measures to Scotland’ was critical of the clause which permitted burial if thirty inches of soil was left above the coffin. They were convinced that interment in towns, and especially in crowded parts of any town, should be prohibited. But, in the event of this object being unattainable at present, they consider that no adult body should be allowed to be buried, in cemeteries within towns, nearer to the surface of the earth than four feet. They are also of the opinion, that such graves should not be disturbed for a period of about ten years; for which purpose it is desirable that a space not less than two feet should be left between the graves.[Original Italics]

Records of burials in other graveyards are available but they do not cover the whole of the period under review. The burial register of Greyfriars’ churchyard for 1835–1842, the Greyfriars’ Account of Mortcloth, 1843–1847 and the Greyfriars’ List of Mortcloth Dues, 1848–1863 are in the City Archive, the latter two volumes providing a list of burials at which a mortcloth was hired. The register of burials, 1835–1842, records the

28 Edinburgh City Archive, Greyfriars’ Burial Register, 1835–1842.
30 Minutes of the Royal College of Physicians of Edinburgh, 3 November 1840, pp.3329 & 3330.
name, age, address and cause of death and from May 1841 until December 1842 a monthly summary, described as a Mortality Bill, is attached.31 Burial records kept by other Edinburgh graveyards have survived also but none are complete for the period being studied; of the six new burial grounds opened in the mid 1840s the Dean Cemetery and Warriston Cemetery have records of burials from 1846. The records of St Cuthbert’s graveyard have been studied in detail but a quantitative analysis has not been carried out because the burials form an unknown proportion of those taking place in the city. Secondly, the population in which the deaths occurred cannot be analysed with confidence, although information regarding social class, geographical origin or economic status can be inferred.

If mortality bills gave only an ‘approximation of the truth’, as Hawkins suggested, they were still of value, a fact that was ignored by the municipal authorities in Edinburgh until in 1841 they were persuaded to consider establishing a system of recording deaths in the city. The magistrates received a letter from Alexander Watt of Glasgow explaining that he understood the magistrates of Edinburgh were keen to have mortality bills drawn up for the city but ‘nothing had been done through lack of interest.’32 He added ‘I could very easily get introductory letters from the most influential Gentlemen in this quarter (who take a deep interest in this subject) to their friends in Edinburgh to induce them to give their assistance to enable me to complete these tables.’ Watt offered to complete the tables if the council would pay his expenses, an offer the council were prepared to accept as long as these did not exceed twenty pounds per annum. The council, apparently having been reminded of the value of such data and perhaps hoping to avoid expenditure, at the same meeting resolved to petition parliament to implement civil registration in Scotland declaring

that a knowledge of the Laws of Mortality is in 1841 intimately connected with the social condition of the people; that hitherto the knowledge has been based on imperfect data ... so much so that in the solution of some of the most important questions affecting the sanatory condition of towns, the

31 Greyfriars’ Burial Register 1835–42.
philanthropist and scientific enquirer, have to depend on the speculative views of men than on the knowledge of incontrovertible facts.\footnote{Edinburgh Council Records, 234, 9 March 1841.}

Despite such fine sentiments nothing more is heard of Watt or indeed of mortality bills, neither being mentioned again in the minutes until Dr James Stark agreed in 1846 to collect death statistics ‘gratuitously’. The council, although convinced of the value of mortality bills, was prepared to wait until a Scottish civil registration measure was passed in parliament but a bill for Scotland failed to appear on the statute books and Edinburgh’s initial enthusiasm for statistics, both local and national, evaporated. In 1849 the council voted against the parliamentary bill to extend registration to Scotland, petitioning the House of Commons that the new measures would be too expensive and a ‘serious abridgement of civil liberty… [it was] an unwarrantable encroachment on the liberties of the people—as creating crime in order to punish it.’ The machinery was too expensive and complicated and would create ‘a vast number of place holders’, thereby increasing the patronage and power of the Government and ‘superseding by a system of centralisation all control over local expenditure.’ The council complained that in Scotland within the last few years there had been put in place Prison Boards, Parochial Boards and a Board of Supervision of the Poor, all with salaried officers, offices and staff.\footnote{Edinburgh Council Records, 251, 24 April 1849.} The council was not the only body in Scotland objecting to statutory registration; the General Assembly of the Church of Scotland in 1848 debated the Marriage and Registration Bills and strongly disapproved of the measure on marriage, found clauses in the Registration Bill ‘of an objectionable character’ and decided to petition the House of Lords to prevent the Bills in their present shape.\footnote{Edinburgh University New College Library, The Principal Acts of the General Assembly of the Church of Scotland 1848, (Edinburgh, 1848), p.55.}

There is no evidence that Watt collected mortality statistics for the city other than those he produced for the British Association and there is no explanation in the council minutes for this outcome. The apathy of the council is understandable since any scheme
was going to cost money and health was not a matter for which the council had any responsibility. The lack of interest shown by the medical faculty and the medical colleges is more difficult to explain and will be discussed later.

Watt may not have been employed by Edinburgh but he was asked by the Statistical Section of the British Association in 1840 to draw up vital statistics for large towns in Scotland.\(^{36}\) In its introduction the committee, whose membership included Col.W.H. Sykes, G.R.Porter, Edwin Chadwick and William Alison, recorded their debt to ‘Mr Alexander Watt of Glasgow who has established a claim to public respect by his Mortality Bills of Glasgow and other statistical works, was good enough to undertake the severe labour of accumulating the facts which constitute the present report.’ Watt produced a report of 199 pages containing 119 tables packed with facts, but before publication the committee reduced the number of pages to eighty seven.\(^{37}\) In the section dealing with Edinburgh he gave the 1841 population of the city including Leith as 166,554 and the total number of deaths as 4,154. Watt explained that he obtained the mortality figures from the recorders at the city’s fifteen burial grounds and was highly critical of ‘the want of a proper system for the registration of deaths’ in Edinburgh and throughout Scotland.\(^{38}\) Whilst he accepted that in several towns and cities the magistrates and kirk sessions had made strenuous efforts to procure registers of burials, ‘no uniform or systematic method of recording the causes of death has been adopted.’

Reviewing the various methods adopted by Edinburgh parishes in recording burials he was complimentary only about the parish of South Leith, ‘where Mr Lyon has introduced the system recommended by the Committee of the Royal College of Physicians of Edinburgh, appointed to consider the best mode of framing public registers of deaths.’\(^{39}\) Watt provided an abstract of the total Edinburgh mortality for the years


\(^{38}\) Report on the Vital Statistics of Large Towns in Scotland, p.147. Watt listed the burial grounds in Edinburgh: in the City District: Greyfriars', St John’s Episcopal Chapel; in St Cuthbert’s: St Cuthbert’s, Calton New and Old, Newington, Buccleuch, Society of Friends, Jewish; Canongate: Canongate, Chapel Royal, Holyrood; North Leith: North Leith, Newhaven; South Leith: South Leith, Restalrig.

1836–1841 inclusive, making it clear that the burials were in the city of Edinburgh and
the suburban districts of St Cuthbert’s and the Canongate. The population of the city he
estimated as 137,127 in 1836, 138,183 in 1841 and the number of burials recorded in
these years is shown in table 5.2.

<table>
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<tr>
<td>1841</td>
<td>3507</td>
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</tbody>
</table>

Table 5.2. Edinburgh’s Mortality 1836–1841 from *Report on the Vital Statistics of
Large Towns in Scotland*.

When finally in 1846 the Town Council agreed to publish mortality bills for the city and
for Leith, the decision was made in response to a letter written to the Lord Provost by
William Balleny, whose occupation was not stated nor was his interest in burial grounds
and mortality explained, but it is probable that he was the superintendent of the newly
opened Edinburgh Cemetery in Warriston Crescent — he is listed in 1846 as the
secretary of the Edinburgh Cemetery Company, a commercial organisation of which the
Lord Provost was chairman. The letter asked that the council consider establishing a
Mortality Bill for Edinburgh and Leith. The council remitted the matter to the Lord
Provost’s Committee where the need for ‘the publication at regular intervals of a
Mortality Bill for Edinburgh and Leith showing the number of burials of the different
sexes, and clarifying the ages and the diseases’ was acknowledged. The council
appointed Dr James Stark, ‘a Gentleman properly qualified who will take the requisite
trouble from motives of a scientific kind’ to undertake the collection of the statistics.

The fact that the secretary of the Cemetery Company wrote to the Lord Provost, its

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40 Oliver & Boyd’s *New Edinburgh Almanac 1847*, (Edinburgh, 1847) and *The Post Office Edinburgh Directory 1846–7*, (Edinburgh, 1846).
41 Edinburgh Council Records, 245, 3 February 1846.
Chairman, suggests that before the letter was written discussions had taken place, perhaps involving Balleny, the Lord Provost and Dr. Stark, since the committee appointed Stark to collect mortality returns the day following receipt of Balleny's letter. Whether Stark had persuaded Balleny and the Lord Provost that the collection of death statistics was in the best interests of both the city and the burying ground or whether there was some perceived financial benefit to the Cemetery Company cannot now be resolved. Stark was prepared to carry out the work without payment and the council undertook to bear the expense of printing the necessary forms, expressing the hope that the extra work would be done by the burial ground Recorders readily and freely 'from a desire to perfect the Statistics of their respective offices'.

Thereafter, Stark produced periodic reports on the city's mortality, published in the press at intervals and first presented as a learned article in the *Edinburgh Medical and Surgical Journal* under the heading 'Statistics' in 1848.

The lack of a formal system for recording vital statistics in mid-nineteenth Edinburgh presents difficulties for the research student but also was a matter of regret in the 1840s, expressed by Dr. Paterson of Leith in his analysis of the epidemic of 1847–48. When estimating the proportion of fever deaths to the total mortality, Paterson was forced to rely on mortality figures from the province of Leinster in Ireland to show that fever deaths there were 10 per cent of the total mortality, in Dublin the figure was 4 per cent and in London 2 per cent. He commented 'we have no statistical data to which we can refer in Edinburgh' but he thought that the fever mortality must be higher than that of London.

There is no evidence that the town council or police commission thought that mortality data might prove useful in the management and prevention of epidemic disease. They did not have the excuse of ignorance being well aware of measures taken elsewhere.

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42 Edinburgh Council Records, 245, 3 February 1846.
In the eighteenth century mortality bills for the city had been authorised and for a time were collected but Stark's figures are the first mortality data collected in the nineteenth century. Whether it was a lack of available data or a lack of interest in statistics that was the problem is unclear; Christison wrote on fever epidemics, beginning with the epidemics of 1817, in reviews published in 1858 and 1863, but few statistics are included.\textsuperscript{45} Alison's observations on the fever epidemic of 1843, which form part of his argument for reform of the Scottish Poor Law, contain figures for fever cases treated in his wards in the infirmary, but the lack of any statistics for the number of deaths in the city obliged him to use Glasgow mortality figures that were given to him by Alexander Watt, the collector of mortality bills for that city.\textsuperscript{46}

Stark produced his first report on the mortality of Edinburgh and Leith for the months of January and February 1846. He wrote in his introduction that 'the leading features of the plan now so successfully carried out in practice over England and Wales, under the registration act, have been adopted' and explained why this had been done; first, it was essential to use a similar classification so that comparisons could be made; second, there was every likelihood that this classification would be adopted when the registration act was extended to Scotland, wrongly claiming that Scotland was 'the only country of Europe in which tables of the births, deaths and marriages of its population are not published.'\textsuperscript{47} Stark's classified table of diseases had thirteen classes, (table 5.3), the form in which the reports continued to be presented and published until 1848 when the council decided to cease publication of mortality figures during the cholera epidemic. At the height of the epidemic Stark asked the council if he should continue to collect mortality bills, saying that he had continued to do so but none had been published since October. The Lord Provost's committee considered the matter and recommended that


the bills should be collected as usual but should be published only in the medical press; the council voted against this and in the end it was agreed that the bills should be prepared in the normal way but should not be published. This debate and outcome in the middle of the cholera epidemic, while typhus was still active, leaves the impression that the council, for whatever reason, wished to keep the true extent of the crisis from the Edinburgh public.\textsuperscript{48} Perhaps they were unwilling to publicise the fact that typhus and cholera were affecting certain sectors of the population almost selectively, a view that will be explored later.

<table>
<thead>
<tr>
<th>Class</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Zymotic (epidemic, endemic and contagious)</td>
</tr>
<tr>
<td>II</td>
<td>Diseases of uncertain or variable seat</td>
</tr>
<tr>
<td>III</td>
<td>Diseases of brain and nervous system</td>
</tr>
<tr>
<td>IV</td>
<td>Diseases of respiratory system</td>
</tr>
<tr>
<td>V</td>
<td>Diseases of heart and blood vessels</td>
</tr>
<tr>
<td>VI</td>
<td>Diseases of stomach, liver and other organs of digestion</td>
</tr>
<tr>
<td>VII</td>
<td>Diseases of kidney and urinary organs</td>
</tr>
<tr>
<td>VIII</td>
<td>Child–birth and diseases of organs of generation</td>
</tr>
<tr>
<td>IX</td>
<td>Rheumatism, diseases of the bones, joints,&amp;c.</td>
</tr>
<tr>
<td>X</td>
<td>Diseases of the integumentary system</td>
</tr>
<tr>
<td>XI</td>
<td>Old age</td>
</tr>
<tr>
<td>XII</td>
<td>Intemperance, privation, violent deaths, suicides, unspecified</td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Still–born</td>
<td></td>
</tr>
</tbody>
</table>

Table 5.3. Stark's Classified Table of Diseases.\textsuperscript{49}

1846 is the first full year for which there is a report written by Stark and in this he

\textsuperscript{48} Edinburgh Council Records, 251, 19 December 1848.
comments on the increased mortality experienced by Edinburgh in common with every other town in Great Britain, an increase he attributes to atmospheric influences. From June until September the average temperatures were several degrees higher than in 1845, a circumstance that Stark claims caused greater evaporation and consequently higher rainfall. 'The heats, too, with this moist loaded atmosphere, acting on decaying animal and vegetable matters, increased the virulence of the emanations from drains, sewers, collections of filth, mud banks, stagnant waters, &c., and tended still further to induce that weakened state of the system which predisposes to disease.'

Stark wrote that the increased mortality was general with no fatal epidemic except for an increase in bowel complaints during the summer quarter. The deaths from bowel disease listed in class I (zymotic disease) totalled 378 but did not include the 160 children who died from teething listed in Class VI, gastro-intestinal disorders. All of these deaths from diarrhoea, dysentery and British cholera, approaching 550 in total, are likely to have been the result of epidemics of enteric infection.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Smallpox</td>
<td>36</td>
</tr>
<tr>
<td>Measles</td>
<td>215</td>
</tr>
<tr>
<td>Scarlet fever</td>
<td>12</td>
</tr>
<tr>
<td>Whooping cough</td>
<td>288</td>
</tr>
<tr>
<td>Croup</td>
<td>69</td>
</tr>
<tr>
<td>Enteric fevers</td>
<td>538</td>
</tr>
<tr>
<td>Typhus fever</td>
<td>269</td>
</tr>
<tr>
<td>Influenza</td>
<td>6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1433</td>
</tr>
</tbody>
</table>

Table 5.4. Deaths from infection, Edinburgh and Leith, 1846.

The number dying from infectious disease in 1846 was 1,433 out of a total mortality of 5752, approximately 34 per cent. Stark calculated that in 1846 the deaths of children under the age of fifteen years amounted to 425 out of 1,000 deaths of all ages, compared

to 453 out of 1,000 deaths in London. The three year average for London was 471, Bristol 474, Birmingham 546, Glasgow 564, Manchester 564, and Liverpool 583. Stark believed that these figures justified the statement made in his inquiry into the sanatory state of Edinburgh ‘that for children it is the healthiest city of the kingdom,’ and demonstrated ‘that Edinburgh is one of the healthiest, if not the very healthiest town of Great Britain,…’ In his 1847 report Stark admits that the mortality was higher than it had ever been but was quick to emphasise that ‘the same high mortality has very generally prevailed in the larger towns of Britain.’ He attributed the increase in mortality to zymotic disease, defining this as epidemic, endemic, and contagious and table 5.5 shows the eight infections responsible for the 2,206 deaths of which typhus fever deaths totalled 1,625. The total mortality from all causes was 8,057.

<table>
<thead>
<tr>
<th>Smallpox</th>
<th>227</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measles</td>
<td>233</td>
</tr>
<tr>
<td>Scarlet Fever</td>
<td>23</td>
</tr>
<tr>
<td>Whooping Cough</td>
<td>321</td>
</tr>
<tr>
<td>Croup</td>
<td>102</td>
</tr>
<tr>
<td>Enteric Fever</td>
<td>535</td>
</tr>
<tr>
<td>Typhus Fever</td>
<td>1625</td>
</tr>
<tr>
<td>Influenza</td>
<td>140</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2206</strong></td>
</tr>
</tbody>
</table>

Table 5.5. Deaths from infection, Edinburgh and Leith, 1847.

The mortality in 1848 from all causes amounted to 7,035 with infections responsible for 3,031 of that total. Table 5.6 on the following page shows the distribution of individual infectious diseases.

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Smallpox | 101
Measles | 64
Scarlet Fever | 689
Whooping Cough | 116
Croup | 69
Enteric Fever | 302
Typhus Fever | 1092
Influenza | 51
Cholera | 663
Total | 3147

Table 5.6. Deaths from Infections, Edinburgh and Leith, 1848.\(^{55}\)

Stark’s statistics for the three years show that there were 6,786 deaths from infection out of a total mortality of 20,844, about 33 per cent.

These statistics demonstrate the importance of epidemic disease in the city’s high mortality and had Stark’s data been broken down according to district of residence the distribution of disease in proportion to the degree of social deprivation might have been revealed, an outcome that was delayed for twenty years until Henry Littlejohn’s report of 1865.\(^{56}\) When considering Watt’s offer to collect statistics the Town Council had in 1841 accepted ‘that a knowledge of the Laws of Mortality is intimately connected with the social condition of the people...’ but this apparent concern for social conditions did not last. Five years went by before Stark was appointed and twenty years passed before Littlejohn’s report demonstrated the relationship between death rates and population density. MacDonald makes the point that the absence of mortality statistics meant that there was considerable public and official indifference to the high death rates in the Old Town, particularly so after the successful campaign to change the Scottish Poor Law.\(^{57}\)


\(^{57}\) H.MacDonald, ‘Public Health Legislation and Problems in Victorian Edinburgh, with Special Reference to the Work of Dr Littlejohn as Medical Officer of Health’, University of Edinburgh PhD, 1972.
The survey of statistics carried out in this chapter suggests that the failure of the municipal authorities to implement the collection of mortality figures for the city had major repercussions for social policy in the city. The next two chapters will therefore explore the theme of social conditions looking at the environment in the most congested areas of the city, concentrating on housing, the homeless and the provision of water and sanitation.
Chapter Six

Living Conditions in the Old Town: Housing and Lodging—Houses

The theory that malnutrition and infectious disease are somehow linked was examined in an earlier chapter, the conclusion being that nutritional deficiencies, both quantitative and qualitative, increase morbidity and case fatality. In the past it was generally held that pestilence followed famine and the belief in a causal link between extreme malnutrition and infection is supported by evidence that in famine disease kills as many people as starvation itself.¹ The mechanisms that adversely affect individual resistance to infection when nutritional status is low are now thought to have less of an impact on the development and spread of disease than socio-economic factors and the breakdown in normal social behaviour. In time of famine, social disruption from the mass migration of people leads to overcrowded refugee camps where water is in short supply, sanitation is absent and there is a failure of personal and group hygiene. It is this environment that exposes people to infection and when disease affects people with low nutritional status the outcome is a mortality crisis.²

Famine is a ‘complex socio-economic phenomenon’ in which migration in search of food, family disintegration, a disregard for normal behaviour and the breakdown of traditional social bonds become crucial elements in the causation of epidemic disease.³ These occurrences are not unique to famine — the crowded slums of rapidly expanding Victorian cities had many similar features. These cities attracted migrants in search of work, food and shelter, who, remote from family and neighbours, often had no alternative but to stay in grossly overcrowded houses or in temporary lodgings without

heating, water or sanitation. The urban squalor and social deprivation evident in cities like Glasgow and Manchester was blamed on the demands of industry and there is a suspicion that in the minds of many the absence of industry therefore denoted an absence of destitution This attitude was being criticised by the author of a regular feature on trade and commerce in Tait's Edinburgh Magazine when he wrote that 'even in Edinburgh, few as her manufactures are, the destitution is considerable;'

4 There was an assumption that destitution and manufactures were linked and from this arose the belief that Edinburgh as a non–industrial city was somehow immune to these social ills. It is this image of Edinburgh that this chapter will address, concentrating on social conditions in the Old Town during the middle of the nineteenth century.

Edinburgh is a city whose eighteenth century architectural and intellectual achievements blinded many commentators to the squalor existing in the midst of elegance and prosperity. The condition of the poor and the destitute in Edinburgh during the period under review will be examined, first analysing the state of the city's housing, focusing on conditions in the Old Town. It is against this background that the chapter will continue by examining issues relating to the influx of migrants and to the number and state of lodging houses for the poor. These topics are critical to an understanding of living conditions and the health of the people. Other key aspects of life in the Old Town, water, cleansing and sewage, will be dealt with in a separate chapter where the fetid irrigation controversy will also be explored.

Robert Chambers in 1868 wrote a new introduction to a revised edition of Traditions of Edinburgh, first published in 1825, explaining that 'this little work came out in the Augustan days of Edinburgh.'

5 He described the changes of the previous hundred years and the division of the city into the Old and the New, but at no point does he mention the appalling conditions endured by the 'humbler classes.' It is a nostalgic memoir.

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5 R. Chambers, Traditions of Edinburgh, (Edinburgh, 1825), p.v. The introduction from which the quotation is taken was written in 1868 for a revised edition. The volume consulted is a 1931 reprint of the 1868 edition.
The foundation was at length laid of that revolution which has ended in making Edinburgh a kind of double city — first, an ancient and picturesque hill-built one occupied chiefly by the humbler classes; and second, an elegant modern one, of much regularity of aspect, and possessed almost as exclusively by the more refined portion of society.  

Later authorities have concentrated on specific aspects of the city as Youngson did in *The Making of Classical Edinburgh*, describing the building of the New Town and as Rodger does in *The Transformation of Edinburgh* when he analyses developments during the second half of the nineteenth century. Rodger does highlight the fact that 'the industrial interests of Edinburgh were almost invariably presented as weak and suggests that the assessment in a guide book of 1849 was not untypical: “Edinburgh’s manufactures are few and on a limited scale”'. Great manufacturing industries may have been rare in Edinburgh but as Rodger points out 63 per cent of men and 23 per cent of women in 1841 worked in industry. It is clear therefore that the consequences of an industrial depression would have serious consequences for the workforce.

Anne Hardy has stressed 'the importance of local conditions, and of social dislocation in particular, in creating an environment favourable to epidemic typhus' and attributes Edinburgh’s outbreak of 1826 to ‘failures in building speculations which disturbed its internal economy’. The building slump and trade depression to which Hardy refers persisted for several years, throwing many out of work. It will be shown later in the chapter that the commercial depressions affecting the city from the 1820s through to the 1840s were not solely responsible for altering the city’s ‘internal economy’; other factors were involved in creating an environment in which epidemic disease could readily take hold. Many of the new inhabitants of the city had little or no experience of urban living and were ill-adapted to life in a city where accommodation was scarce and, when obtainable, crowded and filthy. During the 1840s, Edinburgh, in common with

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many cities in Europe, experienced several major outbreaks of infectious disease. Epidemics of typhus and relapsing fever broke out in 1841 and again in 1843–44; typhus and relapsing fever became epidemic once again in 1846–49 with, on this occasion, an outbreak of typhoid fever to add to the suffering; finally, the world–wide cholera pandemic of 1829–1854, which had first involved Edinburgh in 1831–32, reappeared in 1848–49. 9 In addition to these extreme mortality crises there were other infectious diseases which were always present to some extent, at intervals increasing in severity and virulence with identifiable increased mortality. For example, there were epidemics of whooping cough, or chincough as it was more often called, in 1840–41 and 1843; measles killed many in 1842–43 and again in 1849. Despite vaccination, smallpox was seldom absent from the city and a major epidemic occurred in 1842.10 These episodes will be reviewed in later chapters when the association between social conditions in the Old Town and the spread of disease will be explored further.

Epidemics have a selective demographic outcome, affecting the poor unequally, the impact being greater on the urban poor congregated in overcrowded squalor than on their better–off neighbours living in the prosperous suburbs. The effects on the rural poor and the urban middle class are similar with both groups less affected. There may have been little to choose between nineteenth century socio–economic conditions among the poor in town and country but the less concentrated and more widely distributed rural population and its relative isolation checked the spread of disease.11 It was in the city slums of Victorian Britain that this selective effect on the deprived was most obvious.

10 Edinburgh City Archive, St Cuthbert’s Churchyard Cash Book 1836–1854; Edinburgh City Council, Mortonhall Crematorium, East Preston Street Burial Registers 1841–1850.
The Old Town of Edinburgh during the first half of the nineteenth century had many of the features of a high-density ghetto and the next section will review the alterations and improvement schemes that led to a dangerous degree of overcrowding. Modifications to several of the streets in the Old Town in the decades immediately preceding the period of this thesis led to the removal of hundreds of houses, a loss that had serious implications for the inhabitants at a time of population growth and a mere six years after a disastrous series of fires had destroyed many homes.

In the twenty years before 1840, events and developments, planned and unplanned, reduced the number of houses in the old town. The most dramatic of these were the fires of 1824, among the worst in the city's history and the cause of great loss of property. The destruction of so many tenements, 'while helping to reduce congestion in the High Street, caused congestion in other parts of the old town' is how a twentieth century commentator viewed the disaster.12 There were two major conflagrations, both spreading from the High Street to involve closes near Parliament Square. The first, in June, destroyed five houses each of six storeys in the vicinity of Royal Bank Close. The second, in reality a series of fires, was more serious; it took hold in Old Assembly Close off the High Street on 15 November, destroying twenty-two lands, each of at least six storeys, some of eleven storeys and six tenements in Borthwick's Close.13 There is no contemporary estimate of the number of families made homeless by the June fire but Robert Chambers thought that in the November blaze 'three to four hundred families were burnt out by this dreadful calamity...'.14 Somewhere in the region of four hundred families, at a conservative estimate one thousand people, were forced to find new accommodation in the already overcrowded tenements of the Old Town, in other parts of the city or at a distance.

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14 R.Chambers, Notices of the Most Remarkable Fires in Edinburgh, (Edinburgh, 1824), p.54.
This inadvertent loss of housing stock was followed three years later by deliberate removal of houses through the compulsory purchase and subsequent demolition of tenements when the changes authorised by the Improvement Act of 1827 were implemented.\textsuperscript{15} Reid, writing forty years later on the subject of ‘dwellings for the people’ explained how:

\begin{quote}
In course of years the Old Town mansions were deserted and converted by a process of partitioning into houses for the working classes, whole blocks of buildings were swept away and no adequate provision was made for those whose dwelling place had been removed. While the demand for houses was increasing from the natural growth of the population, the number of houses was being steadily diminished…\textsuperscript{16}
\end{quote}

Reid was criticising the wholesale demolition of dwellings in the Old Town sanctioned by the Improvement Act. The Act was designed to improve communications and allow the city to expand into and develop land lying to the west and south. Sanitary improvement was never an issue, but when it became apparent that there was widespread public resistance to the scheme, the Council promptly claimed that the planned removal of insanitary dwellings was an integral part of the project and therefore a major reason why the plan should be approved and, just as important, funded. It has been pointed out quite correctly that ‘if there were any sanitary results these were never discussed at the time and were simply a by–product.’\textsuperscript{17} Eleven major alterations were included in the Act: first, a road from the west end of the Lawnmarket along the south of the Castlehill was planned in order to develop the so called ‘Western Approach’ to the city; second, the country to the south was to be opened up ‘by a New Street and Bridge over the Cowgate towards Bristo Port’, the present George IV Bridge.\textsuperscript{18} The improvements involved also the widening of Castle Hill by removing houses on the north, improving access to the Grassmarket from the west by widening the West Port

\textsuperscript{15} An Act for Carrying into Effect certain Improvements within the city of Edinburgh and adjacent to the same, 7 and 8 George IV, cap.LXXVI. (1827).
\textsuperscript{18} Youngson, The Making of Classical Edinburgh, pp.170–181 quoting the preamble to the Improvement Act of 1827.
and from the east by opening a route to the Lawnmarket, the present Victoria Street. The streets and wynds of the old town affected by these proposals included the Lawnmarket, the Grassmarket, Castle Hill, north and south, Bank Street, and Candlemaker Row. The plan involved the destruction of many tenements at considerable cost with a total of £29,516 being spent on the purchase of property to enable the south approach to be built and £22,092 on the west approach.19

It was claimed that the new roads and bridges, George IV Bridge, Victoria Street and Johnston Terrace were vital if the city was to develop geographically and commercially. When making space for these new streets many insanitary dwellings were to be swept away, an outcome that was no doubt seen as a bonus by all but the erstwhile inhabitants. The sanitary improvement which would accrue was used by the promoters as a reason for the scheme to go ahead and to persuade the reluctant ratepayers that there would be worthwhile benefits. This propaganda exercise was essential in order to justify the financial outlay that was to be borne by the citizens through taxation, particularly when it is remembered ‘that Edinburgh was the most highly assessed town in the Kingdom.’20 Rodger gives an account of the factionalism that developed in opposition to the bill; there were accusations of architectural vandalism; New Town residents opposed what they saw as a tax subsidy to the Old Town; landlords were concerned about lost rents; there were anxieties expressed about immigrant Irish labour flooding in looking for work on the new schemes.21

The Improvement Bill listed the 834 dwelling houses scheduled for demolition together with the names of their owners and occupiers, the great majority in multiple occupation. The number of families who were to be displaced cannot be calculated but it may well have exceeded 2,000, for the list of occupants in the Bill indicates that there were

21 Rodger, The Transformation of Edinburgh, pp.95 & 96.
seldom fewer than two families in each dwelling and often very many more.\textsuperscript{22} The Act became law in 1827 and the project went ahead. In 1849, the aftermath of the Improvement scheme was described by the Rev. James Begg:

Every one who reflects on the changes which have recently taken place in Edinburgh must see one great cause of the evil. Multitudes of houses have been swept away at the top of the Lawnmarket, at George IV. Bridge, and especially at the Low Calton, and yet scarcely one new tradesman’s house has been built. There is the same population, but, of course, living in far less room, wedged and crowded into filthy cellars and windowless garrets — festering masses piled and heaped together, and spreading disease and crime in every direction.\textsuperscript{23}

The demolition of so many houses at a time of population expansion from inward migration and natural growth with little or no replacement building led inevitably to overcrowding in the Old Town. Apartments that were already occupied by more families than was reasonable were now subdivided over and over again; “made down” was the expression commonly used to describe this process. Reid, wrote ‘that houses already too small and over-crowded were still further subdivided; families and lodgers were crowded into lightless boxes’.\textsuperscript{24} It was not simply the loss of houses that was significant, the creation of new streets in the West Bow, the Lawnmarket and the Grassmarket made matters worse by reducing the acreage of potential building land.\textsuperscript{25} Moreover, clauses in the Improvement Act prohibited new building on certain sites after these had been cleared, including land adjacent to the Western Approach road and space to the east of George IV Bridge that was to be kept free for an extension to the library of the Faculty of Advocates.\textsuperscript{26} This was not the end of the story, however, further demolition of houses and loss of potential building land was the end result of commercial and transport developments.

\textsuperscript{22} An Act for Carrying into Effect certain Improvements within the city of Edinburgh, pp.44–48.
\textsuperscript{23} J.Begg, Pauperism and the Poor Laws, (Edinburgh, 1849), pp.23 & 24. Rev Dr James Begg (1803–1883), a prominent Free Church minister (Newington, Edinburgh), held strong social and political views preaching and writing on pauperism, housing and crime.
\textsuperscript{24} Reid, Past and Present or Social and Religious Life in the North, p.146.
\textsuperscript{26} Wood, ‘Survey of the Development of Edinburgh’, p.49.
The completion of the North British Railway between Edinburgh and Berwick in June 1846 and the opening of the North Bridge station (later Waverley) the following year enabled the Edinburgh and Glasgow and the North British Railway to meet in the same terminus. This was undoubtedly a convenient arrangement for the railway companies and their customers but achieving this was not without price. Many old and distinguished buildings were demolished to make way for the railway but of greater significance was the cost in terms of houses destroyed. The railway scheme involved taking down the Old Orphan Hospital, Lady Glenorchy’s church, Trinity Hospital and a large number of houses on the north of the old town ridge and across the valley towards Calton. The Statistical Account of Edinburghshire noted in 1845 that the inmates of Trinity Hospital were to be ‘boarded out’ until Moray House was made ready for them but made no reference to the loss of private housing nor did it say what arrangements, if any, were made for the occupants of the tenements demolished to enable railway expansion to go ahead. The North British Review commented that the clearing of wynds and slums for a rail terminus would ‘effect a sanatory improvement’ but was critical of the fact that there was no plan to re-house the people left homeless. There was no reference in either the Statistical Account or the North British Review as to the number of houses demolished or the number of people left homeless. However, it has been estimated that between 200 and 300 houses, mostly tenements, were demolished by the Railway Access Company during the years 1830 to 1847. This loss of housing and its replacement by railway yards of course meant that the land previously used for housing was no longer available, exactly as had happened with the construction of the new roads under the Improvement Act of 1827. The improvements in transport and in ease of communication brought benefits to the city but the cost in terms of social deprivation was considerable.

The railway was not the only commercial enterprise that deprived the city and its inhabitants of housing and land. The expansion of breweries in the Canongate further reduced the number of houses and the acreage of land available for new building was also diminished. These transport and business initiatives may have been important for the city's prosperity and economy and the negative effect on housing was perhaps an unfortunate and unavoidable complication of progress but there was another less obvious element that has been implicated in the gross overcrowding in the Old Town.

The loss of housing stock and building land was critical particularly since there had been little new building in the city after about 1825. Rodger highlights this housing deficit commenting on 'the haemorrhaging of housebuilding [having] particularly adverse effects on working men and their families,' and explains that not only was there no affordable housing being built for the poor but the lack of new middle-class housing development halted the process of "filtering-down" by which those at the bottom of the housing market benefited from housing developments higher up the scale. The 'alternative housing strategies' that were essential if overcrowding was to be controlled were not implemented until the 1850s. There was little further extension of the city boundaries and no significant improvement schemes between the implementation of the 1827 Act and 1856, perhaps because the Town Council was engaged with other more pressing matters during this period. This absence of new initiatives at a time when population was increasing simply added to the congestion and what Rodgers describes as the 'arrested development' in the Edinburgh property sector between 1825 and 1850 was an additional factor in the creation of "a contagion of numbers" in the Old Town's already overflowing tenements. He stresses the effect this had on a city where natural growth and inward migration were responsible for population expansion outwith its

experience and 'which only public intervention could address.'

34 Rodger, The Transformation of Edinburgh, p.82.

There was no involvement by central or local authority in the plight of the destitute population of the Old Town, their only advocates being the clergy and some of the medical profession. In the next section population density will be analysed, deprivation and squalor in the Old Town will be explored by reviewing descriptions of social and housing conditions by contemporary observers.

One example from the census of 1851 tells of the extent of overcrowding in the parish of Greyfriars Old where there were 173 houses inhabited, three not inhabited, with 1,034 families, 4,345 people, occupying these dwellings and only one new house under construction. This density of approximately twenty-five people to each house is considerably greater than the estimated density of sixteen persons per house for the whole of the city. The only parish with an appreciable number of houses under construction at the time of the census was St Cuthbert’s with fifty-seven, whereas there were only two new houses being built in the whole of the Old Town, the area of the city with the greatest concentration of people and the worst overcrowding.  

35 Census Enumerators’ Abstract, p.970.

There are many descriptions of life in the wynds and closes of the Old Town during the decades before and after 1850. In 1856, Henry Johnston published a Letter to the Lord Provost, Magistrates and Council of the City of Edinburgh in which are analysed the closes, courts and wynds of the Old Town. Johnston wrote:

I have during this winter examined carefully all the closes and courts (159 in number) on both sides of the Lawnmarket, the High Street, and Canongate from the Castle to the Palace, and also all those on both sides of the Cowgate. This examination has revealed to me a state of affairs, in regard to the majority of the human beings living in them, disgraceful to a civilised community.

William Chambers in his *Report on the Sanitary Condition of the Old Town of Edinburgh* (1840), written in the form of a letter to Edwin Chadwick, Secretary to the Poor Law Commissioners, concluded that ‘society, in the densely peopled closes which I have alluded to, has sunk to something indescribably vile and abject ... at the present moment, the poor of Edinburgh may be said to be deserted by almost everybody but the surgeon or physician.’ There were medical men who struggled to publicise the state of the poor and numerous articles and pamphlets dealing with the situation in the city written by ministers of the kirk. In a collection of tracts on *Social Reform* published in 1851 by the Scottish Association for the Suppression of Drunkenness, the Rev.W.G. Blaikie, Minister of Pilrig Free Church, wrote ‘The Dwellings of the People’ which had three sections: influence of dwellings on health; influence of dwellings on morality and lastly influence of dwellings on social feelings and habits. In his introduction Blaikie said ‘it would not be easy, we think, to over-estimate either the dreadful evils which have flowed directly from the wretched dwellings of many of the working classes, or the blessings that would almost certain to be reaped, if they possessed houses of a better kind.’

One of the few women to write about conditions in the Old Town was Isabella Bird whose *Notes on Old Edinburgh* was published in 1869 and in which she expressed her belief that there was no city in Europe with ‘an area of wretchedness so large and unbroken as Edinburgh.’ She made the point that ‘in other cities the miserable dwellings and their inhabitants hide themselves out of sight in obscure purlieus, scarcely known to the rich by name, much less by observation.’ In Edinburgh, she said, the High Street was at the very centre of the city with the Law Courts, the Parliament House, the City Chambers, the Assembly Halls of the Established and the Free Churches, the Cathedral,

37 W.Chambers, *Report on the Sanitary State of the Residences of the Poorer Classes in the Old Town of Edinburgh*, (London, 1840), p.3. This extract from Chadwick’s official report was published as a separate pamphlet, from which the quotation is taken.
newspaper offices and printing establishments all lying in close proximity to some of the worst slums in the country.39

It is not surprising that many of the most disturbing accounts of life in the Old Town were written by clergymen and doctors, two professional groups with first hand knowledge. Professor W.P.Alison in his Observations on the Management of the Poor in Scotland, and its effects on the Health of the Great Towns wrote that ‘it would be difficult to credit the existence of such a state of misery and distress as is mentioned, on unquestionable authority, to be frequently found in some parts of the old town of Edinburgh.’ Alison quoted William Tait, “In many of the closes leading from the High Street one flat alone, situated in Foulis’ Close, may give an idea of the extent to which dunghills within doors and in houses inhabited by poor families are sometimes carried.”40 William Tait was surgeon to the Edinburgh Police and had given evidence on sickness and living conditions in the wynds of the city to the Poor Law Commissioners gathering information for the Report on the Sanitary Condition of the Labouring Population of Great Britain.41 There were other notable contributions by medical men; Alexander Hunter, a surgeon, arranged for his letters to the Weekly Express to be published in pamphlet form in 1847. These were concerned with the fever then raging in the city and in a description of the homes of the ‘working orders’ he wrote of ‘the loathsome state of their dwellings — dwellings did I say? — human cesspools would be a more correct term, as pointing out more accurately the real state of their habitations,.....’.42

One of the most detailed and graphic accounts of social conditions in the Old Town was that of Dr George Bell who in 1849 wrote Days and Nights in the Wynds of Edinburgh, a monograph which proved so popular that it was re-issued at least four times before the

end of 1850. That year Bell wrote *Blackfriars' Wynd Analysed*, a case study of a well-known Old Town landmark, chosen after considerable thought, 'the difficulty being to discover what may be fairly considered as average.' In his opinion the wynd was as near to being average as was possible, 'both as regards the condition of the population and that of their dwellings.' George Bell (1813–1889), the son of an Edinburgh surgeon, studied medicine at the university, graduating MD in 1835 with a doctoral thesis on *Delirium Tremens*, a condition associated with alcoholic excess and one that he must have had ample opportunity of studying. Bell did not follow his father as a surgeon, becoming a physician and later, influenced by Rev. Thomas Chalmers and Rev. Thomas Guthrie, a champion of the rights of the poor. In his analysis of Blackfriars' Wynd, Bell estimated that there were 597 people living there in February 1848. Two years later in April 1850 this had grown to 1,025, the increase, he explained, was because of the large number of renters of dwellings who took in lodgers. This was certainly possible because the census of 1851 records that there were 1,266 lodging-house keepers in Edinburgh and Leith, an astonishing figure, but perhaps realistic in view of Bell's explanation for the increase in the population of Blackfriars' Wynd. Bell believed that the average population of the wynd was normally closer to 1,000 people than to 500 and estimated that between 600 to 1,000 people were accommodated in 142 buildings with 193 chambers, although he admitted that the shifting population made estimates unreliable.

There were other reasons for this high population density than the loss of housing stock as a result of the changes described earlier. For many workers there was no alternative but to accept space in the squalid rooms on offer as they were too poor to afford anything better; for others, low wages and unemployment, because of seasonal

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45 *Census Enumerators' Abstract*, p.1020.
46 Bell, *Day and Night in the Wynds of Edinburgh and Blackfriars' Wynd Analyzed*, p.15. The first set of figures he ascribes to Mr. Thorburn, inspector of out-door poor, possibly the same Thorburn who analysed the census of 1851 cited above.
variations or cyclical depression in trade, were crucial in persuading many to opt for the cheapest accommodation that they could find. Hugh Miller (1802–1856), writer, geologist and mason by trade was editor of the influential newspaper, the Witness, first issued in January 1840. The Witness has been described as a newspaper which “advocates the interests of commerce, the principles of the Free Church, and of Evangelism generally; and takes the Liberal view of all political questions, though not bound to any political party.” 47 Miller told the Poor Law Inquiry that masons had to learn to save money during the summer to tide them over the winter when they would frequently be out of work because of adverse weather or, if employed, the short winter days would mean fewer hours worked and smaller wages. Moreover, he thought ‘that it would be a very great disadvantage to give parochial relief to the able-bodied and their families when thrown out of work by seasonal severity or extraordinary circumstances.’48 John Wright, an Edinburgh town councillor, believed that ‘a mason lost nearly one third of the year betwixt frost and rain.’49 These employment uncertainties had repercussions in the housing market. In a rental system dominated by the year long let, workers were committed to a regular weekly level of expenditure throughout the year whether they were employed or not and it is hardly surprising that many chose the cheapest option available. Many opted to rent low-priced housing, spreading the financial burden over twelve months when they could perhaps expect to be working for only six or nine months in the year.50

There were other constraints which encouraged builders and property developers to persist with high density housing of the tenement type to maximise the return on capital. Small units in a multi-storey tenement were far more profitable and allowed the owner of the land to obtain the maximum possible return from his feu. Stone, an extremely

49 Report of the Royal Commission on the Poor Law (Scotland), (1844), 20, pp.181 & 209.
expensive building material, was used almost exclusively at this time in Scotland, and when the legal fees incurred by the feuing system were added to the high costs of building in stone the pressure for high returns was great. The outcome was that many homes consisted of a single room, seldom with a water supply and, if there was any kind of sanitation, it took the form of a shared water closet. In the high tenements of the Old Town such conveniences were rare because of the difficulty of access for plumbing modifications and more importantly, as will be demonstrated in a later chapter, the totally inadequate supply of water for flushing drains and sewers.  

The great demand for labour in new expanding industries was not a factor in Edinburgh's population growth where there were few large manufactories requiring a vast labour force but there were other elements responsible for the inward movement of people to the city. Roger Schofield points out, admittedly in an English context, that there were 'significant changes in the structure of the demand for labour in the agricultural sector itself', alterations that had far reaching effects on the rural economy. The decrease in demand for unmarried labour and 'the marginalisation of women from the agricultural labour force' drove many from farm work to look for employment in the city. Those who left the country because of changes in agricultural practice were joined by others, both male and female, who were old or no longer physically capable of carrying on their trade; as was pointed out earlier, many people came in the hope of finding some kind of light work, 'some small job within their physical competence.' There was an influx of labourers to work on major projects such as the Union Canal (1818), the new roads and bridges sanctioned by the 1827 Improvement Act, the Dean Bridge (1832) and the several railways constructed after the passing of the first Railway

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53 Report of the Royal Commission on the Poor Law (Scotland), 20, p.90, the evidence of Rev. Thomas Guthrie, Minister of St. John's Church, Edinburgh.
Act in 1826. 54 Whatever the motivation for coming to the city, the rate of increase in population between 1801 and 1851 was 135 per cent, a rate greater than at any other time in the nineteenth century. 55 So long as work was easily obtained the pressure on poor relief and charity was containable but the slump which began in the 1830s persisted for several years with predictable results.

Figures produced by the Board of Supervision in their abstracts for the two years ending May 1847 and May 1848 show that there were 12,499 paupers and casual poor relieved in Edinburgh in the first twelve month period and 20,071 in the second period. 56 The economic downturn had begun ten years earlier and was particularly severe between 1837 and 1843 when many were thrown out of work in the country and towns alike. In 1840 and 1841, Tait’s Edinburgh Magazine described the difficult economic situation in Scotland, writing that ‘the distress in the manufacturing districts continues to increase’ and commented on the large number of sequestrations in Edinburgh with fourteen in a single week in January, a number not seen since the depression of the 1820s. In March 1840, the magazine recorded that ‘much distress prevails’ with thousands everywhere out of employment and the situation in 1841 was little different: dullness of trade, low profits and unemployed workmen. 57

The boom in railway construction during the 1830s and 1840s brought many workers looking for work on the new railway lines or hoping to be employed in some capacity by the railway companies. The birth of the railway network in Edinburgh was the opening in 1831 of the horse-drawn Edinburgh and Dalkeith Railway and from 1836 when the Edinburgh, Leith and Granton Railway was authorised there was a steady stream of railway workings and station building. The Edinburgh and Glasgow railway and its

56 P.P., Accounts, Number of Paupers Relieved including Casual Poor 1847 and 1848, 1850 (5) L.265.
station at Haymarket were completed in 1842 with the North British Railway and the North Bridge station completed in 1846. The Edinburgh, Leith and Granton opened from Canonmills to Trinity in 1842 and was extended to the Canal Street station by means of the Scotland tunnel in 1847. The last railway enterprise to be completed in the 1840s was the Lothian Road station belonging to the Caledonian Railway, which was opened in 1848.\(^{58}\)

William Johnston, a Town Councillor and deputy chairman of the committee for relief of unemployed operatives, in evidence to the Poor Law Commission, explained that hundreds of men had been thrown out of work on completion of the Edinburgh and Glasgow Railway. He estimated that this applied to 460 of the 1,100 men employed on work organised by his committee, the remaining 640 were of every trade and he surmised that the introduction of printing by machinery had thrown many out of work. Johnston told the Commission that the committee had purchased sickles and advanced money to workers to enable them to go to harvest work. By the end of September they had returned to the city from the harvest and the committee in order to relieve distress was forced to employ 345 men in the Meadows.\(^{59}\) The number of middle-aged and elderly moving to cities in the hope of obtaining a light job to eke out a living was significant and was a point discussed by many witnesses in the *Report of the Royal Commission on the Poor Law (Scotland)*. Neal summarised the situation in urban Britain at this time: 'rapid urban growth, unmatched by a simultaneous investment in infrastructure such as water supplies, sanitation and adequate housing, produced horrific conditions which large numbers of people had to endure.'\(^{60}\)

This chapter has concentrated hitherto on the contraction in the supply of cheap housing that took place during the 1820s and 1830s and has described the living conditions of


\(^{59}\) *Report of the Royal Commission on the Poor Law (Scotland)*, 20, pp.201–203. The men were employed breaking stones to make pathways in a park known as the Meadows near to the centre of the city.

those amongst the poor who could afford to rent space, no matter how tiny and squalid, in which to live. The predicament of the homeless who had no option but “to sleep rough”, to rent a bed in one of the many lodging-houses in the city, or to apply to one of the several charities devoted to helping the homeless has not been addressed. From the annual reports of the charitable homes for the destitute it appears that these were generally well run with acceptable standards of cleanliness and accommodation; it was the vast number of privately owned and run lodging-houses of all sizes devoid of water or sanitation that alarmed the public. The inhabitants of these houses were blamed for the fever epidemics that visited the city at regular intervals and the alarm that they created in the minds of the middle classes continued even after the introduction of Police supervision in 1848.\textsuperscript{61} The next part of this chapter, therefore, will concentrate on charitable homes for the destitute and privately run lodging-houses, focusing on how they were seen by the public and by official investigators.

The extent of contemporary suspicion of the destitute and the lodging-house is clear from the \textit{Report on the Sanitary Condition of the Labouring Population of Great Britain} in which the ‘common lodging house’ was given a special section. The explanation for this approach was that ‘they [lodging-houses] may apparently be better considered independently of the administrative arrangements which affect the resident population of the labouring classes.’\textsuperscript{62} The conviction that these houses and their inhabitants were the source of fever epidemics is stated on page one in which the first two paragraphs contain three references linking lodging-houses, vagrants and disease: ‘...the continued importation, if not the generation, of epidemic disease by the vagrant population who frequent them ...the common lodging-houses are pointed out as \textit{foci} of contagious disease within the district... these houses [and their inhabitants]...spread physical pestilence, as well as moral depravation.’\textsuperscript{63} This perspective on vagrants and lodging houses was not uncommon and was frequently coupled with derogatory comments about

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the Irish. This attitude to the immigrant Irish was typified by Chadwick when he referred to the increasing numbers of vagrants in Edinburgh and Glasgow, ‘especially of Irish vagrants’ and the evils that ensued. 64 This fear that vagrants harboured disease was not new to the city: the Edinburgh Police Act of 1822 obliged keepers of lodging–houses to give notice of residents ill with contagious diseases, failure to comply would result in a fine. 65 The dread of the diseased stranger has existed for centuries. Pullan in an essay on attitudes to the poor in early modern Italy suggests that the idea of contagion ‘accentuated the tendency to blame epidemics on strangers’ and drove vagrants to the very margins of society where ‘with little or no claim on charity they became prime candidates for expulsion.’ 66 This dread intensified as epidemics became more frequent in the early 1840s and one of the three ‘important improvements’ advocated in the Report of the Select Committee on the Health of Towns was in the condition of ‘the neglected and dangerous low lodging–houses, frequented by a wretched and migratory population, who often carry fever and other diseases into distant districts.’ 67

Public anxiety in Edinburgh had been heightened at this time because of fever and as a result of investigations into the condition of lodging–houses in the city. In evidence to the Select Committee on the Health of Towns, Dr James Simpson said that he knew of houses where strangers were lodging and in one of these ‘where a fever has been raging’ there existed a ‘dreadful degree of filth’ 68 It was this anxiety about lodging–houses as the focus of disease that prompted a group of concerned citizens to convene a public meeting in March 1841 presided over by the Lord Provost. A newspaper account of the meeting made a point of emphasising the dangers of lodging–houses and ‘the necessity of avoiding pollution.’ The report quoted the introductory address of Sheriff Speirs, a man of evangelical conviction, who had explained to the meeting that ‘in the present

65 Act for Watching, Cleansing and Lighting the Streets of the City of Edinburgh, G.IV Cap.LXXVIII 1822.
68 Report of the Select Committee on the Health of Towns, 1840, 11, p.119.
state of society, many persons were moving in a migratory state from one place and employment to another ... [and] many people from various causes were obliged to leave their homes and proceed to large towns, that they might obtain sufficient employment.\(^69\)

The police inquiry into the condition of lodging-houses had exposed the 'wretched and dangerous condition' of many and had stressed the efforts needed to remedy matters by 'the establishment of lodging-houses to serve as models for imitation and in some degree counteract the evils of existing state of things.'\(^70\) The desire to improve these establishments undoubtedly had an element of self interest, as the writer of a leading article in *Tait's Edinburgh Magazine* implied in 1840, when he wrote that the affluent classes, 'from their own danger,' were forced to take a greater interest in the plight of the less fortunate.\(^71\) Whether their fears were exaggerated or whether they simply arose out of a need for a scapegoat is difficult to determine. Nevertheless, there is plenty of evidence from contemporary writing that Edinburgh lodging-houses were grossly overcrowded, insanitary and the haunt of criminals and prostitutes, but it is much less certain that they were the only source of disease.\(^72\)

The responsibility for supervising these establishments rested with the Police Commission through its cleaning committee, a duty that was never easy and frequently impossible. The troublesome nature of the task was spelled out in 1847 by Alexander Murray, the Inspector of Cleaning and Lighting, to the Police Commission, explaining the difficulty in enforcing cleaning, white-washing and fumigation of houses: 'the only obstructions offered have been from keepers of low lodging-houses.' Murray was clearly unhappy with his powers under the Police Acts, explaining to the Committee that clauses in the Liverpool Sanatory Act of 1846 gave the city council summary powers and the Act was in his opinion worth scrutinising as an example of what could be

\(^69\) Scotsman, 10 March 1841.


\(^71\) Tait's Edinburgh Magazine, November 1840, p.682.

\(^72\) *Third Report by the Committee of the Edinburgh Lodging-House Association*, Appendix, p.8 has a letter from the Rev John Sym, minister of Greyfriars' Free Church, describing conditions in a number of lodging-houses in the West Port and the Grassmarket; Bell, *Day and Night in the Wynds of Edinburgh*, p.33.
achieved. The Liverpool Act authorised the Borough Council to set up a health committee, a body which could compel lodging-house owners to restrict the number of lodgers, to ensure that they arranged for the removal to hospital of residents with a contagious disease, to keep a register of residents and to maintain basic standards of cleanliness. The Council through its health committee also had the authority to inspect lodging-houses and to order that any disinfecting they deemed necessary was carried out.73

It is obvious that Liverpool’s health committee had greater authority and wider powers than the only body in Edinburgh that could be described as having any responsibility for public health, the voluntary Fever Board. This Board was very much an ad hoc committee, established in 1830 by ‘the leading medical gentlemen of the city’ and the Destitute Sick Society.74 The latter had decided to restrict their social work activities and this quasi public health role was taken over by the new voluntary body. The Board had as its President, the Lord Provost and the committee comprised two members of the Town Council, two physicians from the Infirmary, two medical officers from the Royal Public Dispensary and two from the New Town Dispensary, two members from the Destitute Sick Society, together with three special members, one of whom was Professor Alison.75 The secretary of the Fever board writing in 1844 regretted that ‘no proper system of Medical Police has been established throughout Scotland’ and criticised the refusal of the Police Commissioners to agree to a clause in the Police statute granting a yearly sum of money to the Board. After representations to the Commissioners, a clause was inserted in the latest Police Bill sanctioning a grant of one hundred pounds per annum to enable the Board to continue its functions of removing patients to hospital and cleaning.76 The Fever Board dealt with 9,609 cases of fever between 1830 and 1836 arranging for the fumigation of 4,065 houses and the transfer of 6,243 fever patients to

73 A.Murray, Nuisances in Edinburgh, (Edinburgh, 1847), pp.22–23.
75 Oliver and Boyd’s New Edinburgh Almanac 1845, (Edinburgh, 1845), p.516.
76 Deuchar, Observations on the Prevalence of Epidemic Fever in Edinburgh and Glasgow, p.21, the author, an Edinburgh lawyer, was secretary to the Fever Board.
hospitals in the city between 1840 and 1843. The Board appears to have worked closely with the municipal authorities in matters relating to the control and management of fever cases; a newspaper reported in 1840 that the Fever Board with the support of the council had instituted measures to provide a fever hospital but sufficient funds were not available. The Police Commission and the Fever Board did not see their role as preventative; they merely reacted to each epidemic crisis as it appeared. In contrast, the Liverpool health committee appointed its first Medical Officer of Health, Dr William Duncan in 1847, fifteen years before Edinburgh. Duncan, in common with so many of the protagonists involved in the early public health movement, was an Edinburgh graduate and gave evidence about the condition of the labouring population of Liverpool to the Sanitary Report.

Murray, the Inspector of Cleaning, was only too well aware that under the various Edinburgh Police Acts his powers and those of the Police Commission, particularly those applying to the supervision of lodging-houses, were severely limited and impossible to enforce. Liverpool was not the only British city with stricter and more readily enforceable laws than those in Edinburgh; the regulations in force in some of Glasgow's burghs were similar. In the Calton district of the city, for example, the Police Act legislated for inspection and approval of lodging-houses and gave the police powers to control the number of lodgers permitted in each house as determined by inspection of the premises and its facilities. The Glasgow Act also obliged lodging-house owners to display conspicuously a list of rules concerning health, cleanliness, and ventilation in every room in which lodgers slept. It was not until 1848 that Edinburgh tightened control of lodging-houses when regular inspection was introduced along with measures to ensure that the permitted number of lodgers was not exceeded. The success of these

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78 Scotsman, 8 January 1840.
measures was perhaps limited if the research carried out by James Bruce in 1850 into destitution in Edinburgh is to be believed. Bruce published his findings in *The Scotsman* and also in pamphlet form, describing 'the multitude of common lodging-houses in the West Port, which were the haunts of loose women, liberated prisoners and returned convicts.'82 It was several years before sufficient satisfactory alternatives to the old type of common lodging-house became available and the first of these came into existence as a result of the meeting of citizens referred to earlier. Their initiative in due course led to a better standard of lodging-house accommodation for the homeless by establishing what came to be called model lodging-houses.

The first Edinburgh Lodging-House Association home was opened in September 1844 in the West Port, the need to gather funds and to obtain a suitable property being responsible for the delay. This house was able to accommodate seventy lodgers at a cost of three pence per night and bed occupancy figures were initially fairly low with only 4,577 admissions in the first year, an average of thirteen residents nightly, rising to 17,836, an average of forty-nine nightly, in the third year of its existence.83 It is difficult to explain the reluctance of the homeless to use this new model lodging-house, apart from the possibility that many may have been deterred by its history; the house at 85, West Port was notorious for 'a peculiar interest attaches as the house in which the monster Burke committed his horrible murders'.84 A second house was opened in the Cowgate in 1847 with accommodation for eighty lodgers and by the time of the Committee's report early in 1848, occupancy was averaging almost fifty each night. In a letter to the Secretary, printed as an appendix to the report, the Rev John Sym wrote that an immense proportion of the fever cases, which at some periods have almost doubled the mortality of our city, have been traced to the Lodging-Houses for the poor and while our two Lodging-Houses are situated in the very centre of districts in which fever has been raging, not a single case of fever has originated in the one or the other.85

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82 J.Bruce, *Letters on Destitution and Vice in Edinburgh*, (Edinburgh, 1850), pp.11 & 12.
83 *Third Report by the Committee of the Edinburgh Lodging House Association*, pp.4 & 5.
84 *Appendix to Third Report by the Committee of the Edinburgh Lodging House Association*, p.9.
85 *Third Report by the Committee of the Edinburgh Lodging House Association*, p.10.
The report was critical of the indiscriminate mixing of the sexes in lodging-houses and advocated the establishment of a lodging-house exclusively for women, particularly for those who were 'unmarried and unprotected', claiming that there was a constant influx of such people from the country in search of employment. The committee considered that these women were in danger from the insanitary conditions and the intermingling of the sexes in ordinary lodging-houses. A circular included in the pamphlet and designed to attract charitable donations described the deplorable conditions in more than forty lodging-houses in the vicinity of the West Port and the Grassmarket.\(^{86}\) These forty establishments with room for 331 persons were a fraction of the total, the census of 1851 recording 1,336 lodging-house keepers in Edinburgh and Leith.\(^{87}\) Thorburn in his analysis of the census of 1851 records 567 lodging-letters in Edinburgh but rather surprisingly only 192 of them in the Old Town, an estimate at odds with the findings of other observers.\(^{88}\) For example, in 1865 when Dr. Henry Littlejohn, Edinburgh's first Medical Officer of Health, carried out a survey of lodging-houses he found that most were in the St Giles district of the Old Town which had 142 with 473 apartments and 1,049 lodgers. The total in the Old Town was 361 and the number of lodgers in licensed lodging-houses was 2,564.\(^{89}\) It is difficult to understand why there should be such a lack of agreement between the census figures, Thorburn's 1851 statistics and those of Littlejohn, particularly in view of the fact that Thorburn was the city's official census enumerator and is likely to have had access to accurate figures. It is unlikely that the number increased from 192 to 361 in the course of little more than ten years. The disparity between the two sets of figures for 1851 suggests that a different definition was used by the census enumerators in their abstract and Thorburn in his analysis. Whatever the reasons for the disparity it is certain that in the Old Town in 1840s there were at least 3,000 people living in lodging-houses.

\(^{86}\) Third Report by the Committee of the Edinburgh Lodging House Association, p.11.
\(^{87}\) Edinburgh Central Library, Extracts from the Census of 1831 and 1851, pp.1016 & 1020.
\(^{89}\) Littlejohn, Report on the Sanitary Condition of the City of Edinburgh, Appendix, p.61.
Until the establishment of model lodging–houses, Night Asylums, the Poor House and the crowded and insanitary lodging–house were the only options for the floating population of homeless men, women and children, wandering from place to place in search of work and shelter. The large number of lodging–houses in most Scottish towns and cities reflected the size of the migrant population. For example, in the county of Peebles 6,514 'vagrants, beggars and others' were sheltered in the twelve months to April 1843 and in the same year, the county of Perth was recorded as having 160 lodging–houses for 'vagrants and destitute people', more than seventy in the town of Perth alone, a figure which had risen to ninety-nine by the time of the census of 1851.90 The migrant worker and the destitute vagrant were common in all Scottish towns and cities and as a result several opened Houses of Refuge and Night Asylums to cater for them. Some critics blamed these establishments for attracting paupers by offering them the shelter they could not find in rural districts or in small towns while others, as was discussed earlier, blamed inadequate parochial aid, which drove the poor to the city for work and finding none, were forced to rely on charity.91 It is probable that both mechanisms were at work and those of a charitable outlook accepted this and continued to provide for the poor.

The first Night Asylum in Scotland was opened in Glasgow in 1838, Aberdeen in 1840 and finally Edinburgh in 1842.92 Two years earlier a Night Refuge or shelter, a separate charity from the Night Asylum, had been opened in Edinburgh, (July 1840), associated with the House of Refuge which itself had started life in Morison's Close, off the High Street, in 1832. The stated intention of the Night Refuge was to provide for the 'Houseless Poor', while admission to the House of Refuge depended on 'Destitution'. From its inception the House of Refuge had a degree of semi–official recognition, being used by the Police establishment as a place to send beggars. It was run as 'a

90 Report of the Royal Commission on the Administration and Practical Operation of the Poor Laws in Scotland, 20, 1844; Extracts from the Census of 1831 and 1851, pp.1016 & 1020.
philanthropic workhouse' with 200 inmates, half being children, many remaining in the house for years, even after starting work, and paying for their upkeep by their earnings. A lease of Queensberry House was taken in 1834 and four years later the Night Refuge was opened after the Prison Board had decided to cease sheltering the homeless in the police office, where hitherto between sixty and one hundred persons had been accommodated each night. In the year to the end of September 1840, 4,334 persons had been admitted to the Night Refuge, averaging over fifty per night; of those relieved 1,832 were natives of Edinburgh and Leith, 1,691 were from other parts of Scotland, 582 were from Ireland and the remaining 229 came from England and elsewhere.

The histories of those admitted to the House of Refuge are enlightening: a twenty-eight year old mother of six, abandoned by her husband, was admitted but was transferred to the Royal Infirmary with fever and little hope of recovery; a forty-three year old widow, born in Edinburgh who had lived in Ireland for eighteen years and because of this was refused parochial aid by the West Kirk parish, (St Cuthbert's), and sent back to Ireland where in due course aid was again refused and she was obliged to beg her way back to Edinburgh. The report, from which these histories are taken, was highly critical of the poor relief given to the destitute, demanding to know 'how can a poor widow with three or four small children, provide food, fuel, clothing and lodging on eighteen pence or possibly twenty one pence per week? It is really a matter of necessity, they must either beg or starve.' The following year over 13,000 people were relieved at the Night Refuge and 1,453 were admitted to the House of Refuge, 640 requiring medical treatment of whom thirty five were transferred to the Royal Infirmary. In January 1841, the House of Refuge contained 'the unprecedented number of 400 inmates while at the soup kitchen attached to it 'upwards of 100 individuals are daily supplied with

94 Anon, The Aged Poor, p.8.
soup and bread; at no former period have the resources of this establishment been so fully developed.\(^9\)

There are many contemporary descriptions of conditions in the closes and wynds of the Old Town during the middle of the nineteenth century. If some of these were perhaps exaggerated for effect or to encourage charitable giving and to promote reform, nevertheless, the picture they paint is grim. The account of William Chambers (1800–1883), publisher of the eponymous Edinburgh journal, Lord Provost (1865–1869) and proponent of slum clearance is fairly typical. Chambers described Old Town housing conditions in a letter to Edwin Chadwick, Secretary to the Poor Law Commissioners, an account that was later published as a pamphlet and included in the *Reports on the Sanitary Condition of the Labouring Population of Scotland*. He wrote that ‘after a pretty extensive observation of the condition of towns both on the continent and in Great Britain, I am of the opinion that this city is at present one of the most uncleanly and badly ventilated in this or any adjacent country.’\(^9\) Alexander Miller, an Edinburgh surgeon with sixteen years experience of attending the poor in the city, was asked by Chambers to reply to a series of questions and his answers were sent to Chadwick for inclusion in the official report.\(^1\) In reply to a question concerning the dwellings of the poorer classes, Miller replied

The dwellings of the poor are generally very filthy in their interior, and in many cases seem never to be subjected to any kind of cleaning whatever. Those of the lowest grade often consist only of one small apartment, always ill ventilated, both from the nature of its construction and from the densely peopled and confined locality in which it is situated. Many of them, besides, are damp and partly underground. ...In almost no instance is there a supply of water, nor is there any provision for carrying off filth; the effluvia accumulated from these causes are most offensive, especially when disease is prevailing in such dwellings.\(^1\)

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\(^9\) *Witness*, 22 January 1842.


There were many organisations dedicated to helping the poor by the provision of institutional accommodation but there were fewer that promoted ideas of self-help; one such was the Scottish Patriotic Society ‘for improving the condition of the labouring classes.’ This was not a political organisation with nationalist leanings as its title might lead one to believe but a charity whose first declared object was the improvement of the husbandry of crofters and took as its model the English Society for Improving the Condition of the Labouring Classes. The second objective was ‘to promote the Field Garden system; to improve the Cottages and Gardens of the peasantry, and the residences of the labouring classes in towns, as regards comfort, cleanliness, and sanitary regulations.’ To advance these ideas the Society published *The Industrial Magazine*, which in turn recorded the activities of bodies such as the Model Lodging-house Association and also reports from and membership details of the society’s own committees. Sub-committees included the following: on improvement of crofts; on cottage, dwelling-house, and sanitary improvements; on allotment gardens and on the improvement of fisheries.¹⁰² ‘An appeal in 1848 for building model-dwellings for the working-classes in Edinburgh’ blamed ‘the unhealthy residences of the humbler classes’ for ‘the fearful sacrifices of health and life’ in the city, stating that only three individuals had died of typhus out of seven hundred in the Charity Workhouse the previous spring, whereas sixteen hundred pauper funerals of persons dying of the infection contracted in ‘the loathsome dens of the fever districts of the parish.’ [Original Italics]. The secretary of the Patriotic Society in an address to a public meeting in the city said:

Whole streets have been swept away, and others are now in course of removal, including many hundred humble dwellings, in order to make way for railway extension; and it is a fact, that every ornamental and public improvement has been effected at the expense of those least able to contend with the difficulty of obtaining houses in lieu of those that have been demolished. The humbler classes have thus been compressed within the most contracted limits, and, in repeated instances, have recently complained that they could not find “a hole in which to thrust their heads,” owing to

excessive competition for the most miserable dens — scarcely any small houses for the working classes having been erected in Edinburgh.\textsuperscript{103}

He had recently investigated ‘the worst dwellings and districts in London’ and when he contrasted them with ‘the streaming filth of most of the closes of Edinburgh’ he was convinced that ‘a person would encounter more pestilential effluvia in one hours’ walk between the County Buildings and Holyrood Palace, than he would find in three months’ perambulation in the worst districts of London.\textsuperscript{104}

This chapter has addressed the deficiencies in housing in the Old Town but as Treble has emphasised ‘not all poverty was the product of exogenous forces which the individual was powerless to influence. It could also stem from personal failings.’\textsuperscript{105}

Treble was referring to the quantities of alcohol consumed by the poor and the working class, an aspect that is beyond the scope of this thesis. The next chapter, however, will examine the acute shortage of water and non existent sanitation, factors that were outside the control of the poor and which affected personal and communal hygiene and were crucial in promoting epidemic disease in an environment, already made dangerous by overcrowding.

\textsuperscript{103} The Industrial Magazine, II, 2, (1848), p.149.
\textsuperscript{104} The Industrial Magazine, II, 2, (1848), pp.150–151.
Chapter Seven

Water and Sewage

The main purpose of this section is to review two fundamental aspects of living conditions in the Old Town at this period, namely, water and sanitation, the latter depending to a very large extent on the former. In focusing on these two services, which, when deficient, have been shown in famine studies to be among the major causes of epidemic disease, an effort will be made to clarify the extent to which they were responsible for the high mortality in parts of Edinburgh during the 1840s. The dilemma facing the municipal authorities in respect of these services and the consequences of their scarcity or absence was not unique to Edinburgh; the rapidity with which industrial cities expanded during the first half of the nineteenth century brought with it filth and disease. Cities were simply not equipped to deal with the flood of people attracted by the prospect of employment, nor was their failure to respond surprising — the situation they faced was entirely new. Sigerist contends that hygiene and public health are dependent to a large extent on the technological knowledge available at a given time and on the concurrent organisation of the state.¹ As will be demonstrated later, in Edinburgh, where the authorities responsible for organising cleaning and maintaining hygiene were often unable to cope with the overcrowding arising from the influx of people, the disastrous state of the city’s finances was a further encumbrance in an already grim situation. In the end, it required a national movement promoting sanitation and the implementation of measures advocated by central government before any improvement took place; it was this impetus and the timely progress, not only of technology but also of statistical methods, that enabled change to take place. The British state in the 1830s and 1840s had begun to use statistical methods in a variety of ways, commissioning enquiries and reports on working conditions, living conditions, public health, poverty and the working of the poor laws. The introduction of

statutory registration of births, marriages and deaths in England from 1837 was crucial to the advance of statistics as a science, described by Eyler as the science of social reform.\textsuperscript{2} Advances in technology had a significant role in enabling cities to improve the supply of water and to build sewage schemes but it was concern about the spread of epidemic disease that had the greatest influence in promoting sanitary improvement. Physicians and administrators may have debated as to which theory, contagion or miasmas was the cause of epidemics but whatever their views, there was no disagreement about the importance of public and private hygiene, areas of concern that Edinburgh was slow to address. The extent of the task facing the civic authorities is clear from the reaction of a German visitor to Edinburgh in 1838. He described what he saw thus:

Even as matters now stand, the Augean Stable has by no means been purified from all its filth and impurities. The lower classes bring up their children with as little regard to the cleanliness and comforts of life as they paid themselves, and thus a natural indifference for such essential requisites to promote the health of the inhabitants in general, continues from generation to generation.\textsuperscript{3}

The writer in pointing out the close relationship between personal and public cleanliness was almost certainly unaware of the difficulties facing the poor in the Old Town, especially the availability of water. The next section will concentrate on the city's water supply, reviewing the historical background and describing contemporary developments. The importance of water in the maintenance of the health of the population is unquestionable but as Hardy says 'the history of water in relation to public health remains largely unexplored', commenting that 'in the first parliamentary inquiry into water quality in 1828 there is little evidence of any wider concern with the health of the general population.'\textsuperscript{4}

\textsuperscript{3} S.Naseweis, \textit{Edinburgh and its Society in 1838}, (Edinburgh, 1838), p.156. The name Naseweis, literally white nose, is almost certainly a pseudonym; the identity of the author is unknown.
Edinburgh originally took its water from wells and springs in the Cowgate and after 1598 from the South Loch. In 1681 water was brought from springs in the Pentland hills to supply five cisterns in the High Street but in time these became inadequate for the needs of the inhabitants and many more were established throughout the town; in 1850, twenty-eight of these wells were still in use. The 1681 enterprise took nine years from planning to completion, the delay arising because of fierce opposition to the special tax introduced to pay for the venture. The citizens were so antagonistic to this new rate that special measures were enacted by the Lords of Council prohibiting any interference with the works and imposing heavy penalties on any person so doing. The reluctance of the citizens to pay for the new supply provoked the following comment from Lewis, an Edinburgh businessman and councillor and one of several historians of the city’s water: ‘that a measure of such proved necessity ... should have encountered such opposition appears at this date to be rather unaccountable.’

By the middle of the eighteenth century the supply was insufficient and an Act of 1758 gave Edinburgh the right to take water from springs on the Swanston estate. Water was brought into the city from Swanston in 1762, the Council announcing, ‘that as the city is now plentifully supplied with water, not only private families, but brewers and others having occasion for the same, may be accommodated with bye-pipes if laid at their own expense.’ By 1785 demand again exceeded supply and the Council applied to Parliament for powers to take water from springs within a radius of four miles of the existing fountainhead. There was opposition from landowners but the House of Lords decided in the Council’s favour and the extra water was brought into the city.

Population growth and increased demand from the developing New Town guaranteed that the benefits of the new supply were short lived. Another historian of Edinburgh’s water, James Colston, wrote in 1890:

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2 D.Lewis, Edinburgh Water Supply: A Sketch of its History Past and Present, (Edinburgh, 1908), pp.3 & 4. David Lewis (1828–1909), a shoemaker by trade, was councillor for St. Leonard’s ward from 1863–1873 and for a time was Convener of the Water Trust.
4 Lewis, Edinburgh Water Supply, pp.5–11.
No one in the present day can realise the sufferings which the inhabitants of Edinburgh were called upon to endure, on account of a deficient water supply during the latter part of the last century, and for the first twenty years of the present one. The supply in those days, for the great mass of people, was from the public wells. ... The supply was at all times intermittent, and not at any time abundant. When a dry season occurred, the summer time and the autumn were never by any means pleasant. 9

There were few householders in the Old Town with a supply of piped water and it was only the more prosperous who could afford to employ the "caddies" or water-carriers who would queue to collect water that they would then deliver for a small fee. The poor had no alternative but to queue for hours to obtain a few gallons of water, sometimes unsuccessfully if the water ran out before their turn at the well arrived.10 The wells were filled from the piped supply only intermittently and there seems to have been little regard for the public, the supply usually being turned on at midnight. Colston described the scene at the wells in the Old Town when 'water was expected.'

From the hour of six o'clock at night until often past three in the morning, this motley line of waiters had to hang on, watching for the happy announcement that the clear crystal stream of water had begun to flow. Frequently it occurred that long before some of the expectant waiters were served the supply had ceased. This was a great hardship for the old and infirm, particularly if they were poor.11

An exceptionally dry season in 1810 brought severe shortages and the resulting complaints forced the Town Council to take action. They instructed two university professors to survey potential water sources in the Pentland hills. When they recommended that water should be taken from the Crawley springs the council asked Thomas Telford to report on the feasibility of the proposal but at this time the city was almost bankrupt and the project was shelved.12 Water scarcities continued and in the drought of 1814 the magistrates were obliged to provide for the needs of the city by

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9 J. Colston, *The Edinburgh and District Water Supply: A Historical Sketch*, (Edinburgh, 1890), p.42. Colston (1830–97), a printer and a director of several finance companies, was councillor for Newington 1865–1882 and City Treasurer in 1870. He stood unsuccessfully on three occasions for the position of Lord Provost and this account was printed for private circulation.


11 Colston, *The Edinburgh and District Water Supply*, pp.43 & 44.

sending round carts of water taken from a quarry hole near Leith Walk. The 1811 plan was resuscitated in 1817 when notice was given of a Bill to take water from the Crawley springs but once more lack of money forced the Town Council, permanently on the edge of insolvency, to drop the proposal early in 1818. The problem did not go away and in the dry autumn of 1821 it was reported that 'water was not to be got for the purpose of making porridge for breakfast, nor broth for dinner' and 'families were so frightened at the inconveniences and difficulties with which they were threatened, that they were thinking of betaking themselves to country lodgings.' This clearly was not an option for the majority of the residents of the Old Town.

This latest failure and continued severe shortages encouraged a group of leading citizens to form the Edinburgh Joint Stock Water Company, constituted by Act of Parliament in 1819, to take over the waterworks from the Town Council. The new company had seventeen board members, five from the Town Council, ten from shareholders and two university professors. They purchased the works and supply sources from the council for £30,000, paid in shares, and were given powers to raise £105,000 by a public share issue. The new supply was taken from the Crawley springs and the Glencorse burn and a reservoir was to be constructed to compensate mill owners and other manufacturers on the North Esk river into which the springs and the burn drained. By the act of incorporation the company was obliged to bring in water not only from the south Pentlands at Glencorse but also from the Black springs to the north. Disputes as to the ownership of the Black springs and a mistaken belief that there would be sufficient water from the south alone resulted in a failure to implement the full project. The partial scheme, designed and engineered by Thomas Telford, was completed in 1822 at

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16 Act for more effectually supplying the City of Edinburgh and Places adjacent with Water, 55 G. III. Cap.cxvi. (1819).
17 Colston, The Edinburgh and District Water Supply, p.36.
a cost of £209,000 and for a time the dreadful shortages experienced in previous years were prevented.\textsuperscript{20}

The respite was brief and the Water Company’s monopoly was threatened when a new company, the Edinburgh and Leith Water Company, purchased springs on the north of the Pentlands and retained Robert Stevenson of the Northern Lighthouses as engineer. They proposed to bring water to the city and to Leith but their plan was rejected by parliament ‘on the grounds that the new scheme was an interference with vested rights.’\textsuperscript{21} The brief life of the company ended when it was bought out by the old Water Company who successfully petitioned parliament in 1826 for permission to raise £118,000 of new finance and to carry out the works proposed by the now defunct Edinburgh and Leith Company.\textsuperscript{22} One historian wrote:

The rival Company being now bought up, and fairly out of the field, and the monopoly of supplying the city with water once more in their own hands, the water Company appear soon afterwards to have fallen into a state of indifference to everything but their own dividends. With the exception of supplying the inhabitants of Leith with water, they seem to have allowed the act of 1826 to become a complete dead letter.\textsuperscript{23}

Despite their inactivity and disregard for the welfare of the citizens, the company presented another Bill to parliament in 1835, a measure more concerned with shareholder dividends than the supply of water. They asked parliament to allow them to increase the water rate to 5 per cent, a move so unpopular that the weight of adverse public opinion obliged the company to issue a pamphlet defending their actions. In this document they claimed ‘there is scarcely any city in Europe where the supply is at once so excellent and so abundant, or the price so moderate.’\textsuperscript{24} They were allowed an increase in the rate but ‘the odium which they incurred in 1835 had not the least effect

\textsuperscript{20} Leslie, An Account of the Water Supply of Edinburgh, p.5.
\textsuperscript{21} Q.J., The Supply of Water to Edinburgh, p.30.
\textsuperscript{22} Act for more effectually supplying the City of Edinburgh with Water; and for supplying the Port of Leith, and His Majesty’s Dockyards at Leith with Water, 7, G. IV Cap VIII. (1826).
\textsuperscript{23} Q.J., The Supply of Water to Edinburgh, p.31.
\textsuperscript{24} Edinburgh University Special Collections, QP 94, Pamphlet 19, Case of the Edinburgh Water Company, (Edinburgh, 1835), p.3.
in preventing them from pursuing a similar course in the future.\textsuperscript{125} They returned to Parliament in 1837 with another Bill to augment income by increasing the water rate but this was defeated at the second reading. Later that year they spent £1,000 opposing a Bill sponsored by the Police Commissioners to reduce the water assessment. The Bill was passed and the company's opposition to this measure and its general inactivity renewed public antagonism and suspicion.\textsuperscript{26}

Despite the failure of the 1837 Bill the Water Company went ahead with their original plan to increase the rate but the ratepayers resisted, fighting a successful legal battle through the Sheriff Court, the Court of Session and the House of Lords. The ratepayers' objections were based on the fact that no extra water had been brought to the city since 1824, although the population had increased and Leith had been supplied with water under the Act of 1826, an Act which had also obliged the Company to introduce new springs.\textsuperscript{27} After this flurry of legal and parliamentary activity there was a period of relative peace until a drought emptied the Glencorse reservoir in the summer of 1842, leaving the mill owners without water and unable to continue operating. They went to court and the Company offered the mill owners compensation believed to be in excess of £4,500.\textsuperscript{28} The water supply was criticised by Alexander Ramsay, Inspector of Cleaning and Lighting in a report written in 1843 at the behest of the cleaning committee of the Police Commission. He emphasised the importance of a proper supply of water to enable cleaning to be carried out and he complained that 'no town in the kingdom was worse off than Edinburgh in respect of water supply.'\textsuperscript{29} He stressed the difficulty in propelling the contents of necessaries into the common sewer and the impossibility of washing and cleansing lanes, closes, alleys, pavements and causeways because of the want of a requisite supply of water.\textsuperscript{30} The renewed public disquiet in

\textsuperscript{25} Q.J., \textit{The Supply of Water to Edinburgh}, p.32.
\textsuperscript{26} Q.J., \textit{The Supply of Water to Edinburgh}, p.32.
\textsuperscript{27} Q.J., \textit{The Supply of Water to Edinburgh}, p.35.
\textsuperscript{28} Colston, \textit{The Edinburgh and District Water Supply}, pp.58 & 59.
\textsuperscript{29} A.Ramsay, \textit{Report on Minute of Cleaning Committee of 21 October and Minute of General Board of Commissioners of Police of 10 November 1843}, (Edinburgh, 1843), pp.10 & 11.
1843 forced the Company to act, bringing yet another Bill before parliament, promising immediate action.31

There now developed an almost farcical situation with the formation of a rival company but any possibility of the new company achieving its stated aims evaporated when Learmonth, the chairman of the old company stepped in 'with that strategical cleverness which characterised his subsequent career in the railway world' and purchased the estate of Colzium for £12,000.32 The rival concern now had no water source and was forced out of business, accepting £1,500 as compensation. The Water Company arranged to buy the estate of Colzium from Learmonth at the price he had paid for it and proceeded with the Bill.33

The latest Bill proposed to use the springs at Colzium and Harperrig and to increase the water duty to ten pence in the pound, but met with opposition from a committee of citizens led by Duncan McLaren and supported by the council.34 The company, in evidence to the Parliamentary Committee, stated that the project was essential because of 'the deficient quantity and infamous quality of the supply of water to Edinburgh,' emphasising also 'the evil effects, in a sanitary point of view, of a deficient supply of water to the poorer classes of Edinburgh.'35 These assertions were in stark contrast to the company's claims in the pamphlet of 1835, (see above and footnote 35). The citizens' committee was successful, the Parliamentary Committee rejecting the Bill to prevent the Company having a monopoly. The farce of claim and counter claim continued and in 1845–1846 yet another company, the Edinburgh and Leith, was formed largely at the instigation of the Inhabitants' Committee which had continued to

32 Q.J., The Supply of Water to Edinburgh, p.34.
33 Q.J., The Supply of Water to Edinburgh, p.35. James Learmonth (1789–1858), the last Lord Provost of the unreformed council, owned the Dean estate and was the promoter and financial backer of the Dean Bridge (1831) and chairman of the Edinburgh and Glasgow Railway Company.
34 Duncan McLaren (1800–86), dissenter and anti–Corn Law protester, was Lord Provost 1851–54 and a Liberal MP (1865–81).
monitor the water company and was becoming increasingly concerned about the supply. In 1846 both companies went to Parliament, both Bills were thrown out, the Parliamentary Committee expressing regret 'that this should have been the result of the presence before them of these two Companies, because they [the Committee] are convinced of the great importance of the supply of water to that great city.' The Committee's statement and its rejection of both bills signalled that repeated recourse to Parliament was not going to solve the problem of Edinburgh's water supply — the warning was ignored and the old water Company was again before parliament in 1847 with a new set of proposals. On this occasion, in a changed administrative approach, the matter was delegated to the Commissioners of Woods and Forests who arranged to meet in Edinburgh to take evidence.

An increasing awareness of the importance of hygiene and public health, prompted by the work of Edwin Chadwick, was perhaps the reason that parliament adopted this method of dealing with the Edinburgh water supply dilemma. The First Commissioner of Woods and Forests, Lord Lincoln, introduced public health measures emphasising sanitary improvement in 1846 and 1847. His Whig successor, Lord Morpeth, appointed First Commissioner with a Cabinet seat in Lord John Russell's first administration formed after the fall of Peel, introduced public health measures in 1847 and 1848, the success of the latter; it has been suggested, assisted by fear of cholera. Morpeth had appointed Chadwick his colleague in 1847, thus giving him his full support. For the first time Edinburgh's water supply was about to be assessed by a government department led by a minister who was convinced of the importance of sanitation in the prevention of epidemic disease.

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36 Lewis, Edinburgh Water Supply, p.15.
The application was approved by the Commissioners who expressed the hope that their decision would put an end to competition in the supply of water to the city. There were two important clauses in the Act: the Company was required to supply sufficient pure and wholesome water to meet the needs of all willing to pay the water rate; secondly, the supply was to be continuous and at a pressure great enough to reach the top storey of the highest house. It does not have to be said that these stipulations did not mean much to those who could not afford the water rate nor was there any reference to the amount of water to be supplied, a topic which had been the subject of questioning by the Parliamentary Committee in 1843. In evidence to this committee, Jardine, the civil engineer on the water projects of 1810–11, 1819–23 and 1842–43, gave details of the quantity of water supplied at different periods during the first half of the nineteenth century. On completion of the works of 1819, the average daily amount supplied to the city was nineteen gallons per person, assuming that all the water was for domestic consumption. In 1847, the amount available was ten gallons daily per person, no account being taken of water used in manufacturing, street cleaning, fire control, steam engines, hotels, brewing, stables and in supplying water to ships at Leith. It is clear that the estimate of ten gallons was optimistic since it has been calculated that at least half was not available, leaving a maximum of five gallons for domestic use. It was calculated that twenty-seven and a half gallons per person would become available for private use on completion of the 1847 scheme, again assuming that all would be for private use, which was of course totally unreasonable. In 1843 Jardine estimated that sixteen gallons was required for domestic purposes whereas his 1826 estimate had been ten gallons, the increase he attributed to greater domestic usage after the cholera epidemic of 1832. If half of the supply was used commercially the actual amount available for domestic use was about fourteen gallons, two gallons less than the amount he considered an absolute minimum.

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The latest water scheme, therefore, was not going to supply the quantity of water necessary for personal and domestic use, to establish modern sanitation or to allow for improvements in public hygiene. The extra water made available on completion in 1852–53 was no doubt a boon to those with piped supplies but would not have been noticed by those in the Old Town who had to carry any extra water to homes in high tenements. For at least eight decades Edinburgh’s water supply had been totally inadequate for the needs of a developing city, partly because of the conflict between public good and private gain and partly because of the city’s insolvency at a critical time. During the 1830s the city was bankrupt, parliament in 1838 passing an Act intended to draw a line under the city’s financial problems which were not finally resolved until 1856. The Act stated that ‘...the affairs of the City of Edinburgh have for some years past been in a state of great embarrassment....’ and was intended to bring order to the city’s financial affairs and relief to the city’s creditors but eighteen years passed before the matter was satisfactorily concluded.

The city’s financial embarrassments, aggravated by expensive litigation over water, may have hindered improvements in the water supply, but there were more immediate reasons why the Old Town supply remained poor. The first was the introduction of water closets into New Town dwellings from 1824, an undoubted improvement but as a result the wells on which the Old Town depended were deprived of two of their three supply pipes. This ‘caused serious inconvenience to the poorer folk from the totally inadequate amount of water available, a situation aggravated after dry seasons.’ That this happened and was allowed to continue was perhaps due to the difficulty in implementing civic improvements because of the existence of two administrative bodies

47 Act to Regulate and secure the Debt due by the City of Edinburgh to the Public; to confirm an Agreement between the said City and its Creditors and to effect a Settlement of the Affairs of the City and the Town of Leith, 1 & 2 Vict.55 cap.55.1837–38.
in the city, the Town Council itself and the independently elected Police Commission. The Poor Law Act of 1845 increased the complexity of Edinburgh's administration when the Parochial Boards of the City, Canongate and St Cuthbert's parishes were established, organisations which, among other duties, were responsible for the medical care of the poor but also had nuisance removal powers.

The Council and the Police Commission had different responsibilities and in the case of the Commissioners the area they represented and controlled was quite different to that over which the Town Council had jurisdiction. The Police Commission was not only responsible for watching, cleaning and lighting the Ancient and Extended Royalty but also the separate burghs of barony, Canongate, Calton and Easter and Wester Portsburgh and the developing suburban areas. This arrangement was not peculiar to Edinburgh, similar systems operated in many cities including Glasgow. However in 1846 Glasgow merged the two administrative bodies, replacing its Police Commission with a Police Board, incorporated in the Town Council. The growth in the population of Glasgow, deteriorating living conditions, epidemics and the eventual incorporation of smaller outlying burghs by the Extension Act were the catalysts for change.49

The Municipal Reform Act (Scotland), (1833) did away with the old system whereby the incorporated trades appointed the Council. The new franchise changed the electorate in Edinburgh — five wards were created with thirty-one elected members forming the new council. Shortly after the Act came into force, a Commission was appointed to consider combining the Police Commission and the Council and although the decision was in favour of change the city's financial problems proved an insuperable obstacle. The Council made another attempt to rationalise the administrative structure, bringing a Bill before Parliament in 1848 proposing an amalgamation with the Police Commission. The Bill was resolutely opposed by objectors who claimed that 'the heavy burdens of the city would depreciate their properties and that the Town Council would become “an

unwieldy and unmanageable body, utterly incapable of performing the multifarious duties that would devolve upon it.”

50 The Police Commission continued to function independently until an Act of 1856 extended the municipal boundaries, abolished the separate jurisdictions of the Canongate, Calton and Portsburgh (Easter and Wester) and transferred the powers of the Police Commissioners to the Council. 51 Until its abolition the Commission, as well as other duties concerned with lighting and watching, was responsible for keeping the city clean and had a somewhat tenuous public health role. It is important to understand how this organisation functioned and how its relationship with the council affected matters in the sphere of health and sanitation and it is against this background of dual control that the role of the Police Commission and its employees will be analysed.

The Police Commission was constituted by Act of Parliament (1771) to meet the needs of new suburbs lying outwith the Ancient and Extended Royalty and therefore not in the remit of the Town Council. This first local statute took in the Southern districts and a second Act (1772) covered the Canongate, Pleasance and Leith Wynd with other districts and duties included in Acts of 1805 and 1812. 52 Qualified representatives of the six wards in the city, its suburbs and surrounding districts elected the Commissioners. These wards included districts as distant as Canonmills, Water of Leith, Fountainbridge and Portobello but a special clause exempted the Palace and Sanctuary of Holyroodhouse from both police jurisdiction and assessment. 53 The Act of 1771 gave the Commissioners the right to assess householders and to collect money from ratepayers to finance the various tasks prescribed for them, which included watching, cleaning and lighting. 54 The importance of watching compared to cleaning and lighting is clear; the number of men employed watching in 1848 was 304, whereas

cleaning and lighting required only 157 men, with more employed in winter, reduced to 130 in summer.\textsuperscript{55} A series of Acts and amendments from 1822 empowered the commissioners to administer cleaning, lighting and watching in both the Ancient and Extended Royalty and the suburbs and, although Canongate, Calton, Easter and Wester Portsburgh remained separate burghs, they were given the right to appoint their own commissioners to the new enlarged body. The Commission’s duties were discharged through the Cleaning Committee who were responsible for day to day management through a permanent official, the Inspector of Cleaning and Lighting, ‘who was to have full power and authority to the General Commissioners.’\textsuperscript{56} There were clauses in the 1822 Act that gave the Commission additional powers; for example, keepers of lodging houses were obliged within six days to give notice of contagious diseases to the police or to a medical practitioner. They were authorised to construct proper main drains or sewers; the proprietors of flats were to be allowed to erect one waste or foul water pipe communicating with the common sewer and to bring a mains water pipe into the tenement. If these measures implied that the inspector was a prototype sanitary officer, in truth he was ‘far removed from being a municipal official with public health responsibilities.’\textsuperscript{57}

If the Commission’s powers under the 1822 Act were rudimentary as far as health was concerned, ten years later their duties were becoming more specific and hint at worries about public health, or at least the prevention of the spread of epidemic disease. There were clauses in the 1832 Act obliging the Commission to carry out certain tasks relating to health and sanitary improvement; they were required to examine meat and fish to attest that it was wholesome; they were to ensure that lodging house keepers informed the police office, public dispensary or medical practitioner of any person confined to bed with illness for forty eight hours and if any contagious or infectious disease was


\textsuperscript{56} Act for Watching, Cleansing and Lighting the Streets of the City of Edinburgh, G.IV Cap.Lxxviii.1822.

diagnosed the lodging house was to be washed and fumigated. The Commission was granted permission to build privies and necessary houses, the disposal and sale of ashes and dung were vested in the commissioners; they were to ensure that common stairs were cleaned weekly, the keeping of pigs in dwelling houses was forbidden and the right of the commission to build drains and sewers was again stressed.\textsuperscript{58} It is questionable how well the commission was able to ensure that the cleaning staff of at most 170 men carried out their allotted tasks under the Act. Financial stringency, the chronic water shortage in the dirtiest areas of the city and the practical impossibility of inspecting lodging-houses, examining meat and fish, cleaning common stairs, closes and dwelling houses made many of the clauses in the 1832 Act unworkable.

The work involved in building, maintaining and cleaning privies and necessaries, as laid down by the Act, was not only time consuming but was also beset by administrative and legal difficulties. A frequent complication arose from the location of public conveniences, which were considered to be “nuisances” by those living in their vicinity. The dislike was not merely on aesthetic grounds for there was a genuine but erroneous fear that they generated disease through smells and effluvia, thought by many to be the source of the miasmata — the origin of fever. In a city with few internal water closets, the provision of conveniences was essential for sanitary improvement but was difficult to achieve. The Inspector of Lighting and Cleaning, Alexander Murray, complained in 1847 that ‘the want of public necessaries is another grievance, which will not be removed till the Police Commissioners are invested with powers more extensive than they at present possess.’\textsuperscript{59} Murray was highlighting the difficulties experienced in procuring land and obtaining permission to build conveniences despite having powers laid down by statute. He contrasted the situation in Liverpool and Manchester where the council had the right to erect conveniences ‘in such situations as they deem proper for

\textsuperscript{58} Act for altering and amending certain Acts for regulating the Police of the City of Edinburgh and adjoining districts, W.IV.LXXXVIII.1832.

\textsuperscript{59} A. Murray, Nuisances in Edinburgh and Suggestion for the Removal thereof addressed to the General Commissioners of Police, (Edinburgh, 1847), p.13.
the accommodation of the public.\textsuperscript{60} The lack of public necessaries added to the accumulation of filth in public places and poor maintenance of existing necessaries simply aggravated the problem. The presence of "nuisances" from badly maintained toilets was a common complaint, a problem that had not been solved in 1865 when the first report on the city's sanitary condition was published. The author, Dr. Henry Littlejohn, described the obstacles that prevented the introduction of conveniences into the rambling and decrepit tenements of the Old Town and the probability that conveniences placed outside houses for the use of the occupants would soon come to resemble the public conveniences in the city, where it was "a matter of great difficulty to preserve anything like decent cleanliness; the air becomes tainted; what is intended as a public benefit is very apt to become a nuisance to the surrounding neighbourhood."\textsuperscript{61}

The minutes of the Cleaning Committee reveal the financial constraints under which the Commission had to function. The Inspector's work involved not only supervising the maintenance of conveniences but also arranging for the sale of manure and its transport to dung depots on the outskirts of town. The profits from this trade helped to defray the committee's running costs; in 1841, manure sales were £10,200 while the committee's expenditure was £12,000 of which £6,000 was spent on manure transport, £3,700 on cleaning staff wages, £600 on building and maintaining public necessaries, £500 on street watering and £200 on the rental of manure depots.\textsuperscript{62} The greatest cost was incurred in transporting dung to storage sites whereas only £600 was spent on conveniences for the public. The introduction of a closed system of sewage disposal as advocated by Edwin Chadwick would inevitably result in the loss of the income from manure sales and create serious financial problems for the Commissioners.\textsuperscript{63} An increase in rating would prove highly unpopular and this consideration may have delayed the building of sewers. In Edinburgh's case, if one ignores the possibility of

\textsuperscript{60} Murray, Nuisances in Edinburgh, p.14.
\textsuperscript{61} Littlejohn, Report on the Sanitary Condition of the City of Edinburgh, p.79.
\textsuperscript{62} Edinburgh City Archive, Minute Book of the Cleaning Committee, 17 August 1842.
financial self-interest, the introduction of closed sewers was delayed because of the inadequate water supply. The *Sanitary Report* stressed that 'proper supplies of pure water' were essential if house cleansing and sewerage were to work efficiently, but also emphasised that copious amounts of water were required to flush the new closed system. The difficulty facing the Commissioners was not made any easier by the *Sanitary Report* in which the Edinburgh system of field irrigation by sewage was promoted as an example of good practice. This use of sewage was entirely dependent on open drains and was the reason for the so-called foul burn irrigation controversy, a dispute that will be discussed later in the chapter.

Sewage and rubbish disposal occupied a great deal of the Cleaning Committee’s time but during epidemics there was a distinct change to an emphasis on cleaning and whitewashing of houses, stairs and closes where fever was present. At the height of the fever epidemic in 1843, following meetings of the Committee and the Police Commission, the Inspector was asked to prepare a detailed report for publication and circulation. The committee had discussed extending limewashing to include the houses of the poor to prevent the spread of the epidemic and so great was the general anxiety that the Police Commission were being pressurised to request parliament 'to consolidate and improve the existing Police Acts.'

The Inspector’s report stressed the importance of an adequate supply of water for whitewashing the insides of houses and for ‘thoroughly cleaning the lobbies and staircases of the houses situated in lanes, close, alleys and other confined places, together with the pavements and causeways opposite to or connected therewith.’ He declared that no town in the United Kingdom was worse off than Edinburgh in respect of its water supply and explained the difficulty that people faced, living in tenements at a distance from wells, with no alternative but to carry water to their homes up several

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65 Minute Book of the Cleaning Committee, 21 October 1843; Minute Book of the Police Commission, 10 November 1843.
66 Minute Book of the Cleaning Committee, 10 November 1843.
flights of stairs. This was ‘a great disincentive to clean habits’ and the shortage of water was a continual hindrance to his department who were required by law to wash and cleanse lanes, closes, alleyways, pavements and causeways. At an earlier meeting, the cleaning committee had discussed the water shortage and a proposal was made to obtain water from other sources to allow cleaning to be carried out and after representations by the committee, the Water Company was prevailed upon to open three or four disused wells in the Grassmarket and the Cowgate.

The Inspector assured the Committee that the cleaning and limewashing of houses and closes where fever was epidemic was important and recalled the occasion in 1838 when several families in Fleshmarket Close had contracted fever, the spread of which was controlled only when the building was cleaned and limewashed. He complained that overcrowding and increasing squalor made cleaning and fumigating difficult, if not impossible, and promoting basic hygiene was equally difficult. In the end Chadwick’s Sanitary Report convinced central government of the importance of proper sanitation and an adequate supply of water but Scottish antagonism delayed implementation of many aspects of sanitary reform in Edinburgh.

In his report, Chadwick had no doubt that dirt and disease were cause and effect claiming:

…other reports ascribe a large proportion of the comparative health of the population to advantageous circumstances, in respect to the supplies of water. …it will be manifest that for an efficient system of house cleansing and sewerage, it is indispensable that proper supplies of pure water should be provided, and be laid on in the houses in towns of every size. No previous investigations had led me to conceive the great extent to which the labouring classes are subjected to privations, not only of water for the purpose of ablution, house cleansing, and sewerage, but of wholesome water for drinking, and culinary purposes.

67 Ramsay, Report on Minute of the Cleaning Committee, pp.1–19. This pamphlet is filed in the Minute Book of the Cleaning Committee, 1840–44.
68 Edinburgh City Archive, Minute Book of the Cleaning Committee, 12 & 14 October 1843.
Flinn maintained that the *Sanitary Report* was first and foremost concerned with the prevention of typhus, 'the product of squalor, insanitation and overcrowding, and a perquisite of working-class housing.' Chadwick was greatly influenced by the observations of physicians who described what they saw and experienced in the course of their day to day work; the condition of the wynds and closes of the Old Town was outlined to the sanitary enquiry by an Edinburgh surgeon, William Tait:

The dwellings of the poor are remarkable for their generally uncomfortable appearance, and I attribute this in most instances to a deficient supply of water, necessaries, and such like conveniences. There are no receptacles for filth of any description, and it is either accumulated in the stairs or dwellings themselves, and the stairs are scarcely ever washed. And how can it be otherwise, seeing that the poor have to travel a considerable distance for water, and afterwards carry it up five, six or seven stories.

Tait, a medical practitioner and police surgeon, was well qualified to comment on the sanitary condition of Edinburgh — he had prefaced his observations on the foul water irrigation controversy of 1839–40 by explaining that they were based ‘on a personal survey of different districts of the city, and an examination of their comparative salubrity.' However, it is when Tait discusses miasmata in relation to the fetid irrigation controversy that the dilemma facing those in authority becomes clear. Doctors and municipal officials alike were completely in the dark as to the cause of many diseases and more specifically of fever, the age-old debate between those who believed in contagion and the supporters of the miasma theory adding to their confusion.

The fetid irrigation or foul water controversy of 1839–40 arose from the use of the so-called foul burns of Edinburgh into which the city’s sewage flowed via natural drainage channels; Leith, the New Town, the west and south-western suburbs drained to the Water of Leith or one of its tributaries; the southern suburbs drained to the Jordan or

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Pow Burn; the Nor' Loch, the Cowgate and the Meadows, the three valleys of the Old Town, drained into the foul burn and thence to Craigentinny. Farmers outside the city used the effluent to manure and irrigate their fields and by this method were able to harvest as many as four or five heavy crops of grass each year. The meadows and pastures fertilised in this manner lay at Craigentinny, Lochend, Dalry and Roseburn where land rentals rose from £3-4 per acre to £25-50. In 1832 the first attempt by the Police Commission to enclose the foul burn failed, financial interests in land, improved as a result of irrigation, prevailed. In 1839, when sanitary matters were increasingly topical and Chadwick's enquiry was underway, the Police Commissioners considered that the time was ripe for another effort to enclose the foul burn.

The controversy began in March 1839 with an anonymous letter to the Lord Provost in which the writer blamed the increase in fever cases to the use of sewage as manure in fields close to the city. It is thought that it was written by William Drysdale (1781-1847), later Sir William, a member of both the Town Council and the Police Commission. Tait referred to Drysdale as 'the indefatigable chairman of the committee', that is, the Commissioners of Police, a body who had published a volume of papers on the dangers of fetid irrigation. This was not the first time that the drainage of the Old Town had been the subject of meetings and pamphlets; in 1835, under the chairmanship of the Lord Provost a meeting was called in the Council Chambers to promote 'the formation of drains for the Old Town.' A report of this meeting, at which a committee was formed to lobby for a drain to be constructed along the Cowgate, concluded that its absence had contributed to the depreciation in value of Old Town property and was harmful to the health of the community. The committee included the Lord President, the Dean of the Faculty, Principal Baird, the Dean of Guild.

77 W.Tait, *An Examination of the Statements contained in the Papers relating to the Fetid Irrigations around the City of Edinburgh*, (Edinburgh, 1839), p.3.
and the Convener of the Cleaning Committee of Police but despite the presence of so many men of influence the proprietors of the irrigated lands were able to defeat the proposal.\(^{78}\) The financial implications apart, the controversy was fought essentially between the protagonists of the theory of miasma as the cause of fever and those who were contagionists.

The foul burn had been used by farmers to manure and irrigate open fields near the city since the eighteenth century but now Drysdale was concerned that ‘the nauseous exhalations arising from these fetid marshes’ was responsible for the increase in fever in the city. This irrigation method had been criticised in 1809 by Roberton when he described the ‘collections of filth, chiefly from the termination of the common sewers, which contaminate the atmosphere’ in the fields that lay to the east of the city.\(^{79}\) The announcement in the *Scotsman* that Sunday, 3 March 1839 was the day set aside in all churches and chapels in Edinburgh and neighbourhood to collect for the Fever Board, included the warning ‘that Epidemic Fever has greatly increased during the last two years and that, while it has increased throughout all classes of the community, its malignity has also been greater, the mortality having risen from one in twenty to one in six of those affected.’\(^{80}\) The following week the *Scotsman* referred to the anonymous letter to the Lord Provost which attributed the increase in fever to effluvia from the neighbourhood marshes. The writer claimed that these were worse than ordinary marshes, being made from ‘the irrigation of the ground by the water and filth of the common sewers of Edinburgh’ and he recommended that all ‘the foul-water and filth of the common sewers of the town be conveyed underground to the sea.’\(^{81}\) On 27 March, in an account of a meeting of the Police Commission, headed ‘Old Town Drains’ the *Scotsman* reported that a main drain 830 yards long had been laid from end to end of the Cowgate at a cost of £1,900 raised by voluntary subscription. The article claimed that

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\(^{78}\) G.Grant, *Report of a Sub Committee named at a Meeting to Promote the Draining of the Old Town of Edinburgh*, (Edinburgh, 1835), pp.4 & 5.

\(^{79}\) J.Roberton, *A Treatise on Medical Police and on Diet, Regimen etc.*, (Edinburgh, 1809), p.278.

\(^{80}\) *Scotsman*, 2 March 1839.

\(^{81}\) *Scotsman*, 9 March 1839.
'on the authority of medical gentlemen' the health of the district had been greatly improved and that 'during the recent prevalence of fever the cases have been much fewer than at former periods...'.

On April 3, the newspaper reported the proceedings of the Town Council under the heading 'Foul-Water Irrigation' at which Mr Drysdale's proposal that the practice of irrigation using the products of the sewers should be forbidden was debated. Drysdale told the meeting 'since Edinburgh possessed no dense manufacturing population, it ought to be one of the healthiest, if not the very healthiest, city in Europe; and so it was always considered.' He quoted Arnott and Southwood Smith who had showed that drains, filth and fever were connected and that "poisonous exhalations arising from putrid vegetable and animal matter" were linked to the prevalence of fever. The timing of these letters, announcements and meetings gives the impression that a concerted effort was being orchestrated to ban the use of sewage on the outskirts of the city.

The controversy grew with the opposing parties publishing pamphlets supporting their cause. The Police Commissioners produced a ninety five page collection of papers and letters supporting their claim with an introduction written by William Drysdale. There were articles on marsh effluvia, as described by medical men, a series of 'Opinions of various Medical Gentlemen on the Effects of the Fetid Irrigations of Edinburgh, in generating or fostering disease, Opinions on Holyrood as a Place of Residence, Evidence relative to the Injurious Influence of the Irrigated Lands' and an Appendix in which Medical Men gave their opinion of the preceding documents. Those who did not object to the irrigation system replied with a series of pamphlets, one written by Tait was highly critical of the miasma theory and its supporters was called An Examination of the Statements contained in the Papers relating to the Fetid Irrigations around the City of Edinburgh and was a point by point rebuttal of the arguments put forward band his supporters. Tait wrote

82 Scotsman, 27 March 1839.
83 Scotsman, 3 April 1839.
84 Edinburgh Central Library, Papers relating to the Noxious Effects of the Fetid Irrigations, (Edinburgh, 1839), pp.1–95. This was published by authority of a committee of the Commissioners of Police.
...we may state once for all, that nothing certain is known of the true nature or origin of this bugbear marsh miasma. Its appearance, its physical and chemical properties have never been detected. It is indeed probable, that the effects imputed to it, may at no distant day be referable to some other cause. Being thus in total ignorance of its real nature, it is not surprising that medical men themselves should differ in opinion regarding it. 85

Tait advanced evidence which he claimed proved that the type of effluvia said to be the cause of fever might be unpleasant but was actually harmless. He referred to 'those Augean stables of filth and nastiness' between the back of the Castle and West Port, where stood a tannery with associated dung tanks of a considerable size that imparted to the atmosphere 'volumes of offensive odours' and yet there was no proof that this locality was more unhealthy than any other district in Edinburgh.86 Tait concluded by criticising the dunghills maintained by the Police Commission on the outskirts of the city, saying that these were a greater nuisance than the irrigated fields and that the state of filth in Blackfriar's Wynd was so appalling that 'every one accustomed to inhale the polluted atmosphere of these confined and dirty closes would consider it a privilege to be permitted to breathe that of the irrigated meadows.'87 The agitation had no effect and by 1865 the total area irrigated and manured in this way had increased from less than 200 acres to over 350 acres.88

If the foul burn controversy in the end solved nothing it did at least serve to highlight the sanitary deficiencies in the Old Town and the shortcomings in the city's efforts to deal with the accumulation of filth in the stairs, closes and wynds. In mitigation it can be argued that the Town Council and the Police Commissioners could not have envisaged

85 Edinburgh Central Library, W.Tait, An Examination of the Statements contained in the Papers relating to the Fetid Irrigations around the City of Edinburgh, (Edinburgh, 1839). Other pamphlets in Edinburgh Central Library include Foul Burn Agitation Statement explaining the Nature and History of the Agricultural Irrigation near Edinburgh, (Edinburgh, 1840) and R. Forsyth, Stated to be a refutation of the unfounded and calumnious misrepresentation on that subject in a pamphlet published in the name of a committee of police in which the ancient and beautiful capital of Scotland is falsely described as a residence unsafe to the health of its inhabitants, (Edinburgh, 1840); Tait, An Examination of the Statements contained in the Papers relating to the Fetid Irrigations, p.8.
86 Tait, An Examination of the Statements contained in the Papers relating to the Fetid Irrigations, pp.23 & 24.
87 An Examination of the Statements contained in the Papers relating to the Fetid Irrigations, p.14.
the increase in population in the Old Town with the much greater demands on an already scant water supply and a non-existent sanitary system. On the other hand, the improvement schemes from the 1820s could have only one outcome since they involved the removal of scores of tenements without plans for replacement housing and it was the incompetence of the civic authorities and the water companies that was responsible for the poor water supply and the lack of any form of sanitation in the tenements into which the displaced people crowded. After 1845 and the establishment of the Poor Law Board of Supervision, the Parochial Boards took on duties which clashed with those of the Police Commissioners, particularly where cleaning was concerned. Disagreements as to jurisdiction were common and were partly resolved only in 1848 when in the time of cholera a meeting of representatives of the Police Commissioners, the Parochial Boards and the two Royal Colleges agreed that the Parochial Boards should undertake internal cleaning and the Police Commissioners external cleaning.\textsuperscript{89} This meeting is significant in that it involved physicians consulting with the local appointed administrators and those from central government, implying that the control of epidemic disease was an issue requiring a contribution from the medical profession. The debate as to whether destitution rather than poor sanitation was the prime cause of epidemic disease occupied many physicians at this time and the chapter that follows will explore the epidemics of acute infections and the response of the authorities to these recurrent outbreaks.

\textsuperscript{89} Minutes of the Cleaning Committee, 1844–49, 8 November 1848.
Chapter Eight

Epidemic Disease

This chapter is concerned with the epidemics of communicable disease which affected Edinburgh in common with other British towns and cities during the 1840s. The first section will focus on fever, examining its history, theories of contagion and miasmas and analysing the epidemics which occurred in the city. The responses of the medical profession, the Town Council, the Police Commission and the Fever Board will be explored and compared to the reaction in other cities. The second section will concentrate on the less terrifying and, perhaps as a result, the less well documented epidemics of measles, smallpox, whooping cough, scarlet fever and influenza which appeared in the city at regular intervals with identifiable increased mortality. The third section will concentrate on the cholera epidemic of 1848–49, utilising a database of cholera returns maintained by the Royal College of Physicians of Edinburgh to examine the effect of this scourge on a population already fearful of disease.

Fever

The main purpose of this section will be to examine the nature and severity of the fever epidemics which affected Edinburgh in the 1840s and to look at the measures taken by the medical and civic authorities to deal with the threat. The impact on the city of what is now known to be three distinct diseases will be addressed, but first the history and clinical features of these three, typhus, relapsing fever and typhoid fever, will be described. To place mid nineteenth century medical belief and practice in context, the development of fever theory will be reviewed, referring to the teachings of William Cullen in the eighteenth century and examining the concepts of disease current amongst Edinburgh physicians in the 1840s. In this review the debate between contagionists and miasmatists will be considered, referring to the impact of this controversy on poor law and sanitary reform. This appraisal will illustrate a number of aspects that have a bearing
on the responses of the medical profession and the civic authorities to the management of the fever threat. Contemporary descriptions of outbreaks will be analysed, identifying the diseases responsible and the current literature on these infections and their epidemiology reviewed. A detailed and accurate assessment of morbidity and mortality during these episodes is impossible because the statistics required for such an exercise are not available. Figures for sickness recorded by dispensaries and hospitals give an impression of the severity of these outbreaks but, for reasons that will be explained later, do not reveal the whole picture. Similarly, incomplete data prevent a detailed analysis of overall mortality although the records of St Cuthbert’s and Greyfriars’ burial grounds will be used to illustrate aspects of the repeated mortality crises. The reasons why Edinburgh, compared to other cities, was particularly slow to make use of the new and increasingly popular science of statistics have been examined in an earlier chapter.

In 1948 the historian, Thomas Ferguson, wrote: ‘it is not too much to say that “fever” dominated Scottish life in the first half of the nineteenth century.’\(^1\) It is debatable if this was entirely true of rural society but there is no doubt that by the 1830s urban Scotland was in dread of fever which was endemic in the slums of Glasgow and Edinburgh, at frequent intervals becoming epidemic with massively increased morbidity and mortality. The fear that fever might spread from the poor in the city slums to infect the respectable middle classes proved to be a powerful incentive for good works and charitable giving. Although by the end of the 1850s fever epidemics were largely in the past the apprehension lingered well into the second half of the century. As late as 1865 Dr Henry Littlejohn (1826–1914), Edinburgh’s first medical officer of health, still felt it wise to warn people of the potential danger, writing in his first report on the health of the city, ‘fever is never entirely absent from Edinburgh’ and claiming that ‘it would yearly be manifest in the epidemic form, did not the poor enjoy unusual facilities for the early treatment of the sick, and the removal of infected persons to our large hospital.’\(^2\)

\(^2\) H. D. Littlejohn, *Report on the Sanitary Condition of the City of Edinburgh*, (Edinburgh, 1865), p.27. Littlejohn was appointed as the city’s first medical officer of health in 1862.
This first section will concentrate on the recorded experience of Professor Robert Christison, (1797–1882), who was introduced in the first chapter. He held the chair of materia medica in Edinburgh for forty five years, (1832–1877) and his experience of epidemic disease encompassed the years from 1819 to the 1860s. His knowledge of fever will be explored using material from two sources: his concluding lecture of the academic year 1835–36, in which he drew on clinical details of cases treated in his wards in the infirmary to illustrate the nature of fever and secondly, a paper read to the Medico–Chirurgical Society of Edinburgh in 1857 on the fever epidemics of the previous forty years, beginning with the epidemic of 1817, a paper subsequently published in the Edinburgh Medical and Surgical Journal. These two commentaries will be used to illustrate contemporary knowledge amongst the Edinburgh medical fraternity and to compare their beliefs and teaching with those of physicians in other British and foreign medical centres.

The epidemic of 1817–1820 was Christison’s first experience of fever, an epidemic he later attributed to relapsing fever, insisting as late as 1857 that the correct name was inflammatory fever, ‘the causus of older authors or synocha of Cullen.’ A contemporary nosology explained that synocha was derived from the Greek, meaning ‘to continue’. Continued fever was recognised as different from the intermittent and remittent forms of fever, now known as malaria, and described by William Tait in his pamphlet (1839) on Edinburgh’s foul water irrigation in the following way: ‘malaria and marsh miasm are used synonymously, and are meant to designate an active morbific agent in the production of an intermittent and remittent form of fever, but whose physical properties are entirely unknown.’ In Edinburgh for most of the first half of the nineteenth century continued fever was assumed to have an entirely different origin from malaria and it is

3 J.D.Comrie, History of Scottish Medicine, (London, 1932), pp.616 & 617.
likely that Christison had been taught this principle. Christison’s interest was already evident in 1819, writing his doctoral thesis on continued fever and his experience increased through the epidemics of the 1830s and the 1840s. Fever was endemic in Edinburgh during the first part of the nineteenth century but major epidemics broke out at regular intervals. In his 1857 retrospective Christison described these epidemics, specifying the diseases responsible and his analysis is shown in table 8.1.7

<table>
<thead>
<tr>
<th>Year</th>
<th>Disease Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>1817–20</td>
<td>Relapsing fever, Typhoid fever</td>
</tr>
<tr>
<td>1826–29</td>
<td>Relapsing fever, Typhoid fever</td>
</tr>
<tr>
<td>1832</td>
<td>Not specified</td>
</tr>
<tr>
<td>1837–39</td>
<td>Typhus</td>
</tr>
<tr>
<td>1841–44</td>
<td>Typhus, Relapsing fever</td>
</tr>
<tr>
<td>1847–49</td>
<td>Typhus, Relapsing fever, Typhoid fever</td>
</tr>
</tbody>
</table>

Table 8.1. Christison, 'On the Changes in the Constitution of Fevers.'

Christison could make these judgements in 1857 because considerable progress had been made in the identification of the three infections previously included in the umbrella term “fever”. Although advances in bacteriology lay in the future the correlation of clinical observations and post mortem findings had made it possible for the three to be differentiated and the history of these developments will be reviewed later. Firstly, however, the theories of fever developed in the eighteenth century by Cullen and others were still extant in nineteenth century Edinburgh and this aspect will now be addressed.

Theories of Fever

As Tweedie observed in 1862, the *continuae* of Cullen or continued fevers were until ‘a comparatively recent period’ included in one class, under the generic term typhus.8 In Cullen’s classification these were fevers which were not produced by marsh miasmata and were without remissions or exacerbations. He defined typhus as a contagious, putrid

fever divided into four species, typhus petechialis, mitior, gravior and icteroides. Synochus he defined as a contagious mixed fever composed of synocha and typhus, at the outset of the illness taking the form synocha but ending as typhus. The enduring influence of Cullen's teaching is apparent in the following extract from Christison's review of fevers written in 1858:

So, too, typhus, a very frequent form in 1843–44, showed itself in its most characteristic adynamic, or asthenic shape. And what I have called synochus presented a first stage of reaction, so feebly phlogistic, that the disease was very generally, and perhaps correctly regarded as typhus; and it was only those acquainted with the high phlogistic introductory stage of that form of fever in the earlier epidemics, who might at this time suspect the co-existence of a third form of fever, intermediate between synocha and typhus.

This nineteenth century description of disease could well have been written one hundred years earlier—theories on the nature and causation of fever had altered little since Hippocrates. Physicians continued to use the term "fever", sometimes adding a descriptive adjective indicating place of origin or a sign associated with the disease; in sixteenth century England it was called "spotted fever", a term still in use for tick-spread infections similar to typhus as in Rocky Mountain spotted fever; "gaol fever" was used in the eighteenth century because of frequent fever epidemics in overcrowded, insanitary prisons. In war it became known as "camp fever", at sea "ship fever", its appearance among vagrants and beggars gave it the name of "road fever", and in time of food scarcity it became known as "famine fever". The frequent outbreaks in Ireland and the appearance of the disease in Irish immigrants during the potato famine of the 1840s led to the name "Irish ague" or "Irish fever" becoming common. The term fever was in common use throughout the period under review; St Cuthbert's church burial

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9 W.Cullen, Synopsis nosologiae methodiciae, (Edinburgh, 1772), pp.20 & 21. Footnote 19 has information as to the provenance of the volumes studied.
records generally had fever as the cause of death and the small run of records in Greyfriars’ burial register has fever with no mention of typhus. By 1846, however, some doctors were beginning to employ modern terminology, Dr James Stark using the term typhus fever rather than fever in his report on the mortality of Edinburgh and Leith.

The classification of fevers and of disease in general, that had a lasting influence in the English-speaking world from its publication in 1772 until the middle of the nineteenth century, was that of William Cullen (1710–1790). Cullen became professor of the Institutes of Medicine at Edinburgh in 1766 and on the death of John Gregory in 1773 succeeded to the chair of the Practice of Medicine, a position he held until 1789, a year before his death. Cullen was close to several of the Edinburgh figures of the enlightenment including Joseph Black, Adam Smith and David Hume, caring professionally for both Hume and Smith, the latter dying in the same year as Cullen.

The work that brought Cullen recognition was his nosology (1769) in which he grouped diseases much as Karl von Linné (Linnaeus) (1707–78) had classified plants. In Cullen’s nosology diseases were classified by symptoms and arranged in four classes: pyrexiae, neuroses, cachexias and local diseases. Classes were divided into orders, genera and species, a system that seems complex but in fact was extremely simple when compared to that of Boissier de Sauvages (1706–1767) of Montpellier who classified disease in ten classes, 295 genera and, as Porter writes, ‘a daunting 2,400 species.’ The sixth edition of Cullen’s nosology, published in 1795, remained in print well into the nineteenth century and, as was shown earlier, his diagnostic categories and terminology

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14 Edinburgh City Archive, St Cuthbert’s Burial Records 1840–1849; Edinburgh City Archive, Greyfriars’ Burial Register 1835–1842.
17 Cullen, Synopsis nosologiae methodicae, (Edinburgh, 1772).
were still used by Edinburgh physicians in the second half of the century.\textsuperscript{19} His nosology was not his only contribution; the publication of the four volumes of \textit{First Lines of the Practice of Physic} between 1776 and 1784 confirmed his stature and influenced medical teaching in many countries being reprinted in several English, German and French editions.\textsuperscript{20} His reputation and influence did not survive into the twentieth century, at least not outside the English speaking world; Cullen does not rate a mention in Castiglioni’s wide ranging history of medicine published in Italy in 1936.\textsuperscript{21} Perhaps this was because there was little that was original in Cullen’s work, his importance to eighteenth and nineteenth century medicine being in his novel approach to the systematising of disease. His nosology was simply a new way of looking at disease that helped doctors to attach labels to illnesses of which often they had little understanding. Bynum has pointed out that ‘despite important differences of culture, knowledge, and intent, Hippocrates and William Cullen were part of a common medical tradition’, separated only by time during which theories of disease causation had hardly changed.\textsuperscript{22} Bynum’s opinion was echoed by Temkin in an historical review of the changing concepts of infection when he wrote that by the middle of the nineteenth century ideas about infection and its origin had not progressed much beyond the theories of Fracastoro in the sixteenth and Sydenham in the seventeenth century. Temkin quotes Charles Singer, described by Ackerknecht as ‘the sympathetic historian of animate contagionism’, who believed that no real progress had been achieved between Fracastoro and Pasteur.\textsuperscript{23}

\textsuperscript{19} The copy of Cullen’s \textit{Synopsis nosologiae methodicae} consulted in the National Library of Scotland was an 1823 London reprint of the first edition and was signed “A. Anderson, surgeon, 1835”. One of the copies in the Royal College of Physicians of Edinburgh is a 1795 sixth edition inscribed D.Craigie, 1817. Craigie was an Edinburgh physician and for a time editor of the \textit{Edinburgh Medical and Surgical Journal}.


The appearance of the new disease, syphilis, in the late fifteenth century forced many physicians to abandon traditional Galenic explanations for disease since it was plain that theories of humoral imbalance and miasmas could not explain the connection of syphilis with sexual activity and person to person transmission. Girolamo Fracastoro (Hieronymus Fracastorus: 1478–1553) in his *De Contagione et contagiosis morbis curatione* (1546) explained contagious diseases by postulating that not only was there a transfer of small particles or seeds, *seminaria contagiosa*, from person to person as occurred in syphilis, but this could also take place at a distance or through intermediate objects, *fomites*. Fracastoro described three forms of contagion: that which is transmitted by simple contact, as in leprosy or syphilis; by indirect contact through vehicles of infection, *fomites*, clothing or bedclothes; lastly, transmission from a distance, without contact, either direct or indirect, as in plague and smallpox.

For three hundred and fifty years the concept of contagion went unquestioned until the anticontagionists began to gain the ascendancy in the first half of the nineteenth century. Prominent among this group were Arnott, Southwood Smith and Kay-Shuttleworth, three physicians whose investigation into insanitary living conditions and disease in London was a significant factor in the increasing belief in miasmatic theory. They concluded that contagious fever was caused by a malaria from decayed organic matter, becoming epidemic when people congregated in crowded conditions. Their observations were published in the annual reports of the Poor Law Commission (1838, 1839) and were the prototype for the commission’s sanitary enquiry of 1842. As a result of their work and the advocacy of Edwin Chadwick in a political climate antagonistic to the trade restrictions associated with quarantine regulations, contagionism began to be viewed as an old fashioned, primitive, medieval belief, suspect and without scientific foundation. Morris describes the contagion versus miasma controversy which developed

during the cholera outbreak of 1832 and continued through the 1830s thus:

There was no progressive and orderly accumulation of knowledge, but a heated debate between competing systems of explanation, which filled journals and pamphlets with claim and counter claim, often talking at cross purposes because they were observing different rules of scientific argument.27

Dr John Roberton, an Edinburgh doctor, wrote in 1809: 'the various and vague application of the term contagion has been the source of much confusion. It has sometimes been used for the plague itself; sometimes as synonymous with infection; sometimes for the virulent effluvia issuing from the sick, or from substances infected; and sometimes merely as a property common to various diseases.' Roberton went on to say that he understood contagion as defined in the last of the meanings put forward.28 He grouped contagious diseases into the specific contagions such as measles or small pox and the general contagions, such as 'the fevers variously termed low, nervous, putrid, malignant, jail, hospital, etc, which, in various degrees of malignity, prevail chiefly in the habitations of the poor.' He believed that the infection was generated when people were crowded together in damp ill-ventilated surroundings.29 Roberton's appraisal reveals why the division of theorists into contagionists and miasmatists is altogether too simple, as Porter says 'much theoretical finessing went on.'30 An article in the *Westminster Review* in 1825 on 'Contagion and the Sanitary Laws', by Dr Southwood Smith included the following explanation:

A contagious disease is a disease which is capable of being communicated from person to person. An epidemic disease is a disease which at certain periods prevails generally over the whole, or over a large portion of a community....The cause of a contagious disease is a specific animal poison. The cause of an epidemic is, or rather supposed to be, a certain condition of the air. A contagious disease prevails by the communication from person to person of that specific

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28 Edinburgh University Special Collections, J.Roberton, *A Treatise on Medical Police and on Diet, Regimen etc.*, (Edinburgh, 1809), pp.204 & 205.
animal poison from which the malady derives its existence. An epidemic disease prevails through the influence of the atmosphere.  

The condition of the air was important to the miasmatists and particularly to the London based Scot, Dr Neil Arnott (1788–1874), a physician who had firm ideas about what was called vitiated air. Yet in his submission to the Scottish sanitary inquiry Arnott described two of the five causes of disease as ‘contagions and malaria’ but at the same time criticised ‘the opinion very general among professional men, that diseases might proceed from contagion alone.’ He asserted ‘that no truth in medicine is now better ascertained than that diseases proceeding from the influence of an accidental combination of ordinary circumstances do become contagious, that is, do spread from one person labouring under the disease to another person at the time in health.’

It is not easy to place Edinburgh physicians into the categories of contagionists or miasmatists. The Cullenian belief in specific contagia as the cause of smallpox and measles was accepted generally but his theory that a specific contagion caused typhus was less widely acknowledged. It must be remembered that his nosology was based on signs and symptoms rather than on aetiology, leaving room for doubt as to whether this aspect of Cullen’s teaching had any great effect on his successors in Edinburgh. William Henderson, professor of pathology in Edinburgh, was not a miasmatist, believing that continued fevers were produced by a poison, ‘although it is granted that nothing is known of the actual nature of the poison or poisons...’. He criticised the views of Dr Marsh of Dublin who ‘averts that he has known typhus produced by the contagion of small-pox, and intermittent fever by the contagion of typhus’ and those of

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Dr Southwood Smith who 'goes quite as far, virtually affirming that intermittent, remittent, typhus fever, and plague, all originate from different intensities of the same poison.'\textsuperscript{34} Robert Deuchar, secretary to the Edinburgh Fever Board, although not medically qualified, must have been influenced by the views of the physicians on the board when he wrote in 1844 of stables, byres, tanpits and receptacles for manure, as nuisances, 'which are exceedingly offensive and injurious, but experience has shown that malaria arising from these sources, does not appear to be the true cause of fever, or the leading cause of its extension among the poor.'\textsuperscript{35} Robert Christison in 1835–36 stated that 'continued fever occurs only when work is scarce, wages low, provisions dear and the labouring classes consequently in unusual distress.'\textsuperscript{36} His views are also those of his colleague, William Alison, who, criticising the opinions of Arnott, Kay, and Southwood Smith, declared that the recommendations of these men were erroneous, being 'founded on the supposition, that by removing all such causes of vitiation of the atmosphere, contagious fever may be arrested at its source, and thus all the evils resulting from it be prevented.' Alison regarded their conclusions as merely 'speculative' and declared that 'there was no reason for believing that the contagious fever which has prevailed more or less extensively in Edinburgh for the last 25 years has any such origin, or can be suppressed by any such measures.'\textsuperscript{37}

The debate about fever and its causation had an added dimension in Scotland. This was the controversy that arose over the workings of the Scottish Poor Law and was in part due to the conviction of William Alison that destitution was largely responsible for the fever epidemics in Scottish cities. William Alison (1790–1859), professor of medicine in the university, graduated at Edinburgh in 1811 and was appointed physician to the New Town Dispensary in 1815. The epidemic of 1817–1819 in which 3,119 fever patients

\textsuperscript{34} W.Henderson, 'On some of the Characters which distinguish the Fever at present Epidemic from Typhus Fever', \textit{Edinburgh Medical and Surgical Journal}, 61, (Edinburgh, 1844), pp.202 & 203.
\textsuperscript{36} Edinburgh University Special Collections, Christison Papers, Lectures 1835–36, p.14.
were admitted to hospital not only gave him first hand knowledge of the disease but his work as a dispensary physician also made him aware of the appalling conditions in which many of his patients lived.\textsuperscript{38} He made a special study of fevers, publishing his observations quarterly in the \textit{Edinburgh Medical and Surgical Journal} between 1817 and 1819 and in the succeeding years became convinced that destitution and fever were inextricably related.\textsuperscript{39} His pamphlet, \textit{Observations on the Management of the Poor in Scotland, and its Effects on the Health of the Great Towns}, (1840), was an attack on the provision for the poor in Scotland, pointing out the link between poverty and disease and making a plea for an investigation into poor law provision.\textsuperscript{40} The faction opposed to any change in the poor law was led by the Scottish divine, Rev Dr Thomas Chalmers (1780–1847), whose vision of ‘a godly commonwealth’ in which ‘religious and moral instruction alone would break the cycle of despair’ was based on the supposed success of the St John’s experiment in Glasgow.\textsuperscript{41} The celebrated debate between Alison and Chalmers at the Glasgow meeting of the British Association may have ended in a victory for the oratorical skills of Chalmers but in the end Alison and his supporters were successful in obtaining a commission to investigate poor law provision for Scotland.

The inquiry into the workings of the poor law was preceded by the inquiry into the sanitary condition of the labouring population where Dr Neil Arnott presented his conclusions, \textit{On the Fevers which have prevailed in Edinburgh and Glasgow}, summarising the four views presented at the Glasgow meeting of the British Association in 1840, ‘as to the cause and chief remedy of the misery and diseases prevailing among the poor of Scotland.’ He wrote that Dr Chalmers believed ‘that the want of good religious training was the cause and church extension was the remedy.’ Dr Alison held that destitution was the cause and that a good poor law for Scotland the remedy.’ A third

\textsuperscript{38} Christison, ‘On the Changes in the Constitution of Fevers’, p.17. The figure quoted by Christison is of patients admitted to the Infirmary and takes no account of those treated at home. Christison makes the point also that the hospital statistics of the time did not distinguish the three main types of fever.

\textsuperscript{39} Comrie, \textit{History of Scottish Medicine}, pp.610 & 611.


had stated that ‘the abuse of intoxicating drinks was the cause’ and the last believed that ‘want of national education was the cause.’ The men giving the two final opinions were not named. Arnott examined each opinion and although he concurred with each he concluded:

It follows therefore that the four apparently different proposals have so nearly the same objects in view...And although no one of the proposals furnished a precise answer to the question given to the London reporters, “What is the immediate or proximate cause of spreading fever, and can that cause be removed?” they do in no sense contradict the answer given by the London reporters,—that impurity affecting the air is the cause, and the prevention, diminution, or copious dilution of that impurity the remedy for evil;...  

The opinions of the three London reporters, Arnott, Kay-Shuttleworth and Southwood Smith, were accepted and with the enthusiastic support of Edwin Chadwick sanitary improvement became the recommended solution to the urban squalor that was assumed to be the trigger for epidemic disease.

Typhus, Relapsing Fever and Typhoid Fever

It was not until the 1840s in Edinburgh that physicians began to look at the signs, symptoms and post mortem appearances of fever and to identify the three distinct diseases. This section will examine the history of their recognition, their salient features and will summarise current knowledge of their nature. Typhus has a long history dating back to the eleventh century when spotted fever is said to have broken out in the monastery of La Cava near Salerno in Italy, but it was not until the sixteenth century that accurate descriptions of a disease very much like typhus are found. It is a disease associated with dirt and overcrowding, with wars and military campaigns, and with periods of economic depression and dearth: ‘now it was the constant accompaniment to life in the courts, closes, and wynds of the industrial towns. It was the poor man’s disease ... the product of squalor, insanitation, overcrowding and verminous conditions,

a concomitant of working-class housing." The discovery of the causes of the three infections would not be completed for several decades but to establish the extent of medical knowledge of these diseases in Edinburgh during this period the history of the three fevers will be reviewed, following which their clinical features and modes of transmission will be compared and contrasted.

There are two main varieties of typhus, the epidemic and the murine, and it is the former that appeared at frequent intervals in Edinburgh. The epidemic or classical typhus, the synochus of Cullen, is spread to man by the body louse, *Pediculus humanus corporis* or *Pediculus humanus capitis*, the head louse, the louse becoming infected by ingesting the blood of a typhus victim. The organism responsible is *Rickettsia prowazekii*, one of a group of microorganisms smaller than bacteria but larger than viruses and the sequence of spread is from man to louse to man with transmission usually occurring through the skin. Once rickettsiae have multiplied in the gut of the louse and have been excreted on to the skin, irritation and scratching cause abrasions and it is the contamination of these by rickettsiae that permits infection to enter the blood. Less common portals of entry include the lungs or the conjunctiva. These less important routes are dependent on the ability of rickettsiae to live for prolonged periods in dust, a potential that was first demonstrated by Rocha Lima in 1920 when he showed that rickettsiae could remain infective for as long as twenty four days and this capacity for survival may account for the occasional appearance of seemingly inexplicable cases.

The mode of transmission was discovered in 1911 by the French physician and bacteriologist, Charles Nicolle (1866–1936), and his colleagues working in the Pasteur Institute in Tunis. Nicolle noticed that typhus patients once in hospital no longer spread

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45 Hardy, ‘Urban famine or Urban Crisis?’ p. 406 makes this point about the longevity of rickettsiae, a fact that is not generally known but which is confirmed by Marmion, ‘Rickettsial diseases of man and animals’, p.576.
the disease whereas before admission they infected their families and their doctors and, during the process of admission, often infected the admitting staff. Nicolle saw that the answer must lie in events between admission and arrival in the ward and realised that the part played by simple hygiene was critical. It was routine for patients to be shaved, bathed and changed into fresh clothes, measures that led Nicolle to suspect the common louse, a suspicion that he was able to confirm in the laboratory. Within three years of his discovery measures were adopted which more or less eradicated typhus from Tunis; in 1928 Nicolle was awarded the Nobel Prize for Physiology and Medicine.46 There were earlier physicians who had guessed that the louse might be involved, including James Lind in the eighteenth century and Charles Murchison in the nineteenth but in the era before germ theory their theories could not be substantiated.47 The organism was isolated independently in 1910 by the American, H.T.Ricketts (1871–1910), and in 1913 by the Czech bacteriologist, S.L.von Prowazek (1875–1915) who died two years later when he and Rocha Lima were investigating an epidemic of typhus among Russian prisoners; both men contracted typhus fever, only Rocha Lima survived and named the organism *Rickettsia prowazeki* in recognition of von Prowazek and Ricketts who also died of typhus contracted during research.48

The clinical course consists of an incubation period of between eight to fourteen days, occasionally as long as twenty one days, then an abrupt onset of a high fever with severe headache, chills, muscle pains and vomiting. On about the fifth day a rash spreads from the trunk to cover the body, changing from a rose colour to become dark and purpuric by the tenth day. These red spots, similar to flea bites, are called petechiae, (small red or purple spots as a result of bleeding into the skin), and are the reason for the names spotted fever, petechial fever and exanthematic typhus. During the second and third weeks the victim may become comatose or delirious, the extent of cerebral involvement

47 Ackerknecht, *History and Geography of the Most Important Diseases*, p.42.
being more marked in typhus than in any comparable febrile disease; it is this degree of cerebral clouding and stupor that has given typhus its name, derived from the Greek for smoke, *typhos*. In favourable cases the temperature falls on about the twelfth day and recovery is then rapid, otherwise death occurs during the second week. Immunity in survivors can last for several years but after ten to twenty years this acquired resistance has largely disappeared and further mild infection can result. Epidemic typhus is often a relatively mild illness in children but severity increases with age: there is a 5 per cent mortality rate below the age of twenty, 10 to 15 per cent below forty years of age, 50 per cent below age fifty, and the disease is generally fatal over sixty years of age.

The second of the continued fevers is relapsing fever, like typhus a louse-borne infection but characterised by 'the occurrence of one or more relapses after the subsidence of the primary febrile paroxysm.' The pattern of improvement and relapse might suggest that the infection should properly belong to the intermittent fevers but in these the febrile episodes are widely separated by weeks or months rather than days, as in relapsing fever. Like typhus, relapsing fever is a disease associated with filth, poverty and famine and has been called the most epidemic of all epidemic diseases, frequently occurring at the same time as epidemics of typhus. The causative organism is *Borrelia recurrentis*, a spirochaete, transmitted to man through contamination of skin abrasions by the body fluids of the louse. As with typhus, transmission of relapsing fever can take place through the conjunctiva or the respiratory tract.

After an incubation period of between two to twelve days there is the sudden onset of headache, shivering, body pains and a high temperature. Jaundice appears in 20 to 60 per cent of cases and half of those attacked develop respiratory symptoms. In favourable

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51 Marmion, 'Rickettsial diseases of man and animals', p.575.
52 Wilson and Coghlan, 'Spirochaetal and leptospiral diseases', p.523.
53 Ackerknecht, *History and Geography of the Most Important Diseases*, p.44; Wilson and Coghlan, 'Spirochaetal and leptospiral diseases', p.524.
54 Wilson and Coghlan, 'Spirochaetal and leptospiral diseases', p.525.
cases, the temperature falls between the third and the ninth day, and the patient once again feels well, only to suffer a relapse between the tenth and fifteenth day. Second, third and fourth relapses can occur but are less common, less severe and of shorter duration. Christison described the illness thus:

The onset is abrupt rendering the patient prostrate perhaps within one hour. There is fever, anxiety, restlessness, headache, and irritability of sense. The temperature may reach 107 degrees; there is excessive thirst and extreme suffering because of the acute senses. The cessation of symptoms at about day five is sudden and complete so that the patient may be up and about. There is an abrupt relapse on day fourteen with rigors and severe prostration. On day three of the relapse the crisis appears.55

Christison had not only treated patients with the disease in four epidemics but he had also survived relapsing fever during the epidemic of 1817–19. These epidemics he attributed in his lectures to ‘want of employment and wide–spread penury among the working classes ...so invariable is this connection, that we sometimes hear it called famine fever.’ (Original underlining).56

Modern authorities say that the case fatality in relapsing fever is as low as 5 per cent when conditions are favourable but if the epidemic occurs during war or famine the mortality rate can rise to 70 per cent.57 The variation appears to have been less marked in the nineteenth century; Christison in 1863 wrote of the disease as being ‘far from deadly’, Jenner from his experience of the disease in London considered that ‘death is a rare termination of relapsing fever’ and Jackson, a Leith Dispensary physician, in a careful statistical analysis of the epidemic of 1843, concluded that ‘the general prognosis of the simple epidemic fever is most favourable.’ Jackson found that out of 1,299 dispensary cases forty–five died, a mortality rate of 3.5 per cent and of 216 cases admitted to Leith Hospital ten deaths occurred, a mortality rate of 4.6 per cent, but he explained, ‘it must be remembered that in general the worst cases were sent to the

56 Edinburgh University Special Collections, Christison Papers, Lectures on Continued Fever, (no date but after 1847), pp.33 & 34.
hospital, and few of these ten died simply from fever, but some from some other disease occurring with it.\textsuperscript{58}

The organism responsible for relapsing fever was identified by Dr Otto Obermeier (1843–1873) during the Berlin epidemic of 1867–68 but he did not publish his findings until 1873. F.P. Mackie working in India proved in 1907 that the common louse was the vector and Nicolle and his associates clarified the epidemiology in 1912.\textsuperscript{59} Anne Hardy says that the term relapsing fever was first used in 1843 by David Craigie, physician to the Edinburgh infirmary and from 1832 to 1853 editor of the \textit{Edinburgh Medical and Surgical Journal}.\textsuperscript{60} The Edinburgh physician and pathologist, William Henderson (1810–1872), was the first to differentiate typhus and relapsing fever on clinical grounds. Henderson graduated at Edinburgh in 1831 and the following year was appointed physician to the fever wards and pathologist to the Royal Infirmary, becoming professor of pathology in 1842.\textsuperscript{61} In a paper published in the \textit{Edinburgh Medical and Surgical Journal}, (1844), Henderson demonstrated the quite different nature of the two diseases. He did not rely on post mortem findings or microscopy for proof, perhaps surprisingly in view of his reputation as a pioneer microscopist in the study of diseased organs, based his case on clinical and epidemiological observation using the example of Isabella McDonald, of 327 Canongate. She was admitted to hospital in April 1843 with typhus fever showing ‘the usual exanthematous eruption’ and discharged home, cured in May. Between May and July her mother and three of her sons developed typhus, one of the sons aged thirteen, dying in the fever ward. Henderson writes that ‘the epidemic fever did not commence in the stair till June,— and in the months of August, September, and November, the surviving five members of the family, who had been affected with


\textsuperscript{59} Wilson and Coghlan, ‘Spirochaetal and leptospiral diseases’, p.524.


\textsuperscript{61} Comrie, \textit{History of Scottish Medicine}, p.623.
typhus were now seized with the epidemic fever, which was then abundant among the other families in the stair. The significant factor in these case histories is acquired immunity; physicians knew that an attack of typhus bestowed on the survivor a prolonged period of immunity. Henderson therefore reasoned that the second attack of fever, which he called epidemic fever, must differ from the first and using this and other case studies concluded on clinical and epidemiological grounds that there were two diseases, typhus and epidemic fever. He made no reference to a third infection, typhoid fever which had been identified by a French physician some years earlier. The advances made by these two physicians in clarifying fever are in contrast to the failure of Edinburgh doctors to identify typhoid fever as a separate entity.

The study of morbid anatomy was a relatively new science in Edinburgh in the 1830s but in France and Germany during the early years of the nineteenth century pathology had progressed, with the microscopic examination of diseased tissue becoming increasingly common. Physicians were correlating post mortem appearances with clinical findings and the identification of typhoid fever as a disease distinct from typhus and relapsing fever owed much to these new methods. Porter maintains that William Jenner (1815–1898), professor of pathology in London, and William Gerhard (1809–1872) of Philadelphia were first to distinguish typhoid from typhus on the basis of pathological examination. Castiglioni says that the two were generally regarded as one until Gerhard clearly differentiated them during the Philadelphia epidemic of 1836. The credit, however, should go to French physicians who in the early nineteenth century recognised that typhus and typhoid were different diseases and described the post mortem intestinal changes characteristic of typhoid at least twenty years before Jenner and a decade before Gerhard.

62 Henderson, 'On some of the Characters which distinguish the Fever at present Epidemic from Typhus Fever', p.217.
63 Porter, The Greatest Benefit to Mankind, p.413.
64 Castiglioni, History of Medicine, p.709.
These new techniques enabled Prost in 1804 and Petit and Serres in 1813 to declare that entero–mesenteric or mucous fever and the slow, nervous fever first described in 1739 by the English physician John Huxham (1692–1768) were one and the same disorder.65 The Belgian anatomist Adriaan van den Spieghel described the characteristic lesions in the small intestine early in the seventeenth century but it was not until 1826 that Pierre Fidèle Bretonneau (1778–1862), a physician from Tours, demonstrated conclusively that typhus, the so-called putrid malignant fever, differed from typhoid, the slow nervous fever by showing that the lymphoid tissue in the small intestine in typhoid becomes ulcerated whereas in typhus this does not occur. Bretonneau called this fever of intestinal changes, ‘dothientérite’, (Greek, dothien, a boil and enteron, intestine).66 The French physician, Pierre Louis (1787–1872), was first to use the term typhoid when he described in 1829 the post mortem changes in the small intestine, confirming Bretonneau’s observations.67 One of the most striking contributions to the epidemiology of typhoid fever in the pre germ theory era was made in the 1840s by William Budd (1811–1880), a Bristol physician, who had studied in London, Edinburgh and Paris, graduating MD Edinburgh (1838) with a gold medal. Budd maintained that typhoid was a contagion transmitted by infected material in faeces and suspected that faulty hygiene was responsible through contamination of milk and water. To prevent spread in this way he recommended careful hand washing, the disinfection of utensils and the boiling of water but miasma theorists opposed him and little benefit accrued.68 A further thirty years passed before Georg Gaffky (1850–1918), a colleague of Robert Koch, isolated

the typhoid bacillus in 1884, following earlier work by K.J.Eberth (1835–1926).

The French work was confirmed by Robert Perry (1783–1848), a Glasgow Fever Hospital physician, in a paper (1836) based on his experience of typhoid fever in the city. Perry summarised the features of typhoid and typhus, showing how they differed, correlated these differences with the post mortem changes in the intestinal lymphoid glands and concluded ‘dothinenteritis or gastroenteritis have been too often confounded with typhous fever.’ It was at this time in the 1830s that interest in typhoid fever forced many physicians to consider the possibility that continued fever might not be a single disease. For example, the Edinburgh Medical and Surgical Journal in 1837 reviewed five German articles on abdominal typhus, described variously as inflammatory abdominal typhus, gastric nervous fever, abdominal typhus and enteric typhus, commenting:

the subject of Abdominal typhus or fever with abdominal disorder or affection of the intestinal mucous membrane and follicles, has so engrossed so much of the attention of foreign physicians, that it becomes requisite to bestow some separate consideration of the facts and opinions adduced during the last two or three years.

Study of this paper and that of Perry and remarks about the interest shown by foreign physicians in typhoid fever suggest two possibilities: either the disease was comparatively rare in Edinburgh or Edinburgh physicians had failed to recognise it because they held to old theories or they were simply not interested in possible differences. In 1840 a Glasgow colleague of Perry, Alexander Stewart (1813–1883), had also published a paper on typhus and typhoid based on his experience in the Glasgow Fever Hospital. Stewart was a fluent French speaker and read his paper first to the Medical Society of Paris in April 1840. According to Tweedie, writing twenty years

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69 R.Perry, 'Observations on Continued Fever as occurs in the City of Glasgow Hospitals', Edinburgh Medical and Surgical Journal, 45, (1836), pp.64–70.
70 Anon, 'Articles on Abdominal Typhus,' Edinburgh Medical and Surgical Journal, 48, (1837), p.146.
71 A.P.Stewart, 'Some Considerations on the Nature and Pathology of Typhus and Typhoid Fever, applied to the solution of the question of the identity or non-identity of the two diseases', Edinburgh Medical and Surgical Journal, 45, (1836), pp.289–339.
later, Stewart linked post mortem findings and clinical observations in support of his hypothesis:

It embodied results of investigations made in the Glasgow Fever Hospital, the object being to prove, by comparison of the symptoms during life with the lesions after death, that no two acute diseases can differ more widely from each other, the characteristics of each being so marked, if attentively observed, as to defy misconception.  

There was no doubt that typhus and typhoid were different diseases but Edinburgh doctors were not alone in their reluctance to accept this fact. Patrick, in a review of the enteric fevers, quoted William Jenner who wrote in 1849 that “with few exceptions British physicians had laboured to prove that typhoid and typhus were identical.”

Typhoid fever is one of the enteric fevers, acute intestinal infections caused by organisms of the salmonella group. The clinical picture varies from a relatively mild illness with no fever to a grave prostrating disorder with high fever, the severity depending on the virulence of the salmonella strain and individual resistance. The risk of developing typhoid is directly proportional to the dose but the clinical course is not affected by the number of organisms ingested. The bacilli enter the body orally and epidemiological research has shown that the only source of typhoid bacilli is an infected human. The most common vehicle is water contaminated by infected excreta, infection occurring directly by drinking such water or indirectly by consuming food contaminated either by polluted water or by a carrier. Water borne epidemics have a typically explosive onset with primary cases from direct infection followed by outbreaks of secondary infection spread by carriers infected during the first wave. Typhoid can also be spread by infected milk or dairy products where heat treatment of milk is not carried out; in such cases the origin of the infection is usually a human carrier who through hygiene failure transmits the bacteria to food products. It is estimated that about 5 per

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72 Tweedie, *Lectures on Continued Fevers*, p.31.
76 Parker, ‘Enteric Infections’, p.417.
cent of people who have had typhoid become temporary carriers of the infection and 2 per cent become chronic carriers, excreting the organisms intermittently and constituting a dangerous human reservoir.\textsuperscript{77} After an incubation period of eight to fifteen days, with limits of five and thirty days, there is a slow onset of general debility with headache, fever and gastrointestinal upset. Untreated typhoid lasts for about four weeks with the temperature rising during the first week, levelling out in the second week, falling in the third and becoming normal by the fourth week. On about the seventh day a sparse, rose-coloured rash appears which lasts for three to four days and it has been suggested that this sign was responsible for the disorder being confused for so long with typhus fever but, as Luckin points out, the diarrhoea that occurs in typhoid but not in typhus should have alerted physicians to the different nature of the two diseases.\textsuperscript{78} The fact that the diagnosis was uncommon in Edinburgh is difficult to explain but there are clues to the answer. In the twelve months from October 1841 the total number of post mortem examinations carried out in the infirmary was 219, including twenty eight performed on fever victims and one on a patient who died of dothinenteritis. No indication is given as to whether the diagnosis of dothinenteritis was made on clinical grounds or after post mortem examination but it does show that typhoid fever was a condition recognised by physicians in Edinburgh at this time and suggests that it was not a failure to recognise the condition but its rarity in the city that was responsible for the rarity of this diagnosis.

The Loss of Henderson, Reid and Cormack

It is understandable that Edinburgh's senior physicians at this time were preoccupied with other issues than the contagion versus miasma debate, for example, Alison with his efforts to reform the poor law provision, but there were younger men who were investigating fever, employing new methods and publishing their results. The paper by William Henderson already cited was not his first on fever; in collaboration with his

\textsuperscript{77} Parry, \textit{Communicable Diseases}, p.105.
colleague, John Reid (1809–1849), he had written in 1839 and 1840 on the epidemic fever of Edinburgh, based on clinical observations, treatment and findings at post mortem examination.79 The Infirmary minutes record a letter from Henderson and Reid ‘asking leave [from the hospital managers] to publish details of the symptoms and morbid anatomy of the fever lately so prevalent in the wards of the Infirmary.’80 Reid and Henderson were young, innovative physicians, exploring and developing new techniques and co-operating in pathological and statistical methods. Henderson was a pioneer in the use of the microscope in the examination of post mortem tissue and Reid saw that statistics might produce useful information on the causation of disease. In 1839 he criticised the existing method of collecting statistics in the Infirmary, suggesting to the managers the adoption of a new, improved system based on that of the Statistical Society of London, a recommendation that was accepted.81 In 1841 Reid resigned his post on being appointed to the chair of anatomy in St Andrews and Henderson was ostracised by the Edinburgh medical hierarchy who were outraged when in 1845 he took up homeopathy. The university medical professors attempted to have him ousted from the chair of pathology and although they were unsuccessful his career was effectively ended.82 He resigned his position as physician to the Infirmary but not the chair of pathology, claiming that his views on therapeutics were unlikely to affect his teaching of pathology, an argument that had some merit. His opponents were not appeased, however, and the anti–Henderson faction led by the surgeon, James Syme, having failed to have him removed from his university chair, tried to have pathology declared a non compulsory subject in the undergraduate curriculum.83 The loss of Reid and Henderson undoubtedly held back the study of pathology and statistics in Edinburgh and also reveals the lack of interest in these disciplines shown by senior Edinburgh medical men.

80 Minutes of the Royal Infirmary of Edinburgh, 12, p.221.
81 Edinburgh University Special Collections, Lothian Health Services Archive, Minutes of the Royal Infirmary of Edinburgh, April 1839, 12, pp.202–208.
82 Comrie, History of Scottish Medicine, p.577.
83 Comrie, History of Scottish Medicine, p.623 refers to the paper written by Henderson in 1845 entitled “An Enquiry into the Homeopathic Practice of Medicine.”
A third young Edinburgh man developing a reputation as a fine physician and writer resigned his position at the Royal Infirmary. John Rose Cormack (1815–1882), later Sir John, the author of an important publication on the fever epidemic of 1843–44, graduated at Edinburgh in 1837, having been awarded a Gold Medal for his thesis. He was appointed physician to the Infirmary and the Fever Hospital soon thereafter but on being refused permission to give clinical lectures, mainly bedside teaching, he resigned and moved to London. The reason for the refusal is not clear but it is probable that loss of fee income persuaded more senior men against him. He began the *Monthly Journal of Medical Science* in 1841, continuing as editor until he left Edinburgh, and soon after his arrival in London set up the *London Journal of Medicine*, later becoming editor of the *Association Medical Journal*, the forerunner of the *British Medical Journal*. Cormack described the 1843–44 outbreak, reviewing in detail the case histories of fever patients under his care, including the particulars of nineteen patients, all of whom developed jaundice, with eight deaths. Two of these patients worked in the fever hospital, one a doctor and the other the nurse who cared for him during his illness, fortunately both survived. Cormack was sure that fever was contagious, explaining that almost all the clinical clerks and other workers exposed to the contagion in the fever wards contracted the disease; many nurses, laundry women and others coming into contact either with the patients or their clothes were infected and at one time there were eighteen nurses off duty with fever. He wrote 'from the number of laundry women that have been attacked, it appears, that the clothes of our fever patients are especial repositories and communicators of the morbid poison.' The reason that Dispensary doctors did not succumb to fever, he suggested, was because their period of exposure was too short and although 'there were isolated cases in the best districts of the New Town there was never any instance of the disease propagating itself in these localities.'

The belief that the poison did not extend for more than three or four feet from the patient

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was behind the London method of distributing fever patients throughout general wards in beds placed at what was considered to be a safe distance one from the other. If there were too many fever cases in a ward it was thought that the atmosphere became tainted, allowing the disease to take hold and spread. Cormack explained that similar results had been obtained in the Edinburgh Infirmary and he was certain that 'were the fever patients cautiously diffused through the general wards the lives of many physicians, clerks and nurses might be spared.'[Original Italics].

It is interesting that Cormack described 'a peculiar form of ophthalmitis, usually preceded by amaurotic symptoms' in patients recovering from fever and wrote that identical cases of inflammation and amaurosis (blindness) had followed fever in Glasgow and had also been reported in fever patients during the Dublin epidemic of 1826. Cormack quoted Dr MacKenzie, a Glasgow ophthalmologist, in whose experience no other febrile illness was followed by this type of inflammatory condition of the eye. This condition was also mentioned by Murchison who claimed that it was commented on by almost all writers on the epidemic of relapsing fever of 1843 and was never met with after typhus or enteric fever. Charles Murchison (1830–1879) graduated at Edinburgh in 1850, serving in the Bengal Medical Service, after which he lectured at the Middlesex Hospital and St Thomas's Hospital. He had worked as a clinical clerk in the Royal Infirmary of Edinburgh during the great fever epidemics of 1847–48 and accordingly had experience and knowledge. His Treatise on Continued Fevers of Great Britain (1862) reviewed epidemics in Britain and with its comprehensive bibliography is a useful source. Murchison wrote that the outbreak of enteric fever at the end of 1846 'not only occurred before the Irish fever but came from the country and from the best houses of the New Town— not from the crowded courts of the Old Town, to which the later epidemic of typhus and relapsing fever were restricted', but he did not advance any evidence in support of these contentions. He noted that Dr MacKenzie believed

ophthalmia was most common in the very poor who had insufficient nourishment during convalescence, that poor diet was one of the main causes of the disorder and was the reason 'why the affection in question succeeds no other fever than relapsing.' The condition, xerophthalmia, (dryness of the eye) is caused by vitamin A deficiency and it is now known that increased utilisation of the vitamin by the immune system occurs during acute, severe infections. Cormack's description of ophthalmic signs and symptoms in the poorly nourished after fever suggested that the cause might be vitamin A deficiency, but assessment by an ophthalmic specialist concludes that the appearances described are not those of xerophthalmia.

The Fever Epidemics

In the early nineteenth century the most pressing need amongst physicians was to explain the appearance and disappearance of fever, rather than to analyse differences between diseases. There were numerous theories of causation; destitution, dissipation, prisoners, sailors and the Irish were all blamed at one time or another for epidemic fever. Dr Paterson, physician to the Royal Infirmary and to the Leith Dispensary in 1848 wrote 'a typhous fever of more malignity than usual appeared in Edinburgh in 1779' stating that it originated in the hospital for sick prisoners of war in the castle. He recalls a similar fever brought to Leith by 'a fleet of merchantmen arrived in Leith Roads from Jamaica.' He commented that

It was customary for a time to attribute the increase in fever, which usually occurred during the autumn and winter months, to importation with the Irish reapers; but, in truth, the same disease was in existence before the Irish came over, and was probably at the time increased by having individuals exposed to its contagious influence, with their constitutions in a state which predisposed them to take it, or that the particular season of the year tended to its increase.

Dr Jackson described the epidemic of 1843, emphasising ‘the great misery and destitution [that] prevailed at the time amongst the poor’, similar to Christison’s analysis of the epidemic of 1835–36 writing that the disease ‘occurs only at periods when work is scarce, wages low, provisions dear and the labouring classes consequently in unusual distress.’

Creighton wrote that the typhus epidemic of 1842–44 was purely a Scots affair, quoting Murchison’s view that this epidemic was different from those that preceded it, inasmuch as it did not originate in Ireland. Creighton concluded that this ‘proved once for all that one had not to go to Ireland for the engendering or making of famine–fever.’

The alacrity with which a scapegoat was sought and found is seen in the report by Dr Betty of Fermanagh in the *Quarterly Journal of Medicine* in which he claimed that in the north of Ireland the relapsing fever of 1846 was called “the Scotch fever” on the supposition that it came to Ireland from its focus in the west of Scotland.

There are several detailed descriptions of the first fever epidemic of the 1840s which is thought to have broken out on the coast of Fife in 1841–42 reaching Edinburgh in February 1843. Murchison says that this was mainly relapsing fever, confined to Scotland, with typhus comparatively rare, and he believed that the epidemic broke out in 1841 continuing until 1844. Alison in a paper read to the Statistical Section of the British Association at York in 1844 described ‘the diffusion of fever, particularly of that new form of fever which has sprung up since 1842, almost exclusively in Scotland…’.

Christison talked of two separate epidemics, one in 1841 and a second in 1843–44, the first he attributed to typhus alone but the second he thought was largely relapsing fever. The evidence supports Christison’s view that there were two epidemics, the first in 1841 and the second beginning in late 1842 lasting until 1844, both outbreaks involving typhus and relapsing fever with the latter predominating. The managers of the infirmary believed that the epidemic of 1837–40 returned in the spring of 1841 and recorded 1372

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95 Jackson, 'An Account of the Epidemic Fever as observed in Leith, with the Statistical Details of Three Hundred Cases', p.418; Christison Papers, Clinical Lectures 1835–36, p.14.
patients treated for fever in the twelve months up to October 1841, 590 more than in the previous twelve months.99

The infirmary minutes record that there were 469 fever patients in the hospital on 26 September 1843 and many could not be admitted because no beds were available so that patients were placed in garrets, on floors and on occasion two to a bed. The situation had not improved by October, when, at a meeting with the governors of the Royal Lunatic Asylum to discuss the conversion of the Bedlam into a temporary fever hospital, the chairman explained that 443 fever cases were being treated and that for the previous ten days an average of eighty fever patients daily had been turned away. All available beds were occupied, with the chapel and two temporary wooden buildings holding fifty five patients, being used as wards. Three other emergency fever hospitals were also full, necessitating the temporary use of the City Bedlam to house fever patients.100 The developing crisis is revealed in table 8.2 which shows that fever admissions increased from 842 in 1842 to 3339 two years later.

<table>
<thead>
<tr>
<th>Year</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1841</td>
<td>1372</td>
</tr>
<tr>
<td>1842</td>
<td>842</td>
</tr>
<tr>
<td>1843</td>
<td>2080</td>
</tr>
<tr>
<td>1844</td>
<td>3339</td>
</tr>
<tr>
<td>Total</td>
<td>5833</td>
</tr>
</tbody>
</table>

Table 8.2. Fever Admissions to the Infirmary, 1841–44.101

Whether there were two epidemics in the period 1840–44 is uncertain but the increase in the number of cases in 1843 and 1844 and the contention of Christison and others that there was a second epidemic, on this occasion of relapsing fever in 1843, is strong evidence in support of two outbreaks. Dr Jackson thought that fever had been in Edinburgh for some time before making its appearance in Leith in September 1843;

99 Edinburgh University Special Collections, Pamphlet 107/1, Report by the Managers of the Royal Infirmary for the year from 1 October 1840 to 1 October 1841.
100 Minutes of the Royal Infirmary, 13, 1842–44, 26 September 1843, 16 October 1843.
figures from the Leith Dispensary showed that in the six months from September 1843 1,299 patients were treated for fever, with the greatest number, 417, in October.

St Cuthbert’s churchyard cash book is the only surviving cemetery record for the period with cause of death included but it has to be emphasised that the numbers of burials are a small and unknown percentage of the total mortality for the city. The data from this source are unlikely to be representative because most pauper burials were carried out in the city burying ground, Greyfriars. The percentage of pauper interments at public expense in 1837–39 was approximately 14 per cent of all interments.102 The admittedly limited data from St Cuthbert’s do not support the theory that there were two separate epidemics in 1841 and 1844 but it is possible that most fever victims were buried in other graveyards, There is evidence that the council and the public were becoming increasingly concerned about the state of Greyfriars cemetery — by January 1845 the council was actively seeking land for a new public cemetery and about the same time the Kirk Session of St Cuthbert’s and the managers of Calton and Warriston cemeteries informed the council that they were no longer prepared to bury the poor.103 Unfortunately there are no extant records for pauper burials in these cemeteries but earlier figures show that the number of paupers buried free, after anatomical dissection in the medical school, totalled 638 in one twelve month period in 1837–38.104 Of these, 377 were buried in Greyfriars’ churchyard, the remaining 261 being buried in Canongate and St Cuthbert’s graveyards. The deaths from typhus and fever recorded in St Cuthbert’s between 1841 and 1843 are shown in table 8.3.

103 Edinburgh City Archive, MacLeod Bay D, Bundle 168, Shelf 19, Correspondence relating to cemeteries and burying the poor, 1844–45.
104 Royal College of Surgeons of Edinburgh Records, 4 February 1839.
Table 8.3. St Cuthbert’s churchyard 1841–1844: deaths from typhus and fever.\textsuperscript{105}

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1841</td>
<td>51</td>
</tr>
<tr>
<td>1842</td>
<td>42</td>
</tr>
<tr>
<td>1843</td>
<td>41</td>
</tr>
<tr>
<td>1844</td>
<td>43</td>
</tr>
</tbody>
</table>

Table 8.3. St Cuthbert’s churchyard 1841–1844: deaths from typhus and fever.\textsuperscript{105}

The Statistical Tables of the Royal Infirmary give the number of fever cases and the mortality rate for the twelve month periods ending 30 September 1840, 1841 and 1842, (table 8.4).

<table>
<thead>
<tr>
<th>Period</th>
<th>Cases</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 September 1839 to 30 September 1840</td>
<td>833</td>
<td>12.7 per cent</td>
</tr>
<tr>
<td>30 September 1840 to 30 September 1841</td>
<td>1273</td>
<td>14.6 per cent</td>
</tr>
<tr>
<td>30 September 1841 to 30 September 1842</td>
<td>820</td>
<td>13.9 per cent</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2926</strong></td>
<td><strong>Average mortality 13.7 per cent</strong></td>
</tr>
</tbody>
</table>

Table 8.4. Statistical Tables of the Royal Infirmary: fever cases.\textsuperscript{106}

Of admissions to the infirmary in the year to 30 September 1842, 582 were from Edinburgh, 114 from Leith, forty seven from adjacent districts, seventy one had no fixed residence and six nurses or other attendants contracted fever in the wards. A footnote recorded that an unspecified number of patients caught fever while convalescent from the disease for which they had been admitted.\textsuperscript{107}

It was only with the appointment of Dr James Stark that there became available for a three year period an official record of the city’s mortality, shown in table 8.5. The data for 1845 are taken from Stark’s report for 1847 but he does not cite a source for these and the statistics for the 1846–48 are taken from his three annual reports.

\textsuperscript{105} Edinburgh City Archive, St Cuthbert’s Church Burial Records, 1841–49.

\textsuperscript{106} Edinburgh University Special Collections, Statistical Tables of the Royal Infirmary, 1841–42.

\textsuperscript{107} Statistical Tables of the Royal Infirmary.
<table>
<thead>
<tr>
<th>Year</th>
<th>1845</th>
<th>1846</th>
<th>1847</th>
<th>1848</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Mortality</td>
<td>3688</td>
<td>4887</td>
<td>7026</td>
<td>5754</td>
</tr>
</tbody>
</table>

Table 8.5. Mortality figures for Edinburgh 1845–1848.108

The mortality from ‘zymotic disease (epidemic, endemic, and contagious)’ and the number of typhus victims in that total is shown in table 8.6.

<table>
<thead>
<tr>
<th>Year</th>
<th>1846</th>
<th>1847</th>
<th>1848</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zymotic disease</td>
<td>1141</td>
<td>2679</td>
<td>2468</td>
</tr>
<tr>
<td>Percentage of total deaths</td>
<td>24</td>
<td>39</td>
<td>46.9</td>
</tr>
<tr>
<td>Typhus</td>
<td>212</td>
<td>1517</td>
<td>965</td>
</tr>
</tbody>
</table>

Table 8.6. Zymotic disease and typhus Edinburgh 1846–48.109

Stark states that the average mortality in the city between 1840 and 1846 was 3972 deaths annually, or one death out of every 35.3 inhabitants, according to the 1841 census population; by 1847 this had increased to one in 20.9 of the population and in 1848 it was one in 25.6. The figures for other cities for 1848 included Manchester, one in 26.5, Liverpool, one in 23.6, Glasgow, one in 21.9. Table 8.6 shows that from 1846 the percentage of deaths from infectious disease almost doubled and that typhus was largely responsible for this, even in 1848 when cholera deaths augmented the total. In the three years from 1846 deaths from typhus totalled 2,694 out of a total mortality of 17,667.110

It is not surprising that there is no category for typhoid fever in Stark’s mortality reports since only in 1869 was this included in the Registrar General’s list. Stark lists diarrhoea and dysentery under zymotic disease whereas enteritis and gastritis, which could be

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caused by infection, are listed under diseases of the stomach and other organs of digestion. The number in each category for the three years is shown in table 8.7 and shows that there was a steady increase in deaths from enteritis in 1847 and from enteritis and gastritis in 1848, a year in which the two diagnoses were not listed separately. Without more detail it is impossible to say whether some of these deaths were due to typhoid fever, although this suspicion remains.

<table>
<thead>
<tr>
<th></th>
<th>1846</th>
<th>1847</th>
<th>1848</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysentery</td>
<td>18</td>
<td>32</td>
<td>16</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>171</td>
<td>118</td>
<td>75</td>
</tr>
<tr>
<td>Enteritis &amp; Gastritis</td>
<td>95 (gastritis 4)</td>
<td>193 (gastritis 3)</td>
<td>246 (gastritis not listed separately)</td>
</tr>
</tbody>
</table>

Table 8.7. Mortality from gastrointestinal disease 1846–48.\(^{111}\)

Administrative Response: The Fever Board and the Police Commission

There is no evidence that the city’s three medical institutions, the two Royal Colleges and the Medical School, tried to reach a consensus on the contagion versus miasma controversy. Scrutiny of papers written by prominent physicians and the absence of any reference to a discussion of the issue in the minutes of the Royal Colleges suggests that this was seen as a matter for the individual practitioner.\(^{112}\) The Colleges did not enter the debate because, as their minutes reveal, they were more concerned with matters relating to medical education, qualifications to practise and the protection of the professional status of their fellows. It is perhaps surprising that they did not discuss measures to deal


\(^{112}\) The Edinburgh Medical and Surgical Journal between 1830 and 1850 has no papers dealing specifically with this debate, nor do the minutes of the Royal College of Physicians or the Royal College of Surgeons for this period refer to any discussion of the issue.
with the fever epidemics and it was only with the threat of cholera in 1848 that there is any reference to planned action, perhaps for the reason that the control of fever epidemics was largely in the hands of a voluntary organisation, the Fever Board, on which sat several of the city’s most prominent medical men.

The Fever Board took over their role from the Destitute Sick Society which, from the year of its foundation in 1817, had arranged with the Royal Infirmary for the transfer and admission of fever patients to hospital after medical certification. The Society had been responsible also for the cleaning and fumigation of houses in which fever had been present but decided in 1830 to restrict their activities to charitable work and their quasi public health role was taken over by the Fever Board. The directors of the society, however, did not entirely abandon their previous function for in August 1843 they wrote to the cleaning committee of the Police Commission stressing ‘the necessity of enforcing the police regulations as to cleaning common stairs’, in view of the prevalence of fever. The Commission far from ignoring this apparent interference instructed the Inspector of Cleaning and Lighting to employ extra men to implement their request.

The records of the Board have not survived but it is known that it comprised an ex officio president, the Lord Provost, and ten or twelve committee members; two from the town council, two physicians from the infirmary, two medical officers from the Royal Public Dispensary, two medical officers from the New Town Dispensary, two members from the Destitute Sick Society and three or four special members of the board. In 1836 the special members were Dr John Abercrombie who was the president of the Destitute Sick Society, Dr Alison, Dr Borthwick and Dr MacLagan; in 1845 there were only three special members, Alison, MacLagan and Dr W.Tullis. In 1837 the Town Council

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114 Edinburgh City Archive, Minute Book of the Cleaning Committee, July 1840–September 1844, 3 August 1843, p.152.

115 Edinburgh Almanac 1836, (Edinburgh, n.d.), p.430. This almanac was the forerunner of Oliver & Boyd’s New Edinburgh Almanac; Oliver & Boyd’s New Edinburgh Almanac 1845, (Edinburgh, 1845), p.516.
agreed to grant the Fever Board £100 per annum to enable it to carry out its duties, an action which to a degree formalised the Board's responsibilities. A pamphlet published in 1844 at the height of that year's epidemic, was written by the secretary to the Board, Robert Deuchar, in which he commented on the presence of Irish labourers in the city: 'it cannot be doubted that fever is often introduced to this city by these strangers', and observed that 'a considerable proportion of fever cases in the Hospital are sailors and others connected with shipping.' Quite how he resolved the two statements is not clear. He was surprised that the level of fever in Edinburgh was not greater in view of 'the extent of disease which has so recently prevailed in Glasgow,' and 'the increased facilities of intercourse betwixt the two cities.'

The Fever Board was responsible for 'the cleaning of the bedclothes and wearing apparel of those who had been attacked by fever, rather than in purification of the walls and atmosphere of dwelling houses where fever was likely to prevail.' Deuchar criticised the Glasgow method in which patients were treated at home as happened in 1843 when 12,397 out of 13,703 persons with fever were dealt with in this way, penny tickets for food being distributed to aid the destitute. Deuchar believed that the Edinburgh method of removing patients to hospital was preferable, commenting:

> Indeed it must be palpably evident that nothing less than the removal of the patients to an hospital, and a complete separation effected betwixt the sick and the healthy, can possibly prove a successful check to the increase of fever in Glasgow, or in any other large city. Similar scenes are occasionally seen in this town, but never when the combined exertions of the Fever Board and Royal Infirmary can prevent them.

Deuchar explained that the Edinburgh method had been recommended by the medical members of the Board who had seen not only the necessity of separating the sick and the healthy but also the benefits which would accrue from cleaning and fumigating the

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houses of fever patients and washing their bedclothes. The work of the Board included raising funds by public subscription to convert the old Surgeons’ Hall into an auxiliary fever hospital of sixty beds. The Fever Board, a voluntary body, therefore had considerable responsibility in disease prevention and in arranging the treatment of patients with fever or other infectious disease. Their public health function overlapped that of the Police Commission and the Parochial Boards after the establishment of the Scottish Poor Law Board of Supervision in 1845.

A minute of October 1843 reveals the extent to which the Police Commission and the Town Council relied on the Fever Board and the Destitute Sick Society. Deuchar, secretary of the Board and Mr Jack of the Destitute Sick Society were present at a meeting of the cleaning committee when Deuchar read a letter from Dr Alison and presented the books of the Board, indicating that his present staff could not cope with cleaning and fumigating. The difficulties experienced by the Fever Board and the steady increase in the number of fever cases so concerned the Police Commissioners that they considered applying to parliament for a new act ‘to consolidate and improve the existing Police Acts.’ Instead, the Commission instructed the Inspector of Cleaning and Lighting, Alexander Ramsay, to prepare a report on fever and its prevention. Ramsay wrote that his report had been ‘originated in circumstances connected with the recent extensive prevalence of fever and the need to carry out lime-washing of the dwellings of the poor.’ His figures from the records of the Infirmary and the Fever Board show the number of fever cases from 1840 to 2nd December 1843 in table 8.8.

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Deuchar had also collected figures for fever cases admitted to the Infirmary and to auxiliary hospitals in Edinburgh from 1840 to 1843 and his data is presented in Table 8.9. There is a large discrepancy between his figures and those of Alexander Ramsay, the Inspector of Cleaning and Lighting, shown in table 8.8. The difference is considerable for the years 1840 to 1842 and it is only in 1843 that there is any similarity.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1840</td>
<td>786</td>
</tr>
<tr>
<td>1841</td>
<td>1187</td>
</tr>
<tr>
<td>1842</td>
<td>737</td>
</tr>
<tr>
<td>1843</td>
<td>3533</td>
</tr>
<tr>
<td>Total</td>
<td>6243</td>
</tr>
</tbody>
</table>

Table 8.9. Infirmary and Fever Board cases 1838–1843 (Deuchar).

Ramsay and Deuchar both produced figures for fever cases occurring in each month of 1843 and in these there are also discrepancies although the eleven month totals differ by only twenty five cases. There is a pattern which suggests that Ramsay recorded his figures in the month when the person took ill while Deuchar recorded his in the month he received them; in July, for example, Ramsay’s total of 267 cases, seventy eight more than Deuchar is partly balanced the following month when the latter’s total is greater by fifty.

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<table>
<thead>
<tr>
<th></th>
<th>Ramsay</th>
<th>Deuchar</th>
</tr>
</thead>
<tbody>
<tr>
<td>January 1843</td>
<td>48</td>
<td>68</td>
</tr>
<tr>
<td>February 1843</td>
<td>75</td>
<td>74</td>
</tr>
<tr>
<td>March 1843</td>
<td>77</td>
<td>83</td>
</tr>
<tr>
<td>April 1843</td>
<td>91</td>
<td>96</td>
</tr>
<tr>
<td>May 1843</td>
<td>114</td>
<td>134</td>
</tr>
<tr>
<td>June 1843</td>
<td>161</td>
<td>164</td>
</tr>
<tr>
<td>July 1843</td>
<td>267</td>
<td>189</td>
</tr>
<tr>
<td>August 1843</td>
<td>376</td>
<td>426</td>
</tr>
<tr>
<td>September 1843</td>
<td>636</td>
<td>531</td>
</tr>
<tr>
<td>October 1843</td>
<td>489</td>
<td>638</td>
</tr>
<tr>
<td>November 1843</td>
<td>674</td>
<td>586</td>
</tr>
<tr>
<td>Total: 11 months</td>
<td>3008</td>
<td>2983</td>
</tr>
</tbody>
</table>

Table 8.10. Fever cases 1843 (Ramsay and Deuchar).

It is impossible to establish accurately how many fever patients were admitted to the infirmary under the aegis of the Fever Board because of the discrepancies in the figures recorded. The differences may be due to the fact that some calculations were based on the calendar year whereas the Infirmary’s data were based on twelve month periods from the end of September, a practice that fitted with the hospital’s accounting year and the annual meeting of the managers. The Fever Board and Infirmary totals for the period from 30 September 1839 to 30 September 1842 differ by at least 1,000, perhaps because the Fever Board total includes fever patients treated at home.

There was a division of responsibilities between the Fever Board and the Police Commission but the duties of each were not entirely clear to the two administrations in supposed charge. After the establishment of the Scottish Poor Law Board of Supervision

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(1845) the uncertainty as to who was responsible for cleaning and fumigating houses, stairs or closes became even more pronounced. In 1847 on receipt of a letter from the City Parochial Board requesting the Police Commission to clean houses in the Grassmarket, the Inspector of Cleaning informed the cleaning committee that the Parochial Board was mistaken as to the provisions of the Police Act. The Inspector suggested that if fever prevailed in houses which were not lodging houses the Fever Board should be applied to "as the Police Commission annually contribute £100 to the funds of that society, and it would rather be anomalous in the Commission to contribute to the funds of a Society to take steps to arrest the progress of a disease and at the same time be called to perform the work themselves." In June the minutes record a meeting where discussion on the responsibilities of the Police Commission and the Fever Board took place and where the two representatives of the Commission on the Board explained that they had never been called to any meeting of the Fever Board and accordingly could not speak of its funds or operations.128 The dispute continued into the following year when in February the cleaning committee decided that although they were not bound by the Police Statute to clean the houses of the poor, under the prevailing circumstances, they would so do.129 This lack of direction and the absence of concerted effort did not allow the best use of the limited resources available.

The management of fever crises in other cities was very different. In Belfast at the height of the fever epidemic of 1847 a meeting agreed to petition the government to establish a local Board of Health, an application that received immediate sanction. The Board was dissolved in November when its duties were taken over by the Board of Guardians, established under the Amended Poor Law Act of August 1847.130 When the fever of 1847 struck the city of Liverpool the authorities had already appointed a

128 Minute Book of the Cleaning Committee, 8 June 1847.
129 Minute Book of the Cleaning Committee, 26 February 1848.
medical officer of health, Dr W. Duncan, the first such appointment in Britain. The position in Glasgow was in some respects similar to that in Edinburgh but, as Stephanie Blackden makes clear, after 1845 the Parochial Boards in Glasgow had an increasingly important role. Comparing membership of the Boards and the Town Council, she concluded that they had more in common with each other than either had with the Board of Supervision in Edinburgh and as a result the two bodies co‐operated well.131

Until 1843 fever patients once admitted to the infirmary were treated in isolation in special fever wards but this practice changed as a result of evidence from London hospitals. Cormack, in his paper quoted earlier, referred to the distribution of fever patients throughout the hospital the London hospital approach, and one that was eventually adopted in Edinburgh. The Infirmary minutes of June 1842 record Dr Graham’s proposal ‘that their [fever patients] diffusion through the ordinary medical wards with certain precautionary stipulations would be safe and beneficial. The concentration of patients in fever wards was dangerous to the medical and nursing staff since ‘a certain degree of concentration in contagious matter was necessary for its efficacy’ and he could not recollect ‘a single instance of a gentleman acting as his clerk in a fever hospital escaping without an attack of the disease’.132 The minutes of the next meeting record that among the items discussed and implemented were the advisability of bathing of fever patients, their donning of hospital clothes on admission and the space between beds.133 In October the results were analysed and when it was shown to have been fairly successful, in spite of ten patients contracting fever in non fever wards with three deaths, the trial was extended. The one condition (which was not explained) was that fever patients were not to be placed in the servants’ ward, perhaps the reason for this was to minimise the risk of a servant contracting the disease and transmitting it to his or

132 Minute Book of the Royal Infirmary, 13, 1842–1844, 20 June 1842.
133 Minute Book of the Royal Infirmary, 13, 1842–1844, 27 June 1842.
her employers. Cases in which 'no direct communication with fever patients could be demonstrated' were thought to have been caused by mattresses which had been used too soon after a previous fever patient. It was agreed in December to prolong the trial with ten strict conditions: patients to be washed on admission; clothes to be removed immediately and taken away to be cleaned; special mattresses and bedding to be set aside for the sole use of fever patients; sufficient space to be left between beds 'to prevent the atmosphere becoming charged with the exhalations from his person or his bedclothes'; fever patients to be washed regularly; and adequate ventilation to be ensured by fitting ventilation boards to all windows. These deliberations reveal the confusion in the minds of doctors and laymen as to how fever spread. There was a suspicion that mattresses, bedclothes and clothing could transmit the poison but as steps were being taken to prevent this, measures were being implemented to avoid atmospheric spread by diluting the amount of poison in the air. The new system of placing fever patients in general wards was not without its critics and there were concerns about adverse comments if it became public. At the 2 January 1843 meeting it was agreed 'to delay any public discussion of this important and delicate subject ...and the Reporters for the Newspapers should be requested to take no notice of the subject in their report of the proceedings of the meeting.'

It was the threat of cholera and the setting up of the Board of Health (1848) that brought order and some co-operation to the management of the public health and the control of epidemics. In Edinburgh, the constant presence of fever amongst the poor and apprehension about its dissemination throughout the city were used to persuade the more prosperous in society to give to the many charities caring for the sick poor. A lack of money and a shortage of beds for fever patients was a recurring theme in the minutes of

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134 The servants' ward was set aside to receive those who were employed as servants in the city. In 1841 it was proposed that no person 'be admitted to the privilege and seclusion of the Female Servants' Ward except servants of those who contributed to the Infirmary to the extent of one pound annually.', Minutes of the Managers of the Royal Infirmary, 12, Report for 1841.
135 Minute Book of the Royal Infirmary, 13, 1842-1844, 20 June 1842, 10 October 1842, 26 December 1842, 31 December 1842.
136 Minute Book of the Royal Infirmary, 13, 1842-1844, 2 January 1843.
the infirmary managers during the 1830s and 1840s: in 1837, the managers reported an excess of expenditure over income of £1,346 and, although an appeal for donations brought in extra funds, the growth in subscriptions was insufficient to pay for the increased activity in 1838 as the result of fever. Fresh efforts were made to encourage the public to subscribe. The following extract, from the Infirmary contributors’ report, demonstrates the fervour with which the committee played on middle class fears and, in emphasising the duty of Christians to care for the poor, the report is quite explicit that this is not a plea for selfless giving. The Christian doctrine of charity in which there is no thought of reward has been altered to one in which charitable donations can preserve and perhaps guarantee personal and family welfare.

It is physically impossible that epidemics or contagious diseases can long or violently prevail in one class of a society, or in one district of a town, without soon extending in some degree over all. It is impossible that the higher classes can so effectually insulate themselves from their poorer brethren, as that they, and the dearest objects of their regard shall not be frequently exposed to the mortal attacks of any disorder that may gain a general footing. To attack the evil in its stronghold by an efficient system of medical treatment or prevention required money from those who had it. Perhaps this very law of society is a dispensation of providence for enforcing more powerfully the obligations of compassion and beneficence, and it ought to be a source of satisfaction to the comparatively wealthy, that they may thus at once consult the temporary welfare of themselves and families, and discharge those duties to the poor which are among the most essential and conspicuous parts of Christian charity.

The danger from fever was not exaggerated for the Infirmary minutes of 25 February 1839 record the deaths from fever of Dr Brown, the House Governor, and the Apothecary.

It was perhaps the increasing scale of the fever epidemics in the 1830s that forced the Destitute Sick Society to abandon its role and persuaded the leading physicians of the city to become involved in the Fever Board. The Police Commissioners’ £100

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137 Minutes of the Royal Infirmary, 12, 1838–1842, 25 February 1839, pp.147–148.
138 Minutes of the Royal Infirmary, 12, 1838–1842, 12, 14 January 1839, pp.162–163.
139 Minutes of the Royal Infirmary, 12, 1838–1842, 12, 25 February 1839, p.148.
contribution to the Fever Board implied that if they did not function as medical police in
the European sense at least they understood that they had a public health role. The
participation of prominent doctors in the work of the Fever Board hints at a move away
from the concept of health as a private matter towards a realisation that the health of the
community was vital. The acceptance of the link between fever and destitution was a
recurrent theme in the analyses of fever written by physicians in the 1840s; they
recognised that the lack of food, if not the cause, had some part to play in the condition
but they were much less certain of how the contagion was transmitted and were
therefore unsure as to how to deal with the recurrent epidemics. The preferred method
was hospitalisation but then the confusion becomes apparent with a blunderbuss
approach to management in an attempt to cover all possible risks: clothes and mattresses
were to be washed and fumigated, sufficient space was to be left between beds to
prevent accumulation of the poison and the homes from which the patient had come
were to be fumigated. It is hardly surprising that so haphazard an approach was a failure
in Edinburgh as in other cities largely because of the magnitude of the problem. In the
pre germ era doctors were helpless in the face of infectious disease although some
physicians advance theories that produced an embryo public health system when Henry
Littlejohn was appointed the city's first medical officer of health. It is hard to say how
much influence the sanitary movement had in the changes that took place but it is certain
that the concentration of fever and cholera victims in the same neighbourhoods
persuaded the city administration that improvements in housing, water and sewage were
essential. These epidemics undoubtedly had a major impact on society but the epidemics
of measles, whooping cough, smallpox and scarlet fever whose influence was less
striking but which were responsible for great childhood mortality will be described next.

Other Epidemic Diseases

This section is concerned with the epidemics of measles, whooping cough, scarlet fever
and smallpox which appeared in the city at regular intervals during the nineteenth
century. There is no yearly record of the morbidity and mortality from these diseases but
it is possible to gain an impression of their effects from other sources. For example, the Royal Public Dispensary published annually statistics of the previous year's activities and Table 8.11 lists the number of patients treated each year between 1841 and 1846 showing that there were approximately twice as many in 1842 and 1843 as in 1841.

<table>
<thead>
<tr>
<th>Year</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1841</td>
<td>5482</td>
</tr>
<tr>
<td>1842</td>
<td>10888</td>
</tr>
<tr>
<td>1843</td>
<td>11840</td>
</tr>
<tr>
<td>1844</td>
<td>8862</td>
</tr>
<tr>
<td>1845</td>
<td>7416</td>
</tr>
</tbody>
</table>

Table 8.11. Royal Public Dispensary

This increased activity can be partly explained by fever but outbreaks of smallpox, measles, whooping cough and scarlet fever at regular intervals throughout the 1840s were also to blame. St Cuthbert's churchyard records show that in 1841 there were no burials where the cause of death was measles or whooping cough and only one smallpox death. During the first six months of 1842 no measles deaths were recorded but for the rest of the year and for the first eight months of 1843 there were at least three deaths from measles each month, the ages ranging from eleven months to five years. The age range is similar to that reported by Alexander Watt of Glasgow in his report on the vital statistics of large towns in Scotland for 1839, 1840 and 1841. He collected statistics from the recorders at Edinburgh's burial grounds and Table 8.1.2 shows the number of measles deaths in each of these years. Of the 325 deaths only twenty three were over five years of age and only one of these was over the age of ten. For infants at this time measles was a disease which carried a significant risk of death and possible reasons for this high mortality will be discussed later.

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140 Edinburgh University Special Collections, Annual Reports of the Royal Public Dispensary 1841–46.
Table 8.12. Deaths from measles, 1839–1841.  

<table>
<thead>
<tr>
<th>Year</th>
<th>Measles</th>
<th>Smallpox</th>
<th>Whooping Cough</th>
</tr>
</thead>
<tbody>
<tr>
<td>1842</td>
<td>26</td>
<td>23</td>
<td>0</td>
</tr>
<tr>
<td>1843</td>
<td>30</td>
<td>9</td>
<td>70</td>
</tr>
<tr>
<td>1844</td>
<td>11</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>1845</td>
<td>9</td>
<td>15</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 8.13. Deaths from measles, smallpox and whooping cough: St Cuthbert’s Churchyard, Cash Book, 1842, 1843 and 1844.  

The history of Edinburgh’s vital statistics was dealt with in chapter five explaining that the first and only comprehensive mortality records for the 1840s were those collected by Dr James Stark in 1846, 1847 and 1848. Stark collected statistics in 1849 but during the cholera epidemic the town council recommended that although he should continue to collect mortality figures he should not arrange for their publication, a surprising decision, perhaps taken to prevent public unrest. Table 8.14 shows the number of deaths from measles, smallpox, and whooping cough in each of these three years; the figures in parentheses are the deaths from these infections buried in St Cuthbert’s graveyard during the same period.

142 Edinburgh City Archives, St Cuthbert’s Churchyard Cash Book.
<table>
<thead>
<tr>
<th>Year</th>
<th>Measles</th>
<th>Smallpox</th>
<th>Whooping cough</th>
</tr>
</thead>
<tbody>
<tr>
<td>1846</td>
<td>183 (17)</td>
<td>3 (1)</td>
<td>251</td>
</tr>
<tr>
<td>1847</td>
<td>217 (14)</td>
<td>6 (6)</td>
<td>279</td>
</tr>
<tr>
<td>1848</td>
<td>55 (5)</td>
<td>96 (8)</td>
<td>94 (10)</td>
</tr>
</tbody>
</table>

Table 8.14. Stark's mortality figures for 1846–48; burials in St Cuthbert's graveyard are in parentheses.

The analysis of measles deaths in Watt's data, St Cuthbert's records and Stark's statistics suggests that epidemics occurred in 1839, 1842–43 and again in 1846–47. This three to four year cycle is the result of the immunity developed by a cohort of infants infected in earlier outbreaks and partly from the protection bestowed on infants by their mothers. Children born in 1839 and 1842 would have been immune for several months from the transfer of maternal antibodies but by the time of the next epidemic immunity would have lapsed and many would have contracted measles, some dying from secondary infection, frequently of the respiratory tract. As noted in an earlier chapter the susceptibility to respiratory tract infection in children is now thought to be the result of damage to the protective function of the lining of the respiratory tract from vitamin A deficiency.

In 1845 infectious disease accounted for 2,522 of the total of 7,416 cases treated at the Royal Public Dispensary, table 8.15. Epidemic fever was by far the biggest killer, followed by whooping cough and measles but St Cuthbert's 1845 figures for measles and whooping cough deaths show no change from that of previous years with nine measles deaths and four deaths from whooping cough suggesting that either the epidemic was not associated with high mortality or if there was increased mortality those who died were buried elsewhere.
Epidemic Fever | 2150
Whooping Cough | 268
Measles | 87
Smallpox | 10
Scarlet Fever | 7

Table 8.15. Royal Public Dispensary, 1845.143

Stark does not have a category for diphtheria, a disease that was often confused with croup and scarlet fever and listed under these headings. The total mortality from measles, whooping cough, scarlet fever and croup in the three years 1846–1848 for which figures are available amounted to 1,468. In 1846 there were 251 deaths from whooping cough and 183 deaths from measles; in 1847, 279 whooping cough fatalities and 217 from measles; in 1848 there were 270 deaths from scarlet fever. These figures indicate the lethal nature of these diseases in a non immunised population of children whose natural resistance was impaired by nutritional deficiencies and social deprivation.

Cholera

This section is concerned with cholera, a disease that reached Britain in the autumn of 1848, the second great epidemic to reach the British Isles. The danger from cholera was fresh after the previous outbreak in 1832 and its appearance when typhus and relapsing fever were still rife stretched the already overburdened hospitals and municipal authorities. Before examining how cholera affected the city the history of the disease and its identification will be reviewed, following which its characteristics and mode of transmission will be examined. The reasons for the virulence of the organism and the variations in host susceptibility will be discussed before describing the clinical features of the illness. The discovery of two volumes of the records of cholera patients in the archives of the Royal College of Physicians of Edinburgh was a significant find and has yielded material for a database of the 740 cases. This will be analysed later in the

chapter: a detailed appraisal in collaboration with a colleague is planned with a view to publication.

The gastro-intestinal disorder called cholera is caused by a bacterium which is peculiar to man and frequently appears in epidemic form. The term was first used by Hippocrates to describe an illness like mild cholera but there is no convincing etymological explanation for the word except the suggestion that it derives from the Greek word, cholera, meaning diarrhoea.\footnote{S.N.De, \textit{Cholera: Its Pathology and Pathogenesis}, (Edinburgh, 1961), pp.3 & 4.} In the mid nineteenth century, Dr John Snow, the London physician, whose work on cholera in London is discussed later, argued that Asiatic Cholera could not be positively identified in India before 1769 but, since the greater part of the country was unknown to European physicians, outbreaks of the disease may not have been recognised or recorded.\footnote{J.Snow, \textit{On the Mode of Communication of Cholera}, p.1.} More recent authorities consider that the disease is considerably older; Pollitzer in 1965 cited Garcia da Orta as the first medical writer on the illness in his treatise on Indian medicine published in 1563. In an earlier monograph (1959), Pollitzer refers to an account by Gaspar Correa of a cholera like illness affecting the soldiers of the army of the king of Calicut in 1503.\footnote{R.Pollitzer, ‘Cholera Advances in Historical Perspective’, \textit{Proceedings of the Cholera Research Symposium}, (Washington, 1965), p.380; R.Pollitzer, \textit{Cholera}, (Geneva, 1959), p.12.} Underwood in 1948 quoted Macnamara whose nineteenth century history of the disease includes references to outbreaks in India in the fifteenth century.\footnote{E.A.Underwood, ‘The History of Cholera in Great Britain’, \textit{Proceedings of the Royal Society of Medicine}, 61, (1968), p.1.} Professor De, who has written extensively on cholera from his experience of the disease in Calcutta, may be alone in believing that a type of cholera existed in Europe long before the arrival of the first pandemic in the 1820s and 1830s. This new, severe, epidemic form, he suggests, was given the name cholera because of its similarity to an earlier, milder disease and in order to distinguish this illness from so called British Cholera a qualifying descriptive adjective was added, for example, Asiatic, Oriental, or epidemic. Pollitzer contends that the disorder prevalent in London between 1679 and 1682 and called cholera by Sydenham never became epidemic, was confined to the metropolis and was a different
disease to Asiatic cholera.\textsuperscript{148} Accepting the evidence of these authorities, it is probable that the first great pandemic was a disease new to the west and that cholera had existed in the east for some hundreds of years before it reached the west.

The first pandemic is generally agreed to have started in the second or third decade of the nineteenth century but there is less unanimity about the number of pandemics and when each began and ended. Ackerknecht says the first pandemic started in 1826 and ended in 1837.\textsuperscript{149} Cartwright argues that ‘the fateful year’ was 1817, when the disease which had confined itself to India for hundreds of years suddenly and inexplicably erupted in worldwide epidemics. He suggests that there were six great and two lesser pandemics between 1817 and 1902.\textsuperscript{150} Pollitzer considers that the first pandemic started in 1817 and ended in 1823; the second lasted from 1829 to 1851; the third from 1852 to 1859; the fourth from 1863 to 1879; the fifth from 1881 to 1896 and the sixth from 1899 to 1923.\textsuperscript{151} If Pollitzer’s opinion is accepted, the second cholera pandemic swept through Europe in 1831–32 and again in 1848–49. The ‘devastating epidemic’ in Bengal in 1817 had repercussions throughout Asia, Europe and America; for the first time the disease involved the west triggering an interest in British university medical schools that had not hitherto been apparent.\textsuperscript{152}

The proof of the germ theory lay decades in the future but, as will be shown later in the chapter, from the 1830s on fungal and animalculi theories for cholera were being investigated. It is now known that the organisms responsible for cholera belong to the genus \textit{Vibrio}, ‘a name which attempts to catch the flash of movement as these minute

\textsuperscript{150} F.F.Cartwright, \textit{Disease and History}, (New York, 1972), pp.157 & 158.
\textsuperscript{152} Underwood, ‘The History of Cholera in Great Britain’, p.165; Underwood cites three Edinburgh MD theses on the subject of cholera between 1822 and 1827.
bodies dart across a dark microscopic field.\textsuperscript{153} \textit{Vibrio cholerae} is the type species of the genus but the etiologic agent has been known by many names: \textit{bacillus cholerae}, \textit{vibrio cholerae–asiatica}, \textit{water vibrio}, \textit{cholerigenic vibrio}, the cholera bacillus, the comma bacillus (so called because of the shape of the bacterium) and many others.\textsuperscript{154} Filippo Pacini (1812–1883), an Italian anatomist and histologist, first used the name “vibrio” when he described the “milioni di vibrioni” in the intestines of cholera victims during the 1848 epidemic in Florence and suggested that these “\textit{vibrio cholerae}” were the cause of the disease.\textsuperscript{155} Pacini’s primary interest was in microscopical research, (he designed his own microscope in 1844), and this interest and expertise enabled him to identify the microorganisms in the dejecta of cholera victims and to describe the intestinal lesions they produced.\textsuperscript{156} He published his findings in 1854 but little notice was taken at the time and over a century passed before his work received the international recognition it merited when the International Committee on Bacteriological Nomenclature in 1965 recognised Pacini’s discovery and recommended that \textit{vibrio cholerae Pacini 1854} should be adopted as the official name for the bacillus.\textsuperscript{157} Theories of a \textit{contagium vivum} had circulated from as early as 1819 when, as a result of an investigation into the cholera epidemic of 1817, the Bengal Medical Board had declared that the disease was caused by ‘a pestilential virus, which acted primarily upon the stomach and small intestines.’\textsuperscript{158} This did not have much impact on contemporary medical thinking, obscured by the debate between contagionists and those who were convinced that atmospheric influences, foul air and filth were to blame.

\begin{itemize}
\item \textsuperscript{153} A.B.Christie, \textit{Infectious Diseases: Epidemiology and Clinical Practice}, (Edinburgh, 1980), p.125.
\item \textsuperscript{155} Pollitzer, ‘Cholera Advances in Historical Perspective’, p.381. The article he cites is by F.Pacini, ‘Osservazioni microscopiche e deduzioni patologiche sul Colera Asiatico’, \textit{Gazzetta Medica Italiano Toscana}, Serie 2, Tomo 4, 6, (1854), pp.405–412.
\item \textsuperscript{156} Castiglioni, \textit{A History of Medicine}, p.678. Curiously, Castiglioni gives little prominence to his compatriot’s discovery, simply stating the year and the circumstance.
\item \textsuperscript{158} Pollitzer, ‘Cholera Advances in Historical Perspective’, p.380.
\end{itemize}
Pacini may not have been first; the German scientist, Boehm, claimed in 1838 to have seen the causative organisms of cholera under the microscope.\(^{159}\) Pollitzer doubts the validity of Boehm’s claim but accepts that of the Frenchman, F.A. Pouchet, (1800–1872), who stated that he had seen microscopic bodies in the dejecta of cholera patients during the epidemic of 1848–49. Pouchet’s findings were published in a letter to the Paris Academy of Sciences in April 1849 in which he described the animalculi he had found in the characteristic rice-water stools of cholera victims, emphasising that these were seen only if the microscopic examination was carried out soon after the stools were voided.\(^{160}\)

In reviewing the theories of contagion and miasma, the Royal College of Physicians of London concluded in 1831 that cholera was contagious and, as a result, the government, in an effort to contain the epidemic, ordered quarantine measures. Despite the College’s verdict, official support for the contagious theory of cholera lasted less than twenty years when the epidemic of 1848 was met with a declaration by the newly formed General Board of Health that the disease was non-contagious, a decision influenced to a great extent by the fact that ‘the recently created Board of Health, like its secretary Edwin Chadwick, was aggressively miasmatic’.\(^{161}\) De thinks that this may have been influenced also by the failure of quarantine to control the spread of cholera in earlier epidemics and that the inevitable loss of trade from the enforcement of quarantine may have been another factor.\(^{162}\) The result was that laymen and physicians became convinced that contagion theory was mistaken, a conviction that was greatly influenced not only by the enthusiasm for sanitary reform of Chadwick but also by the evidence of Arnott, Southwood Smith and Kay–Shuttleworth. A variety of non-contagion theories became popular and De concludes that ‘the manufacture of so many theories clearly indicates not only the extensive interest in disease causation but also the confusion of

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\(^{162}\) De, *Cholera*, pp.3–6.
scientists at that time over the origin and nature of cholera. It shows also how the theory of contagion that gained general acceptance by the medical profession during the preceding pandemic was now threatened with possible dissolution.\(^{163}\) Another historian of nineteenth century cholera, Richard Evans, wrote ‘the weight of opinion was firmly against contagion, thus reinforcing the decision of many governments to do nothing,’\(^{164}\) The General Board of Health report dismisses out of hand any possibility that contagion might have been responsible for cholera in nurses and a washerwoman, explaining that these unfortunate women had contracted cholera because ‘there had been premonitory diarrhoea which had been neglected’, or there was evidence of ‘over fatigue’ or there had been ‘some act of intemperance.’\(^{165}\) The Board put forward reasons for the disease; overcrowding, filth, malaria from putrescent mud, dampness, want of drains and bad drains, graveyards, unwholesome water and finally food. In support of these, many examples and reports of cases of cholera appearing where such conditions existed were advanced.\(^{166}\) There is an obvious similarity between these examples of conditions which the Board claimed provoked cholera to those put forward by Chadwick and his colleagues as the cause of fever.

It was at this point that the theory of contagion should have been given fresh impetus by the work of Dr John Snow (1813–58) if this had received the attention it deserved. In the epidemic of 1831–32, Snow as a young surgical apprentice in Newcastle had attended cases of cholera and became convinced that the ‘morbid poison’ of cholera entered by the alimentary canal.\(^{167}\) Snow’s observations and conclusions were published in a classical monograph of epidemiological research, *On the Mode of Communication of Cholera* (1849), in which he wrote ‘diseases which are communicated from person to person are caused by some material which passes from the sick to the healthy, and which

\(^{163}\) De, *Cholera*, pp.6–9.


\(^{165}\) Report of the General Board of Health on the Epidemic Cholera, p.34.


has the property of increasing and multiplying in the systems of the persons it
attacks.168 He continued:

Now many medical men, whilst they admit the influence of polluted water on the
prevalence of cholera, believe that it acts by predisposing or preparing the system
to be acted on by some unknown cause of the disease existing in the atmosphere or
elsewhere. ...if the effect of contaminated water be admitted, it must lead to the
conclusion that it acts by containing the true and specific cause of the malady,...if
the contaminated water merely acted by predisposing or preparing the system to be
affected by some other cause, it would be impossible to explain why nearly all the
persons drinking it should be attacked together, in cases where a pump–well or
some other limited supply is polluted, while the population around experience no
increase of the malady.169

Pollitzer observes that these seemingly irrefutable contentions attracted at the time as
little attention as did the claims of Hassall, who, when examining cholera stools during
the 1854 epidemic in London, found ‘myriads of vibriones in every drop of every
sample of rice water discharge’ that he examined.170 Miasma theory, vigorously
promoted by Chadwick and his supporters, was in the ascendant at least for a period
until the development of the discipline of bacteriology.

Robert Koch (1843–1910), a German bacteriologist, is accredited with the discovery of
the cholera bacillus.171 Koch went to Egypt as the leader of a German Commission to
investigate the clinical, epidemiological and bacteriological aspects of cholera during an
epidemic in 1883 where he isolated what he called the comma shaped bacillus and
successfully demonstrated its presence in the intestines of ten cholera victims. At the end
of the epidemic he went on to Calcutta and there he confirmed his original findings
detecting bacilli in material from forty two post mortem examinations carried out on
cholera victims and in thirty two samples of cholera stools. Koch’s sophisticated

170 De, Cholera, p. 9; Politzer, ‘Cholera Advances in Historical Perspective’, pp.381–383; E.D.Mintz,
T.Popovic and P.A.Blake, ‘Transmission of Vibrio Cholerae O1’ in I.K.Wachsmuth, P.A.Blake and
Ö.Olavik, (eds.), Vibrio cholerae and Cholera: Molecular to Global Perspectives, (Washington, 1994),
p.346.
accrediting Koch with the discovery of the cholera bacillus.
laboratory equipment enabled him to describe in detail the properties of the microorganisms to which he gave the name 'Kommabacilli', apparently unaware of Pacini's earlier work.\textsuperscript{172} Koch's findings were met with determined opposition, some of which, it has been alleged, may have been the result of national bias preventing objectivity.\textsuperscript{173} Some of the disagreement was extreme—eleven European researchers swallowed cultures of \textit{Vibrio cholerae} in an attempt to prove that the bacillus acting alone could not produce cholera. It is astonishing that none of the eleven developed the disease, an outcome which De says 'demonstrated that the implantation of the bacillus does not necessarily ensure the establishment of the disease.'\textsuperscript{174} The opposition to Koch was carried to extraordinary lengths by Max von Pettenkofer (1818–1901), a German hygienist, physiologist and epidemiologist who did not deny that the bacilli existed, agreeing that in the right circumstance the vibrios could cause cholera. Pettenkofer argued in environmental terms putting forward what Porter describes as 'a version of the miasmatic theory', claiming that the vibrios survived only in a soil with a certain level of ground water and that spread of the disease occurred only when these conditions were met. In 1892, he persuaded Koch to send him a culture of cholera vibrios. In a letter he expressed his gratitude to Koch for letting him have the sample and assured him that he remained in good health after swallowing the vibrios.\textsuperscript{175} This episode is merely an extreme example of the lingering influence of non contagion and the belief that other precipitating causes acted in conjunction with the bacteria to produce the disease. As late as 1903, a manual of medicine and hygiene for use in India claimed that 'the precise cause of cholera is not known, but is generally admitted to be a poison, ... a peculiar microbe (the "comma" \textit{bacillus} or \textit{spirillum}) has been found in the intestines and

\textsuperscript{172} Pollitzer, 'Cholera Advances in Historical Perspective', p.382. Koch published his findings in 1884 in the \textit{Deutsche medicinische Wochenschrift} and in the \textit{British Medical Journal}, 2, (1884).
\textsuperscript{173} Ackerknecht, \textit{History and Geography of the Most Important Diseases}, p.30.
\textsuperscript{174} De, \textit{Cholera}, pp.17–18.
discharges of cholera patients; but there is not sufficient evidence to show whether this is the cause of the disease.\textsuperscript{176}

Despite these opinions, in time the causal link between \textit{vibrio cholerae} infection and the clinical condition became accepted and bacteriological, chemical and immunological research into the bacillus developed leading to a vast increase in knowledge of the behaviour of vibrios, how they are transmitted and how they achieve their lethal effects. The pathogenicity of \textit{Vibrio cholerae} is due to the cholera enterotoxin (CT) which triggers the massive outpouring of fluid and electrolytes from the blood into the small intestine of cholera patients. The motility of the vibrio enables it to penetrate the mucus layer of the intestinal epithelium and to adhere to its surface, actions that allow the enterotoxin to gain access to the cells of the small intestine and bring about the loss of fluid typically seen in the acute stage of the disease.\textsuperscript{177} It is not only the fluid loss which is catastrophic to the victim, the loss of salts and minerals also has a major impact on the body's metabolism.

Until the 1980s \textit{V. cholerae} was believed to be incapable of surviving for more than a few days outside the human intestine but research has shown that vibrios have survived for over a month in sewage and that they are capable of living for up to thirty days in clean tap water. More recent work has revealed \textit{V. cholerae} in brackish water and estuaries, frequently in a dormant state but capable of becoming active if the environmental conditions are suitable.\textsuperscript{178} Man is the only known host of \textit{V. cholerae} and it is man who contaminates the environment. A cholera patient may excrete between one and sixty litres of fluid, the typical amount lying between ten and twenty litres and in the acute phase there are several million vibrios in each millilitre of fluid stool, thus the


\textsuperscript{177} Christie, \textit{Infectious Diseases}, p.125.

potential for environmental contamination is enormous.\textsuperscript{179} Cholera is generally spread by faulty or absent sanitation when infected faeces pollute water supplies. When \textit{V. cholerae} 'by some epidemiological mischance finds its way into a normally safe public water supply the outcome can approach the catastrophic.'\textsuperscript{180} Water for domestic use may also become contaminated during household storage and research has shown that this is particularly likely to happen when drinking water is kept in wide mouthed open containers which allow the entry of unclean hands contaminated by vibrios.\textsuperscript{181}

Food contamination can occur when there is a failure in hygiene on the part of a food handler or preparer, when contaminated water is used to wash the food or when contaminated sewage and night soil is used to fertilise growing crops.\textsuperscript{182} The nature and type of food and the conditions in which the food is kept influence the survival of vibrios, being enhanced by low temperatures, a near neutral pH, high moisture content and the absence of competing bacteria.

The final mode of transmission to be considered is by fomites, which is dependent on the length of time vibrios survive on certain materials and objects, in turn influenced by the degree of contamination, temperature, pH, moisture, salt content, presence of organic matter, texture and on the nature of the substance. Vibrios can live on linen for five days at room temperature and on moist linen strips for five weeks, for four days on wool, two days on leather; on coal and certain metals for one or two days; on tobacco for three to seven days; on cutlery for one or two days. One bacteriologist considers that it is theoretically possible for infection to be transmitted by fomites in a family group but says that it is doubtful if the dose of vibrios would ever be large enough and evidence for direct person to person spread is hard to find. There is confusion as to the meaning of

\textsuperscript{179} Christie, \textit{Infectious Diseases}, p.128.
\textsuperscript{181} Mintz, Popovic and Blake, 'Transmission of \textit{Vibrio cholerae} O1', p.348.
“person to person”, some defining the term as direct contact only, while others include in the definition contamination of food and water within a family group or household. In this thesis direct person to person transmission is understood to mean through direct contact only.\textsuperscript{183} Contact infection may be direct when the dejecta of a victim soil the hands of a family member or neighbour and poor hygiene then permits the transfer of vibrios into the mouth of the contact; in other cases the contact may be indirect through shared food, water or household utensils.\textsuperscript{184}

‘Man is not easily infected except by very large doses of \textit{V. cholerae}, and large doses are likely not from direct personal contact but only when a person swallows mouthfuls of heavily contaminated water or food.’ The same authority contends that if a plentiful supply of pure water for drinking and washing is available cholera does not take hold and spread and that the prevalence of the disease is related to overcrowding, poor sanitation and polluted water; it is a disease of poor environments.\textsuperscript{185} The observation that contacts can share food, water, sanitary facilities, the same house and sometimes the same bed as a cholera patient and yet show no evidence of the disease or only minor signs or symptoms of a sub clinical infection has puzzled many investigators. Although poor hygiene, overcrowding and lack of sanitation, as emphasised by Pollitzer, can account for many of these inconsistencies it has been shown that there are other factors at work.\textsuperscript{186} These epidemiological puzzles have led to an examination of the concept of individual susceptibility, an aspect of the epidemiology of cholera that has been investigated with results that suggest several possible explanations of these variations.

Chronic under nutrition harms the immune response and may also harm the body’s ability to respond to illness once established. There is no firm evidence that malnutrition


\textsuperscript{184} Pollitzer, \textit{Cholera}, p.846.

\textsuperscript{185} Christie, \textit{Infectious Diseases}, p.130.

increases susceptibility to cholera although it has been postulated that the lower level of stomach acid found in the malnourished may increase the risk of contracting the disease.187 In certain studies blood group has been shown to influence susceptibility, persons with blood group O are significantly more at risk and group A more resistant. Those with low or absent stomach acid are at greater risk of developing cholera and the converse is also true, those with high stomach acid have greater protection. Beer drinking is known to increase acid production and therefore has a protective effect, whereas cannabis smoking reduces gastric acid, possibly explaining the prevalence of hypochlorhydria (low gastric acid) in parts of India and the high incidence of cholera.188 Recent work on the micro organism Helicobacter pylori concludes that its presence may increase the risk of cholera infection by damaging the gastric acid defence mechanism and it is interesting that its incidence is in indirect proportion to the general level of sanitation, similar to cholera. Colonisation with this organism causes irreversible damage to the stomach lining, diminishing gastric acid and the position has been summarised thus: ‘an organism that is probably transmitted person to person by the same faecal–oral route as Vibrio cholerae...may increase the risk of cholera infection by lowering the pH barrier so that fewer vibrios are required for infection and disease.’189 Normal gastric acid provides protection against the vibrio, as demonstrated by the experiments carried out by Pettenkoffer and R.Emmerich (1852–1914) who swallowed bicarbonate followed by vibrios in water. Pettenkoffer suffered from slight diarrhoea only but Emmerich developed severe cholera, an outcome that illustrates the complexities of individual susceptibility and outcome.190

Most people who become infected with V. cholerae do not develop cholera, remaining asymptomatic, but none the less they are infectious and can spread the disease. It has

188 Christie, Infectious Diseases, p.132.  
189 Richardson, ‘Host Susceptibility’ p.275.  
190 Christie, Infectious Diseases, pp.131 & 132; Richardson, ‘Host Susceptibility’, pp.274 & 275.
been estimated that the asymptomatic may outnumber those with symptoms by a factor of seven to one, an estimate which gives some indication of the difficulty in controlling spread.191

The incubation period of cholera varies according to the dose of vibrios and individual susceptibility; it can be as short as twelve hours or as long as five days but more usually it is two or three days. There are few prodromal symptoms, the illness commencing with moderate to severe diarrhoea and vomiting, both of which are usually uncontrollable. Within an hour or two the so-called rice-water stools appear; the fluid is clear, devoid of faecal matter and is simply a watery solution of salt, bicarbonate, potassium and other minerals secreted into the small intestine. The massive amount of fluid flows along the intestinal tract to the exterior without effort on the part of the patient and unless the loss of fluid is made good severe dehydration results. When the fluid loss approaches 5 per cent of body weight the pulse quickens and at this stage the patient is extremely weak and worried. Fluid continues to pour from the rectum, blood pressure falls, urine production diminishes and the loss of fluid is such that the victim is in shock and is critically ill. The patient is cold, with a dry mouth and if he can speak he or she complains of great thirst, of pain and cramps in the limbs; his breathing is rapid, he is now passing no urine and although still conscious he is becoming confused. The rapid and progressive dehydration is accompanied by extreme thirst and he appears shrunken. Without intravenous infusion of fluid and salts he is doomed. The death rate in untreated outbreaks can exceed 50 per cent but is reduced to 10 per cent or less with early effective treatment.192

The rapidity with which the disease struck and the appalling image of the dying cholera victim, ‘perhaps young and attractive in the morning, by nightfall they had become shrivelled wrecks with darkened bluish skin, sunken eyes and protruding teeth,’ explain

191 Christie, *Infectious Diseases*, p.137.
why no affliction was feared more than cholera. Death from cholera was called a dog’s death, *mort de chien*, or the blue terror.\(^{193}\) The French physician Magendie described cholera as “a disease which begins where other diseases end, with death”, cholera cadaverised its victim.\(^{194}\)

Creighton says that the cholera epidemic in Edinburgh lasted from 1 October 1848 until 18 January 1849, affecting 801 persons with either 448 or 478 deaths, of which 196 were males and 282 females.\(^{195}\) He records that a Cholera Hospital was opened in Surgeons’ Square on 28 October and that of 248 admissions up to 14 December, 154 died. The epidemic came through Russia which it reached from Kabul in 1847, following the caravan routes through Samarkand, Bokhara and Astrabad, within a year one million people died of the disease in Russia alone.\(^{196}\) In August 1848 cholera was in Poland and from then on the *Scotsman* reported its progress.\(^{197}\) A parliamentary debate on the Health of Towns Bill in which Lord Morpeth referred to the progress of cholera, quoting communications from British consuls at foreign ports, and suggesting that the imminent epidemic might prove to be an additional inducement to sanitary reform. The Scotsman commented that the absence of provision in the bill to extend the new sanitary measures to Scotland meant that ‘we are left wholly unprepared, and we greatly fear that our cities and towns will remain as dirty, undrained and ill-ventilated as ever, till the actual entrance of the dreaded plague shall arouse us to some feverish efforts of hasty and superficial reformation.’\(^{198}\) The citizens were also becoming anxious; on 18 September a ‘Stockbridge resident’ complained to the *Scotsman* of the filthy condition of the Water of Leith, reminding the public that the 1832 epidemic had broken out in the Water of Leith village — a claim that is not borne out by accounts of that epidemic. Nevertheless

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193 De, *Cholera*, p.2.
196 Scotsman, 30 August 1848 reported cholera in Poland and referred to the measures adopted in Paris to deal with the expected epidemic; 9 September, cholera in Lithuania and Estonia.
197 Scotsman, 12 August 1848.
it is plain that conditions in the city were causing alarm and the feared epidemic was not long in arriving.199

The first case appeared at Newhaven, a fishing village on the Firth of Forth, on 1 October, the first cases in Edinburgh were seen the following day and in Leith seven days later. On 7 October the *Scotsman* reported six to eight cases in the West Port, the Grassmarket, Canongate and Low Calton and three or four in Newhaven. The source of the outbreak in Edinburgh and neighbourhood cannot be identified but it is likely that it was brought from mainland Europe by sea. In the weeks immediately before the outbreak ships had docked in Leith from Hamburg, Rotterdam, Riga, Dantzig, Antwerp, St Petersburg and Hull.200 There were three ships from Hull at Leith in late September and early October and although the official report on the epidemic states that the Edinburgh cases were the first in Britain, there is a later reference to cholera in Hull and London in September. Creighton says that the infection reached Scotland within a few weeks of its appearance at Hamburg on the German coast.201 Cholera was expected in Edinburgh but, as will be shown, the municipal and medical authorities were relatively unprepared.

The Royal College of Physicians of Edinburgh minutes record a meeting on 12 October 1848 ‘to consider the steps to be taken by the College with reference to the present appearance of Cholera in Edinburgh.’ The President, Robert Christison, explained that in January the Councils of the two Royal Colleges had met with representatives of the Town Council to prepare for the cholera ‘but that the time for preparation had not arrived.’ Christison went on to say that in August he had told the Lord Provost ‘that the necessary preparations ought to be no longer delayed.’ As a result of this conversation a preliminary meeting of the two College Presidents with the civic authorities took place

200 *Scotsman*, 20 September 1848, 23 September 1848, 27 September 1848, 30 September 1848 and 4 October 1848.
on 29 September and a full meeting was held on the 5 October at which were present representatives from the Parochial Boards of Edinburgh.\(^{202}\)

The President’s account of the meeting of 5 October stated that all present were anxious to implement urgent measures to deal with the threat but, subsequent to the passing of the act for removal of nuisances and prevention of epidemic diseases, the initiative had been taken from local authorities and given to the General Board of Health in London.\(^{203}\) The opinion of the meeting was that no measures could be adopted, except for cleaning and removing nuisances, without the approval of the London Board and accordingly ‘the Lord Provost was instructed to beg that the Board would immediately announce the measures which they deemed advisable, because the Cholera had appeared in more quarters of the city than one since the 1\(^{st}\) instant.’\(^{204}\) Christison said that he had written to Mr Chadwick, a Board member, with whom he was personally acquainted, ‘urging the necessity of immediate measures’ and as a result the Board had sent Dr Sutherland, to assist the Edinburgh authorities.\(^{205}\) Christison told his colleagues that a Local Board was now in place, consisting of the Lord Advocate, the Dean of Guild, the Sheriff of Edinburgh, the Chairman of the Board of Supervision for the Poor and the two College Presidents.\(^{206}\) This Board had already met and had prepared several important measures but had delayed their implementation ‘on account of a doubt being entertained as to the extent of the Powers conferred by the General Board of Health upon the Local Board.’\(^{207}\)

The impression given is that the appearance of cholera was not being taken sufficiently seriously by the General Board who were not reacting with the urgency that the Edinburgh committee expected. The letter from Christison to Chadwick conveys this...

\(^{202}\) Minutes of the Royal College of Physicians of Edinburgh, pp.3732 & 3733, 12 October 1848.

\(^{203}\) P.P., Act to Amend the Nuisances Removal and Disease Prevention Act, 1848, 12 and 13 Vict., cap. cxii.1849.

\(^{204}\) Minutes of the Royal College of Physicians of Edinburgh, p.3733, 12 October 1848.

\(^{205}\) Minutes of the Royal College of Physicians of Edinburgh, p.3734, 12 October 1848.

\(^{206}\) The Dean of Guild was an ex officio member of the council appointed on the recommendation of the Incorporation of the Brethren of the Guildry, an organisation that protected the interests of those who worked in skilled occupations or commercial concerns.

\(^{207}\) Minutes of the Royal College of Physicians of Edinburgh, p.3734, 12 October 1848.
point of view very clearly. 'If this be the advent of the epidemic we are taken quite unprepared... its [the cholera] slow progress on the Continent led us here, as it seems to have also led you all in London, to be slow to believe that any great preparation would be necessary... the initiative rests with your Board.'

On 7 October the Police Commissioners' Cleaning Committee met and instructed the inspector to employ thirty extra men and, since cholera followed in the track of epidemic fever, Dr Glover, the police surgeon, was instructed to apply to the managers of the infirmary, dispensaries and parochial boards for access to their fever records. On the same day a letter from Dr James Stark was published in the Scotsman in which he wrote that the report of two cases of Asiatic Cholera at Dundee, and three at Hull, led him to enquire 'why nothing as yet been done in Edinburgh to prepare us for a visitation of this formidable epidemic.' Stark praised the 'able reports' of the Metropolitan Sanitary Commission which 'proved to demonstration, that this disease is not infectious, is not communicable from the sick to the healthy, from the dead to the living.'

There were disagreements among medical personnel; Dr Glover complained that Dr Stark had demanded access to reports of cholera which was refused but later Stark presented a signed order from Dr Sutherland, countersigned by the Lord Provost, requesting Glover to hand over the documents, explaining that he had been requested by the General Board of Health 'to procure and report to them the numbers of cases of cholera as they occurred.'

There were other administrative disagreements as when the Parochial Boards' instructions interfered with the duties of the Police Commissioners as laid down by Act of Parliament and the minutes recorded 'as much good might be done by the united efforts of the Parochial Boards and Commissioners of Police, it is desirable that the jurisdictions of the respective Boards be preserved and an effective co-operation secured.'

The disputes continued for several weeks as to which body was responsible...
for the cleaning of houses, closes and stairs and who should pay for these activities. Finally a meeting on 8 November of representatives of the Police Commission, the Parochial Boards and the two medical colleges agreed that the Police Commission would undertake external cleaning and the Parochial Boards would be responsible for internal cleaning. There were further rows involving Dr Glover and his employers concerning discrepancies in Glover’s figures for cholera cases and those of the College of Physicians and concerns about his expenses, the commissioners regretting that he had exceeded his authority incurring more than he was allowed but finally agreeing to pay him sixty three pounds. Dr Glover was in the unenviable position of having to investigate cases of cholera, to collate returns, co-operating with the local boards, the medical colleges, the Board of Health in London and Dr John Sutherland, the Board’s representative in Scotland.

Professor Christison in his letter to Chadwick quoted above, wrote ‘it is no matter whether the disease arises from infection, Malaria, or any other cause — in any view one may take of it there is a Local cause which, if it do not produce, at all events promotes, the disease……’. This equivocation may have reflected genuine doubt in Christison’s mind or may have been included to forestall unwelcome and, in Christison’s view, unhelpful proposals from Chadwick and the Board of Health. There is no doubt that in 1832 Christison believed in contagion. In a letter to David Moir (1798–1851), a Musselburgh surgeon and member of the town’s Board of Health during the epidemic of 1831–32, Christison indicated his support for ‘the doctrine of contagion’ and suggested that Moir arrange to record the names of people from nearby localities in the habit of visiting towns where cholera was present but who did not enter the houses where there were cholera patients. Christison believed that the results would show ‘that scarcely any such person has been attacked’, clear evidence in support of the contagion theory.

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214 Royal College of Physicians of Edinburgh, Letter from Christison to Chadwick, 5 October, 1848.
215 Royal College of Physicians of Edinburgh, Letter from R. Christison, 15 March 1832 in Cholera Correspondence, Moir Papers.
During the sixteen years between the first and second cholera epidemics miasma theory gained increasing acceptance, largely through the prominence of the sanitary movement, and it is possible that Christison began to be persuaded that contagion might not be the whole explanation.

Creighton says that the epidemic began in Newhaven and Edinburgh on the 1 and 2 of October respectively, at Leith on 9 October and maintains that the Edinburgh outbreak ceased on 18 January 1849 having been responsible for 801 attacks with either 448 or 478 deaths, 196 males and 282 females.\(^{216}\) He does not explain why he gives two figures for the number of deaths and it is equally strange that he entirely omits the second wave of attacks in the summer and autumn of 1849. Creighton’s inaccuracies are difficult to explain since he must have been aware of the official report on the epidemic which, although critical of Scottish mortality statistics, did at least have records as to when the epidemic began and ended.\(^{217}\) Stark’s statistics seem also to have been ignored by Creighton and the City graveyard records were not mentioned. St Cuthbert’s figures are not only accurate but well kept and show that twelve cholera victims were buried in November 1848, eleven in December, seven in January and one in February, the first victim being Mary Hossack, aged forty nine from Hastie’s Close, Canongate.\(^{218}\) These graveyard records are interesting but of limited use since complete records of all the city’s graveyards are no longer available but there is a comprehensive record of the epidemic.

**Returns of Cholera Cases, Royal College of Physicians of Edinburgh 1848–49**

This hitherto unused and unrecognised source of whose existence Creighton appeared unaware, consists of two volumes in which were recorded the names and details of 740

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\(^{218}\) Edinburgh City Archive, St Cuthbert’s Churchyard Cash Book, 1847–1854.
cholera patients from Edinburgh and those from neighbouring towns and districts such as Leith, Portobello and Dalkeith and from more distant places in the Scottish Borders.219 The returns, which were completed by the reporting medical officer and sent to the College, consist of a pre-printed form in which was recorded the name of the patient, his/her age, occupation, address, state of living accommodation, habits of victim, contact with other cholera patients, date of onset and outcome, outcome, treatment, whether treated in hospital or at home and whether a post mortem examination was carried out. The data present in these returns is an extremely detailed and valuable resource from which a database has been prepared (Appendix 1). Although certain analyses have been carried out for the purpose of this thesis, it is considered that the material in the College returns is more extensive than can be dealt with adequately in this thesis and accordingly the contents of the volumes will be the subject of a future detailed, collaborative analysis.

The returns show a case of cholera in late August 1848 but it is improbable that the diagnosis was correct and it is more likely that the first case occurred in Edinburgh on 3 October 1848. The first wave lasted until April 1849 after which there were no further cases until 12 July 1849 when a second outbreak began and continued until the end of the year. The number of cases in the first wave was 534 and in the second 185. The present analysis has not reviewed the diagnoses but from the study that has been carried out it appears that at least some cases labelled as cholera were more likely to have been dysentery or typhoid fever. The monthly number of cases, survivors and deaths is shown in table 8.16 but it has to be stressed that figures do not always reflect the total of 740 cases because there are cases in which age, outcome, occupation, date of diagnosis or outcome has not been recorded.

It has to be pointed out that the numbers of those who were cured and those who died do not tally with the total in the first column which itself differs by twenty one from the

219 Daily returns of cholera cases to the committee of the Royal College of Physicians of Edinburgh, November 1848 to November 1849.
A total of 740 cases. This is explained by the fact that eleven cases were listed but no outcome was given and twenty one cases did not have a date of onset or outcome recorded. The final column gives the cases occurring each month as a percentage of the total.

<table>
<thead>
<tr>
<th>Month</th>
<th>Cases</th>
<th>Cured</th>
<th>Died</th>
<th>% Mortality</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>October 1848</td>
<td>86</td>
<td>22</td>
<td>64</td>
<td>74.4</td>
<td>11.6</td>
</tr>
<tr>
<td>November 1848</td>
<td>234</td>
<td>86</td>
<td>148</td>
<td>63.2</td>
<td>31.6</td>
</tr>
<tr>
<td>December 1848</td>
<td>133</td>
<td>68</td>
<td>65</td>
<td>48.8</td>
<td>17.9</td>
</tr>
<tr>
<td>January 1849</td>
<td>42</td>
<td>15</td>
<td>27</td>
<td>64.2</td>
<td>5.6</td>
</tr>
<tr>
<td>February 1849</td>
<td>24</td>
<td>8</td>
<td>16</td>
<td>66.6</td>
<td>3.2</td>
</tr>
<tr>
<td>March 1849</td>
<td>13</td>
<td>5</td>
<td>8</td>
<td>61.5</td>
<td>1.7</td>
</tr>
<tr>
<td>April 1849</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>100.0</td>
<td>.27</td>
</tr>
<tr>
<td>May 1849</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>June 1849</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>July 1849</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>40.0</td>
<td>0.67</td>
</tr>
<tr>
<td>August 1849</td>
<td>42</td>
<td>15</td>
<td>27</td>
<td>64.2</td>
<td>5.6</td>
</tr>
<tr>
<td>September 1849</td>
<td>100</td>
<td>37</td>
<td>63</td>
<td>63.0</td>
<td>13.5</td>
</tr>
<tr>
<td>October 1849</td>
<td>36</td>
<td>14</td>
<td>22</td>
<td>61.1</td>
<td>4.8</td>
</tr>
<tr>
<td>November 1849</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>December 1849</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>50.0</td>
<td>.27</td>
</tr>
<tr>
<td>Total</td>
<td>719</td>
<td>274</td>
<td>445</td>
<td>64.2</td>
<td></td>
</tr>
</tbody>
</table>

Table 8.16. Cholera Returns, Royal College of Physicians of Edinburgh 1848-49.

The mortality was 64.2 per cent which compares with the 50 per cent mortality in other European outbreaks quoted by Pollitzer who says that mortality rates as high as 60 per cent were common and that survival is worst in young children and the elderly. The age distribution and the percentage survival in each age cohort in the Edinburgh epidemic of 1848–49 is shown in table 8.17 and reveals that the mortality in those under
nine years was 67 per cent with the worst survival rates among the over 60s where only 13 per cent survived, conclusions similar to those of Pollitzer. The greatest number attacked was in the age group twenty to forty nine, a statistic that was confirmed in the report of the General Board of Health. The same document recorded that in Glasgow of 2322 attacked, 1058 died, a percentage mortality of 45.5 per cent.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number of cases</th>
<th>% Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–9</td>
<td>67</td>
<td>32.84</td>
</tr>
<tr>
<td>10–19</td>
<td>91</td>
<td>62.64</td>
</tr>
<tr>
<td>20–29</td>
<td>150</td>
<td>41.25</td>
</tr>
<tr>
<td>30–39</td>
<td>168</td>
<td>36.31</td>
</tr>
<tr>
<td>40–49</td>
<td>140</td>
<td>28.57</td>
</tr>
<tr>
<td>50–59</td>
<td>55</td>
<td>30.91</td>
</tr>
<tr>
<td>60–69</td>
<td>37</td>
<td>13.51</td>
</tr>
<tr>
<td>70–79</td>
<td>5</td>
<td>0.00</td>
</tr>
<tr>
<td>80+</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>Age not stated</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>726</td>
<td>36.68</td>
</tr>
</tbody>
</table>

Table 8.17. Age distribution and percentage survival of cholera patients, cholera Returns, Royal College of Physicians of Edinburgh 1848–49. Note: 14 patients did not have their age recorded.

The geographical distribution of cases in Edinburgh is overwhelmingly centred in the Old Town with less than 5 per cent from the New Town although there may have been other episodes in this part of the city where patients were cared for at home and their names not passed on to the cholera board or the police surgeon. Some practitioners may have decided not to record their cholera patients to prevent unwelcome visits from


officials of the Board of Health as happened to Dr John Myrtle’s patient who lived in Howe Street in the New Town.\textsuperscript{222}

The epidemic hit the poor hardest, with 261 patients (35 per cent) being labourers, servants or their families, mostly living in the Old Town where the Canongate, Cowgate, High Street and Grassmarket accounted for at least 490 (66 per cent) of those who contracted cholera. This figure is likely to be an underestimate since patients whose address was simply recorded as the House of Refuge, Quarantine Hospital or Cholera Hospital have not been included.

\begin{table}
\centering
\begin{tabular}{|l|c|}
\hline
Address & Number of cases \\
\hline
Burt's Close & 20 \\
Blackfriars' Wynd & 17 \\
21 College Wynd & 16 \\
Whitehorse Close, Cowgate & 15 \\
St Mary's Wynd & 15 \\
Toddryck’s Wynd & 15 \\
Covenant Close & 13 \\
Old Fishmarket Close & 13 \\
Middle Meal Market Stair & 12 \\
Havelaw’s Close & 8 \\
Bull’s Close, Cowgate & 8 \\
Aird’s Close, Grassmarket & 7 \\
Baillie Fyffe’s Close & 7 \\
Havelaw’s Close & 6 \\
\hline
Total & 172 \\
\hline
\end{tabular}
\caption{Locations with the highest number of cholera cases.\textsuperscript{223}}
\end{table}

This concentration of cholera cases (172) in a relatively small area (table 8.18 lists the

\textsuperscript{222} Edinburgh City Archive, Minutes of the Cleaning Committee, 1844–49, 19 January 1849, pp.260–263. This was the first time that the Committee was described as the Sanitary Committee.

\textsuperscript{223} Royal College of Physicians: Cholera Returns.
addresses in the Old Town with the highest number of cases) could indicate that the outbreak was caused by contamination of a public water source as occurred in the Broad Street water pump case investigated by Dr John Snow.\textsuperscript{224} Snow recorded 522 cases in ten days whereas by the end of December in Edinburgh there had been 453 cases. These statistics and the way in which the Old Town was supplied by water piped to wells suggest that the Old Town outbreak may not have been water borne. It is more likely to have been spread by contact, by hands soiled with infected dejecta, by contaminated food, by water utensils or through fomites, for example bed linen or clothing. This possibility is supported by the predominance of females affected, (60 per cent), a figure that can only be explained by direct or indirect contact spread rather than by water.

There is a difference of opinion amongst authorities regarding the frequency of contact spread, some considering it extremely rare, pointing out that ‘the disease does not usually show a tendency to familial spread, infection in one inhabitant as a rule remaining confined to that person.’ Pollitzer says ‘that these marked differences depend upon living conditions and that contact infection may become rampant in premises where people live crowded together under particularly insanitary conditions ...’. The role of bed linen and clothing in the transmission of the disease was recognised by Koch; among the 306 patients in Edinburgh listed as having had certain contact with cholera, were twenty-eight who had nursed cholera patients and nineteen women who had developed cholera after washing the bed linen or clothing of cholera sufferers.\textsuperscript{225} ‘The tendency to familial spread’ was not a rare phenomenon in the Edinburgh outbreak; table 8.19 lists eight families with three or more members who contracted cholera. It is accepted that these family groups may have contracted the disease by drinking contaminated water or eating food carrying the vibrios; on the other hand Pollitzer believes that contact infection is more likely where there is overcrowding and poor sanitation. In addition to the family groups in table 8.19 there were forty-three examples of two family members developing cholera; if these are added to the number of women

\textsuperscript{225} Pollitzer, \textit{Cholera}, pp.846 & 863.
who had nursed cholera victims and washed their linen, a total of 157 victims is obtained, approximately 21 per cent of the total recorded in the college returns, confirming the frequency of spread by contact, either direct or indirect, or by fomites.

<table>
<thead>
<tr>
<th>Family Groups</th>
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<tbody>
<tr>
<td>5 members of the Swan family</td>
</tr>
<tr>
<td>Father and 2 children</td>
</tr>
<tr>
<td>Mother and 2 children</td>
</tr>
<tr>
<td>Husband, wife and husband’s mother</td>
</tr>
<tr>
<td>19 year old man and his father and mother</td>
</tr>
<tr>
<td>24 year old man and his wife and mother</td>
</tr>
<tr>
<td>8 year old girl and her parents</td>
</tr>
<tr>
<td>3 year boy and his father and aunt</td>
</tr>
</tbody>
</table>

Table 8.19. Family groups with three or more cases of cholera.

The high mortality in this epidemic is plain, almost 20 per cent higher than in Glasgow but roughly similar to rates quoted by Pollitzer. A further unusual feature is the excess of females, 448, as against 292 males; in England the opposite was the case. The percentage mortality amongst females was 55 per cent, smaller than the overall mortality of 64 per cent. The higher morbidity in women is the opposite of the usual experience and supports spread by contact and fomites; had the transmission been mainly water borne the sex incidence would have been the same.\textsuperscript{226} Women were the carers in the community, at greater risk of infection as they nursed the sick and in doing so may have ingested a high dose of cholera vibrios with an appreciably greater chance of developing a more severe infection.\textsuperscript{227} Christie suggests that poor nutrition with a consequent decrease in stomach acid allows more vibrios to pass through into the small intestine and that poorly nourished women may be less able to withstand the physiologic shock of extreme dehydration and electrolyte loss from diarrhoea and vomiting. It is intended to

\textsuperscript{226} Christie, \textit{Infectious Diseases}, p.131.
examine these and other issues in future work on the cholera returns in more depth than is possible in this thesis.

This examination of the fever epidemics, the lesser epidemics and the cholera epidemic of 1848–49, using several sources, has confirmed the severity of these, revealing the substantial morbidity and excess mortality. Study of the epidemics of fever show that these were mainly caused by typhus and relapsing fever with typhoid fever comparatively rare — as Christison commented ‘this is a rare form of fever in Edinburgh, scarcely belonging to its epidemics at all.’\footnote{Christison, ‘On the Changes in the Constitution of Fevers’, p.595.} A large part of the chapter has been devoted to the cholera epidemic which, although relatively small in terms of morbidity and mortality compared to the great fever outbreaks, had a disproportionate impact. It was a condition which terrified the populace and as a result encouraged the municipal authorities and the city’s innately conservative medical profession to work together for the first time in an organised way. This change of attitude in a city whose self image was non industrial and that thought that it was spared many of the problems associated with the manufacturing centres was remarkable. Edinburgh and in particular the inhabitants of its slums, suffered greatly from communicable disease during the 1840s. What is remarkable is that despite this, there was a continued influx of people hoping to find some form of employment or merely seeking ‘the vitality that made the old slums bearable.’\footnote{I.H.Adams, The Making of Urban Scotland, (Montreal, 1978), p.182.}

Conclusion

The thesis has examined the relationship of nutrition and living conditions to disease, specifically nutritional deficiency disorders and infections, in Edinburgh during the 1840s. The history of nutritional science and mid-nineteenth century nutritional knowledge were reviewed and weighed against present day dietary experience and standards. Primary and secondary sources were studied to ascertain the food eaten by the poor, an exercise that was difficult because of the lack of detail in primary source material. However, an important and hitherto unknown source of dietary information was located in the papers of Sir Robert Christison, professor of materia medica in the University.¹ In a similar manner to Jonathan Pereira, the London physician and nutritional expert whose work was described in chapter one, Christison collected dietaries and tables of nutriments which he had printed, presumably for the benefit of his students and colleagues.² Analysis of four of these dietaries yielded significant information about the composition of the diets and, more importantly, on the amounts of the essential micronutrients, vitamins A and C, provided by these diets.

The impact of nutritional deficiency on a population's susceptibility to infectious disease cannot easily be separated from the effects of social deprivation. The congestion in the Old Town of Edinburgh was explored in chapter six, describing how the overcrowding developed; extracts from contemporary texts depicted the squalor in which the inhabitants of the tenements lived. The history of Edinburgh's water supply was reviewed showing how mismanagement of this vital resource led to a chronic shortage of water in the Old Town, of crucial importance if personal and public cleanliness was to be maintained.

¹ Edinburgh University Special Collections, Christison Papers, Tables of Nutriments in Various Dietaries, (Edinburgh, 1854).
² J.Pereira, A Treatise on Food and Diet, (London, 1843).
Prior to a review of epidemic disease, the absence of detailed statistical information on Edinburgh's mortality was discussed, contrasting the mortality data recorded not only in those parts of Britain required by law to register deaths but also in areas not subject to official death recording, for example the cities of Aberdeen and Glasgow. The significance of this deficiency in morbidity and mortality data was emphasised in relation to public health matters in the city, highlighting the apparent indifference of the city administration and the other bodies responsible for control of epidemic disease to statistical evidence and the disregard for this new science by the Edinburgh medical Royal Colleges and the university medical faculty.

The concluding chapters described the epidemics of typhus, relapsing fever, typhoid fever and cholera, touching briefly on the less dramatic epidemics of measles, scarlet fever, smallpox and whooping cough which broke out at regular intervals throughout the 1840s. Finally, an important source of epidemiological, social and medical information was found in the volumes of cholera daily returns maintained by the Royal College of Physicians of Edinburgh in 1848 and 1849. A database has been created from this detailed record and is presented in Appendix 1.3

This study of the nutrition and the living conditions of the poor in the Old Town of Edinburgh in the 1840s has revealed nutritional deficiencies and environmental shortcomings which contributed to the frequency and severity of the epidemics of the 1840s. The research has also shown that there were failings in the response of the medical profession to the repeated crises and this will be addressed briefly before focussing on the crucial aspects of nutrition and living conditions.

The medical profession's indifference to sanitary improvement was part of a more widespread, temporary deterioration in Edinburgh medical standards of which one aspect was the failure to differentiate between typhus, relapsing fever and typhoid until

3 Royal College of Physicians of Edinburgh, Cholera Returns, volumes 1 and 2, 1848-49.
much later than their colleagues in Europe and other parts of Britain. This is surprising
in a city with a renowned medical school and two Royal Colleges, but can perhaps be
explained by a lack of enthusiasm for the new speciality of pathology. The first
Edinburgh chair of pathology was founded in 1831 at the instigation of John Thomson
(1765–1846) who was himself the first incumbent of the chair, succeeded in 1842 by
William Henderson to whom reference has already been made.\(^4\) Jacyna implies that
Thomson was driven more by a desire for personal advancement than by a conviction of
the importance of pathology and that the arrival of a Whig government in 1830 had
‘revived his prospects of preferment’.\(^5\) The survival of the chair was in doubt when
William Henderson, Thomson’s successor, took up homeopathy and when moves to
have him dismissed failed, the medical faculty attempted to remove the teaching of
pathology from the undergraduate curriculum. Personalities and prejudices were
evidently more important than advances in medical science. It was during this period
also that the infirmary lost two young physicians, John Rose Cormack and John Reid,
the former resigning when his request to give clinical lectures to medical students was
refused and the latter leaving to become professor of anatomy at St Andrews. Cormack’s
monograph on epidemic fever, described in chapter seven, is a comprehensive review of
all aspects of the epidemic of 1843; Reid published a work on fever in 1840 and in 1839
was responsible for encouraging the Royal Infirmary to adopt the London Statistical
Society’s method of gathering statistical information.\(^6\) The loss of these men implies that
careers in Edinburgh were unattractive because the prospects of advancement were poor
in a medical school with a declining reputation where the loss of student fees reduced
the income of the leading physicians and may have persuaded them to dispense with
younger ambitious colleagues. There were many factors responsible for the decline. Sir

\(^6\) J.R.Cormack, *Natural History, Pathology, and Treatment of the Epidemic Fever prevailing in Edinburgh
and other Towns*, (London,1843); J.Reid, ‘On the Statistics and Pathology of the Continued Fever of
Edinburgh’, *Edinburgh Medical and Surgical Journal*, 51, (1840); Edinburgh University Special
Collections, Lothian Health Board Archive, Minutes of the Royal Infirmary of Edinburgh, 12, August
1839, p.221.
Alexander Grant (1826–1884), a former Principal, implies in his history of the university that at this time the quality of appointments to the medical school was inferior, an assertion that appears justified when the career of Thomas Stewart Traill (1781–1862) is considered. Traill was fifty-one years old in 1832 when he succeeded Christison in the chair of medical jurisprudence, having practised in Liverpool from the time of his Edinburgh graduation in 1802. Medical jurisprudence in the nineteenth century included the principles of medical police and public health but there is no evidence that Traill showed much interest in these subjects, unlike his counterpart, Robert Cowan, in Glasgow. It is difficult to establish the grounds for Traill’s appointment when it is recalled that his predecessor, Christison, was appointed at the age of twenty-five. Age alone may not have been the determining factor and it has been suggested that political pressure and patronage were important factors in appointments to university chairs. In Edinburgh, the Town Council was the university’s patron, controlling most academic appointments. Until the Reform Act of 1832 Town Councils elected the members of parliament for Scottish cities, an arrangement in which the opportunities for corruption presented by this alliance of local, national and university politics were considerable and in which the ‘Tory guardians of this system were both ruthless in the defence of their privileges and shameless in exploiting its potentials...’ The Council made sure that appointments to university chairs went to supporters of the Tories and it is worthy of note that the winner of the contest for the chair of medical jurisprudence in 1822 was Robert Christison, described in the Dictionary of National Biography as ‘a liberal in religion but a tory in politics.’

7 Comrie, History of Scottish Medicine, p.626; Cowan who was appointed to the post in 1839, five years after graduation, was a founder member of the two Glasgow based Statistical Societies and the author of Statistics of Fever and Smallpox in Glasgow, (Glasgow, 1837) and ‘Vital Statistics of Glasgow’, Journal of the Statistical Society, 3, (1840).
Physicians did not see the maintenance of health and the prevention of disease as matters of public concern: 'to practice medicine was not to make health policy: medicine was a private profession'.\textsuperscript{11} Physicians like William Alison and James Stark who did venture into the realm of public health were involved with specific issues: Alison concentrated on his campaign for a review of the Poor Law and James Stark, the city's statistician, who might have been expected to put forward arguments for change, based on statistical evidence, instead reassured the Council and the public, blaming the immigrant Irish for the increase in fever. In his report for 1848 he wrote, 'we at once see how much has been effected by improvements in the dwellings, drainage, and modes of living of the inhabitants ... since the advent of the low Irish and their increase among us, typhus fever and other epidemics are on the increase ...'. His report for 1846 included the following conclusion: 'these facts, then, demonstrate that Edinburgh is one of the healthiest, if not the very healthiest town of Great Britain,' a conclusion based on extremely doubtful premises.\textsuperscript{12} These professional limitations were important but are not the most significant findings of this investigation. There have been revealed in this thesis crucial and particular factors responsible for the spread and the severity of epidemic disease in Edinburgh during the 1840s, namely the shortage of water in the Old Town and the presence of specific nutritional deficiency. It is the latter that is of critical importance in the relationship between food and epidemic disease but nutrition and water were not the only factors implicated in the development and spread of infectious disease; the fever epidemics in the ghetto of the Old Town were caused by social breakdown, similar to that seen amongst refugees in time of famine when lack of water, overcrowding, absent sanitation and collapse of personal and communal hygiene brought disease in their wake.\textsuperscript{13}

One of the major causes of the congestion and squalor in the Old Town was the loss of housing during ‘the wholesale demolition of dwellings’ sanctioned by the Improvement Act of 1827.14 These urban clearances were carried out in the name of improvement, both architectural and commercial, and can be seen in the same light as the equivalent rural, agricultural and commercial changes. ‘The rationalist, progressive milieu which set the framework for so much energetic re-casting of the world…’ is how Richards has described Enlightenment Edinburgh and it was the perceived need for the city to expand to encourage commercial development that was the driving force behind these changes.15 The removal of thousands of apparently unproductive and disease ridden humans was carried through in the name of progress and seemingly without thought as to the consequences, not only for the newly homeless but also for the increased risk of epidemic disease. It is strange that the fear of disease, which Allan argues was a strong motive for slum improvement, was not a factor in Edinburgh; the clearances that did take place merely exacerbated the situation and ‘the affront to civic dignity’ that he suggests was an equally important force for improvement, although undoubtedly present, apparently did not influence the authorities.16

The Old Town into which the displaced flocked suffered from lack of water and the absence of sanitation, the two inextricably linked for without copious supplies of water sewers cannot function. The dissemination of infection was encouraged by overcrowding and the shortage of water meant that it was almost impossible to maintain any degree of private or public hygiene. Ferguson suggests that ‘Scottish reformers underestimated the importance of strictly hygienic considerations’, concentrating their efforts on the poor law and its shortcomings.17 It is true that, with the notable exception

14 Chapter six, p.130.
15 This concept of urban clearance was stimulated by E.Richards, ‘Patrick Sellar and his World’, Transactions of the Gaelic Society of Inverness, 61, (1998–2000), p.166. Sellar, one of the architects of clearance in Sutherland, was a student in Edinburgh and Richards suggests was influenced by the Enlightenment theories of Adam Smith and Dugald Stewart.
of William Alison, Edinburgh physicians contributed little to the sanitary debate during the 1840s — and Alison’s focus was on the poor law. Doctors did offer opinions at the time of the foul burn controversy described in chapter seven, generally at the request of the leading protagonists of both sides, but thereafter they appeared to lose interest, perhaps resigned to or overwhelmed by the frightful conditions in their midst, unable or unwilling to act until the implementation of what came to be known as the *Nuisances Removal Act* of 1847, renewed and improved in 1848 and again in 1849.\(^{18}\)

After the establishment of the Scottish Poor Law Board of Supervision in 1845 the administration of poor relief remained parish based but the church was no longer in control. In chapter seven the clash of responsibilities between the Parochial Boards and the Police Commissioners, particularly in areas concerning cleaning and sanitation, was explained. There were long–standing administrative inadequacies in the city, the result of a dual structure in which the Town Council and the Police Commissioners had separate responsibilities; both were often in financial difficulties but, important as these were, it was the lack of water that prevented proper cleaning of the wynds, closes and streets of the town — in October 1842 the supply was so poor that the Cleaning Committee discussed procuring water from other sources than the water company.\(^{19}\)

Water shortages undoubtedly affected street cleaning but more importantly it made personal cleanliness difficult and this was the single most important factor in the spread of typhus, relapsing fever and the diseases spread by the faecal–oral route. Razzell argues that in England ‘*it was an improvement in personal hygiene rather than a change in public health that was responsible for the reduction in mortality between 1801 and 1841.*’ [original italics]. He stresses the importance of water, soap and clean clothing in preventing infestation with lice and maintains that among the working classes in

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\(^{18}\) *An Act for the more speedy Removal of certain Nuisances and to enable the Privy Council to make Regulations for the Prevention of Contagion and Epidemic Disease, 9 and 10 Vict., cap. 96, 1847; the Act was renewed in 1848 as 11 and 12 Vict., cap.123, 1848; the Act was amended as an Act to amend the Nuisance Removal and Diseases Prevention Act, 1848, 12 and 13 Vict., cap.111, 1849.*

\(^{19}\) *Edinburgh City Archive, Minutes of the Cleaning Committee, 1844–49, 12 October 1842.*
England the change to a measure of cleanliness had taken place before 1841. 20 It is plain that in Edinburgh this did not happen; there was insufficient water, no public baths and no facilities for the washing of clothes. In the tenements of the Old Town infestation with lice was common and the spread of typhus and relapsing fever the result. Horsley Gantt in a review of the typhus epidemic during the Russian famine of 1932–33 argues that undernutrition, overcrowding, and the lack of soap and hot water for bathing were important contributory factors in the mortality crisis.21 It was the overcrowding with poor personal cleanliness in filthy surroundings that triggered the epidemics of typhus and relapsing fever but acute food shortages were critical: during the 1840s the nutritional state of the poor of Edinburgh was precarious at best and, when work was scarce because of seasonal downturn or commercial recession, many of the working class were malnourished.

In this study the analysis of Christison’s dietaries has proved that certain institutional diets were seriously deficient in vitamins A and C and that his own convalescent diet lacked sufficient vitamin A. Reference was made earlier to work in which estimates of the nutritional status of historical populations were made by measuring the likely number of calories available to a given population group, calculated from wage levels and the price of common foods.22 Other studies have employed anthropometric techniques to estimate nutritional status but none of these have measured specific nutrients in historical diets; for example, the amounts of proteins, vitamins and essential fatty acids.23 The analysis recorded in chapter three has provided hard evidence as to the levels of certain essential dietary factors, revealing deficiencies in vitamin A and vitamin C.

22 Introduction, p.10.
23 Chapter 2, pp.56–58.
The scurvy which appeared in the city at the time of the potato blight is likely to have been a temporary phenomenon, becoming less common in all but the poorest areas when the potato again became available. There is plenty of evidence for scurvy in the 1840s but no records of disease which might indicate widespread vitamin A deficiency, despite the proof of insufficient amounts in the institutional diets analysed. A solitary case of night blindness (almost certainly due to lack of vitamin A) in a twenty-five year old man was described in 1844 by Dr Robert Hamilton; that it was thought worthy of publication attests to its apparent rarity. Preformed vitamin A is found exclusively in animal products such as liver, fish, dairy products and eggs, of which milk was the most readily available to the poor. How much vitamin A was present in the milk sold in cities is uncertain — Drummond and Wilbraham argued that the quality of milk in towns was unbelievably bad, with watering and fat removal very common until well after the middle of the century, practices which reduced the amount of vitamin A. There are other sources of the vitamin: for example, carotenoids from fruit and green and yellow vegetables can be converted into vitamin A and it is known that in the diets of developing countries these often contribute more than 80 per cent of the total. The green outer leaves of vegetables like cabbage and lettuce are good sources of carotene as are yellow vegetables such as carrots and turnips, the latter being a cheap and readily available root vegetable in Scotland. Despite these possible sources and the clear evidence that vegetables were available in English cities, Drummond considered that vitamin A deficiency ‘must have been common and not recognised’ in Britain whereas ‘...across the channel the situation was quite different.’ He quoted John Hughes Bennett (1812–1875), an Edinburgh graduate who for a time practised in Paris where he described xerophthalmia, caused by vitamin A deficiency, in a paper published in

24 R. Hamilton, ‘Case of Imperfect Vision from Irregular Refraction with Night-Blindness, etc.’, Monthly Journal of Medical Science, 12, (1847), pp.891–893.
26 Davidson, Passmore, Brock and Truswell, Human Nutrition and Dietetics, p.146.
27 Chapter 3, pp.95 & 96.
1841. Bennett returned to Edinburgh in 1842 but there is no record that he diagnosed the condition amongst the poor in Edinburgh; it seems unlikely that Edinburgh's poor ate more animal food or fruit and vegetables than their counterparts in Paris. The analysis proves that the diet commonly eaten not only by the poorest in Edinburgh but also on occasion by the well off was deficient in two essential vitamins. The improvement in the level of mortality evident by the 1820s was not maintained — as Szreter says 'overall mortality ceased to fall for almost half a century throughout the central decades of the mid-nineteenth century.' The part played by specific dietary deficiency in the increase in mortality levels, particularly from infectious disease, must have been substantial. If the danger to adults of specific nutrient deficiency was considerable the risk to infants was appreciably greater 'because they were so often reared on diets that contained amounts of the vitamins far below those required for growth and health.' The increased mortality of children in underdeveloped countries from measles is now known to be associated with vitamin A deficiency and is likely to have been the cause of the high mortality from this infection in Edinburgh during the middle of the nineteenth century.

This examination of Edinburgh's mid nineteenth century society has confirmed the theory that living conditions in the tenements of the Old Town were comparable to those found in present day refugee camps. There was gross overcrowding in conditions of squalor which was the result of an acute shortage of water and non-existent sanitation. The epidemics of infectious disease which visited the city at frequent intervals throughout the 1840s were the consequence of these environmental conditions.

and of the impaired nutritional status of many of the inhabitants. This research has revealed that many people in the city were living on a diet lacking specific essential nutrients, namely vitamins A and C. The deficiency of vitamin C was a temporary phenomenon triggered by the loss of a dietary staple because of the potato blight. The lack of vitamin A was not the result of crop failure but was caused by a chronic shortage of food containing adequate levels of vitamin A or its precursors. In Edinburgh in the 1840s it was increased exposure to infection in a population existing on a diet deficient in specific essential nutrients that was responsible for the increase in infectious mortality.
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Theses
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<th>Occupation</th>
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<td>M</td>
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<td>13</td>
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<td>20 h</td>
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<td>27</td>
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<td>44 h</td>
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<td>46</td>
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<td>D</td>
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<td>02-Aug-49 20</td>
<td>20 h</td>
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<td>D</td>
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<td>washed a ch patients clothes a few days</td>
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<td>03-Aug-49 18</td>
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<td>24-Jul-49 3</td>
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<td>51</td>
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<td>17-Aug-49 17</td>
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<td>23-Aug-49 20</td>
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<td>17-Aug-49 20</td>
<td>20 h</td>
<td>T.ext/St</td>
<td>D</td>
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<td>McKay</td>
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<td>F</td>
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<td>64</td>
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<td>28-Dec-48 2</td>
<td>2 d</td>
<td>L+O/S/U/D</td>
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<td>D/C</td>
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<td>Roden</td>
<td>M</td>
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<td>52</td>
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<td>15 d</td>
<td>A/ST/D</td>
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<td>9 h</td>
<td>CA/2 drops of laudanum every ten minutes/</td>
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<td>30 h</td>
<td>CA/2 drops of laudanum every ten minutes/</td>
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<td>40</td>
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<td>24-Jul-49</td>
<td>31 h</td>
<td>CO/T/bichlor of mercury</td>
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<td>223 h</td>
<td>CO/T/Gin/mustard emetic</td>
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<td>51 Blackfriar's Wynd</td>
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<td>F labourer</td>
<td>30-Oct-48</td>
<td>20 h</td>
<td>CH/CA/O/Cardamom CA/O/B</td>
<td>D</td>
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<td>M</td>
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<td>9 h</td>
<td>Salines/SP/Camphor +Nitr</td>
<td>D</td>
<td>Y</td>
<td>3 others ill in house; many PMs refer to</td>
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<td>85</td>
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<td>26</td>
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<td>17 h</td>
<td>Peyes</td>
<td>Y</td>
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<td>29-Oct-48</td>
<td>27 h</td>
<td>B/Petroleum/IV</td>
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<td>Philip</td>
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<td>19 h</td>
<td>W</td>
<td>D</td>
<td>Y</td>
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<td>F</td>
<td>43 Abbey Hill</td>
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<td>22 h</td>
<td>D/W/O+ Capsicum</td>
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<td>89</td>
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<td>20</td>
<td>H coachman</td>
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<td>1.5 h</td>
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<td>17 h</td>
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<td>92</td>
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<td>25</td>
<td>H Clerk</td>
<td>31-Oct-48</td>
<td>36 h</td>
<td>D/CA+O/Ch inhalation/Wh</td>
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<td>01-Nov-48</td>
<td>31 h</td>
<td>Wh/V</td>
<td>D</td>
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<td>32 h</td>
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<td>Treatment</td>
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<td>D/Ca-O/Sal</td>
<td>Y</td>
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<td>John ?</td>
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<td>3 d</td>
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<td>3 d</td>
<td>D/Sal/W/Iv</td>
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<td>F</td>
<td>? Purves Land Canongate</td>
<td>18</td>
<td>mill girl</td>
<td>03-Nov-48</td>
<td>15 h</td>
<td>W</td>
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<td>mason</td>
<td>29-Oct-48</td>
<td>6 d</td>
<td>W/CA+O/CH/Sal</td>
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<td>60 h</td>
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<td>sweeps widow</td>
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<td>3 d</td>
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<td>20 h</td>
<td>CH/ST/H</td>
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<td>Ford</td>
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<td>20 h</td>
<td>CA+O/ST/H</td>
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<td>05-Nov-48</td>
<td>14 h</td>
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<td>36 h</td>
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<td>18 Greenside Row</td>
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<td>33 h</td>
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<td>cutter</td>
<td>05-Nov-48</td>
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<td>2 d</td>
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<td>D</td>
<td></td>
<td>ch in house</td>
</tr>
<tr>
<td>203</td>
<td>Alex</td>
<td>Partane</td>
<td>M</td>
<td>Hastie's Close Cowgate</td>
<td>56</td>
<td>coal carrier</td>
<td>17-Nov-48</td>
<td>2 d</td>
<td>W/CA/O</td>
<td>D</td>
<td></td>
<td>ch in close</td>
</tr>
<tr>
<td>204</td>
<td>Stephen</td>
<td>Reilly</td>
<td>M</td>
<td>10 Toddryck's Wynd</td>
<td>22</td>
<td>labourer</td>
<td>20-Nov-48</td>
<td>30 d</td>
<td>CA/O7/SIV</td>
<td>D</td>
<td>Y</td>
<td>ch in stair; iv 4 oz revived him, colour better, pulse also</td>
</tr>
<tr>
<td>205</td>
<td>Jane</td>
<td>Samuel</td>
<td>F</td>
<td>247 Canongate</td>
<td>23</td>
<td>paper mill</td>
<td>19-Nov-48</td>
<td>29 h</td>
<td>CA/O/Ch</td>
<td>D</td>
<td></td>
<td>Recent delivery</td>
</tr>
<tr>
<td>206</td>
<td>Thomas</td>
<td>Williamson</td>
<td>M</td>
<td>Toddryck's Wynd/ Baillie Fyfe's</td>
<td>28</td>
<td>saddler</td>
<td>19-Nov-48</td>
<td>2 d</td>
<td>W/CA/O/Lenema</td>
<td>D</td>
<td></td>
<td>ch in both places</td>
</tr>
<tr>
<td>207</td>
<td>Agnes</td>
<td>Gould</td>
<td>F</td>
<td>13 High Riggs</td>
<td>28</td>
<td>H gardener</td>
<td>04-Nov-48</td>
<td>18 d</td>
<td>CA/O/P/CH/Wi/Wi</td>
<td>C</td>
<td></td>
<td>sister ch; aborted while in H</td>
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<tr>
<td>No.</td>
<td>Name</td>
<td>Surname</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
<td></td>
</tr>
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<td>-----</td>
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<td>---------------</td>
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<td>208</td>
<td>?</td>
<td>Weir</td>
<td>Baillie Hamilton's Close</td>
<td>4</td>
<td>labourer</td>
<td>02-Nov-48</td>
<td>20 d</td>
<td>F, M, Sr all in H</td>
<td>C</td>
<td></td>
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<tr>
<td>209</td>
<td>Francis</td>
<td>Mc Ara</td>
<td>35 Tolbooth Wyd Leith</td>
<td>26</td>
<td>tailor</td>
<td>10-Nov-48</td>
<td>12 d</td>
<td>P/CA/O/</td>
<td>C</td>
<td></td>
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<td>210</td>
<td>John</td>
<td>Featherstone</td>
<td>57 Blackfriar's Wynd</td>
<td>42</td>
<td>labourer</td>
<td>13-Nov-48</td>
<td>9 d</td>
<td>CA/O/</td>
<td>C</td>
<td></td>
<td>ch in house</td>
<td></td>
</tr>
<tr>
<td>211</td>
<td>Margaret</td>
<td>Alexander</td>
<td>2 Rose St</td>
<td>23</td>
<td>servant</td>
<td>13-Nov-48</td>
<td>9 d</td>
<td>Ca/O/Ve</td>
<td>C</td>
<td></td>
<td>no contact with ch</td>
<td></td>
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<tr>
<td>212</td>
<td>John</td>
<td>Pack</td>
<td>Mill Court Lawnmarket</td>
<td>14</td>
<td>bookbinder</td>
<td>17-Nov-48</td>
<td>5 d</td>
<td>CA/O/salines</td>
<td>C</td>
<td></td>
<td>none in close</td>
<td></td>
</tr>
<tr>
<td>213</td>
<td>George</td>
<td>Lamb</td>
<td>Skinners Close High St</td>
<td>35</td>
<td>quarrier</td>
<td>19-Nov-48</td>
<td>3 d</td>
<td>CA/O/Ve</td>
<td>C</td>
<td></td>
<td>none in close</td>
<td></td>
</tr>
<tr>
<td>214</td>
<td>James</td>
<td>McCabe</td>
<td>Forrester's Close, Cowgate</td>
<td>28</td>
<td>labourer</td>
<td>21-Nov-48</td>
<td>1 d</td>
<td>CA/O/</td>
<td>C</td>
<td></td>
<td>ch in close</td>
<td></td>
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<tr>
<td>215</td>
<td>Thomas</td>
<td>Peddie</td>
<td>105 Grassmarket</td>
<td>40</td>
<td>labourer</td>
<td>19-Nov-48</td>
<td>4 d</td>
<td>W/CA/OP/</td>
<td>D</td>
<td></td>
<td>ch in vicinity</td>
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</tr>
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<td>216</td>
<td>Mary</td>
<td>Cove</td>
<td>Plainstone Close Grassmarket</td>
<td>16</td>
<td>F labourer</td>
<td>20-Nov-48</td>
<td>3 d</td>
<td>O/ Mustard emetic</td>
<td>C</td>
<td></td>
<td>ch in close; v doubtful</td>
<td></td>
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<tr>
<td>217</td>
<td>Fanny</td>
<td>Carruthers</td>
<td>Bannermans Close Cowgate</td>
<td>17</td>
<td>servant</td>
<td>20-Nov-48</td>
<td>3 d</td>
<td>W/fo/CM/ Blister</td>
<td>D</td>
<td></td>
<td>master died of ch in</td>
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<td></td>
<td>Case in wynd</td>
<td></td>
<td></td>
<td>house</td>
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<tr>
<td>218</td>
<td>John</td>
<td>Mc Neamey</td>
<td>Blackfriar's Wynd</td>
<td>40</td>
<td>labourer</td>
<td>23-Nov-48</td>
<td>18 h</td>
<td>CH/ Ve/CA/O/</td>
<td>D</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>219</td>
<td>Cath</td>
<td>Stewart</td>
<td>Jail/ Police Office</td>
<td>27</td>
<td>none</td>
<td>06-Nov-48</td>
<td>17 d</td>
<td>VE/O/P/Enema</td>
<td>C</td>
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<tr>
<td>220</td>
<td>Isabella</td>
<td>Munro</td>
<td>Covenant Close</td>
<td>19</td>
<td>prostitute</td>
<td>17-Nov-48</td>
<td>8 d</td>
<td>CA/O/V/e</td>
<td>C</td>
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<tr>
<td>221</td>
<td>Elizabeth</td>
<td>Dunsmore</td>
<td>Hyndfords Close</td>
<td>40</td>
<td>washerwoman</td>
<td>20-Nov-48</td>
<td>5 d</td>
<td>W/O/D/P</td>
<td>D</td>
<td></td>
<td>Y unknown</td>
<td></td>
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<tr>
<td>222</td>
<td>David</td>
<td>Mc Diarmid</td>
<td>Victoria Lodging House Cowgate</td>
<td>45</td>
<td>tailor</td>
<td>14-Nov-48</td>
<td>11 d</td>
<td>CA/O</td>
<td>D</td>
<td></td>
<td>no contact</td>
<td></td>
</tr>
<tr>
<td>223</td>
<td>Jane</td>
<td>Mc Pherson</td>
<td>Borthwick's Close</td>
<td>22</td>
<td>seamstress</td>
<td>08-Nov-48</td>
<td>17 d</td>
<td>none</td>
<td>C</td>
<td></td>
<td>case of TB</td>
<td></td>
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<td>224</td>
<td>Alex</td>
<td>Reid</td>
<td>Baillie Fye's Close</td>
<td>38</td>
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<td>08-Nov-48</td>
<td>11 d</td>
<td>CA/O/salines</td>
<td>C</td>
<td></td>
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<tr>
<td>225</td>
<td>Helen</td>
<td>Murphy</td>
<td>Plainstone Close Grassmarket</td>
<td>20</td>
<td>H labourer</td>
<td>19-Nov-48</td>
<td>4 d</td>
<td>CA/O/VE</td>
<td>C</td>
<td></td>
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<tr>
<td>226</td>
<td>Martha</td>
<td>McGee</td>
<td>24 Old Fish Market Close</td>
<td>18</td>
<td>servant</td>
<td>20-Nov-48</td>
<td>5 d</td>
<td>CH/CA/O/Sal/Ve</td>
<td>D</td>
<td></td>
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<td>227</td>
<td>Thomas</td>
<td>Murphy</td>
<td>36 Cowgatehead</td>
<td>36</td>
<td>labourer</td>
<td>23-Nov-48</td>
<td></td>
<td></td>
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<tr>
<td>228</td>
<td>Ann</td>
<td>Mc Ardle</td>
<td>Curries Close</td>
<td>46</td>
<td>hawker</td>
<td>25-Nov-48</td>
<td>2 d</td>
<td>CA/O/Ch</td>
<td>D</td>
<td></td>
<td>Y ch in house</td>
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</tr>
<tr>
<td>229</td>
<td>Michael</td>
<td>Hawkins</td>
<td>Bull Land 303 Cowgate</td>
<td>80</td>
<td>labourer</td>
<td>26-Nov-48</td>
<td>7 h</td>
<td>CA/OW/VE</td>
<td>D</td>
<td></td>
<td>ch in neighbourhood</td>
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<tr>
<td>230</td>
<td>Alison</td>
<td>Young</td>
<td>Lodging House Pleasance</td>
<td>30</td>
<td>servant</td>
<td>06-Nov-48</td>
<td>21 d</td>
<td>D/CA/O/sal</td>
<td>C</td>
<td></td>
<td>no contact</td>
<td></td>
</tr>
<tr>
<td>231</td>
<td>Anne</td>
<td>Mc Leod</td>
<td>Jollys Land Canongate</td>
<td>38</td>
<td>washerwoman</td>
<td>13-Nov-48</td>
<td>14 d</td>
<td>CA/O/V/E</td>
<td>C</td>
<td></td>
<td>ch in neighbourhood</td>
<td></td>
</tr>
<tr>
<td>232</td>
<td>Margaret</td>
<td>Half Penny</td>
<td>Bulls Close Cowgate</td>
<td>26</td>
<td>H labourer</td>
<td>12-Nov-48</td>
<td>15 d</td>
<td>CA/O/bismuth</td>
<td>C</td>
<td></td>
<td>no contact; Synochara</td>
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<td>233</td>
<td>James</td>
<td>Simpson</td>
<td>45 Pleasance</td>
<td>26</td>
<td>coop</td>
<td>24-Nov-48</td>
<td>3 d</td>
<td>CA/O/salines</td>
<td>C</td>
<td></td>
<td>no contact</td>
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<tr>
<td>234</td>
<td>Jas ?</td>
<td>Boan</td>
<td>Burt's Close Grassmarket</td>
<td>10</td>
<td>F labourer</td>
<td>24-Nov-48</td>
<td>3 d</td>
<td>heat</td>
<td>C</td>
<td></td>
<td>chin stair</td>
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<tr>
<td>235</td>
<td>Martin</td>
<td>Chamley</td>
<td>68? Cameron's Close Grassmarket</td>
<td>11</td>
<td>orphan at Ind School</td>
<td>23-Nov-48</td>
<td>4 d</td>
<td>salines</td>
<td>C</td>
<td></td>
<td>ch in stair</td>
<td></td>
</tr>
<tr>
<td>236</td>
<td>Alex</td>
<td>Martin</td>
<td>155 Rose</td>
<td>40</td>
<td>servant</td>
<td>18-Nov-48</td>
<td>10 d</td>
<td>SVO</td>
<td>D</td>
<td></td>
<td>ch in St seen none; Typhus had urethral stricture also</td>
<td></td>
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<tr>
<td>ID</td>
<td>First Name</td>
<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
</tr>
<tr>
<td>-----</td>
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</tr>
<tr>
<td>237</td>
<td>James</td>
<td>Lyndey</td>
<td>M</td>
<td>Goodells Lodgings Inks Close</td>
<td>32</td>
<td>jeweller</td>
<td>20-Nov-48 7 d</td>
<td>CA/O/D/St/CO</td>
<td></td>
<td>D</td>
<td></td>
<td>in neighbourhood</td>
</tr>
<tr>
<td>238</td>
<td>Thos</td>
<td>Cullen</td>
<td>M</td>
<td>Bulls Land 303 Cowgate</td>
<td>5 F</td>
<td>tailor</td>
<td>27-Nov-48 2 d</td>
<td>CA/Ipecech</td>
<td></td>
<td>D</td>
<td>Y</td>
<td>in house</td>
</tr>
<tr>
<td>239</td>
<td>Cath</td>
<td>McMurdoch</td>
<td>F</td>
<td>Moore's Land Pleasance</td>
<td>50</td>
<td>hawker</td>
<td>28-Nov-48 21 h</td>
<td>W/CA/O</td>
<td></td>
<td>D</td>
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<td>in a ch locality visiting th sick</td>
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<tr>
<td>240</td>
<td>James</td>
<td>Sheriffs</td>
<td>M</td>
<td>Curries Close Grassmarket</td>
<td>45</td>
<td>tailor</td>
<td>28-Nov-48 35 h</td>
<td>CA/O/P/W/Ch/D/Sail nes</td>
<td></td>
<td>D</td>
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<td>241</td>
<td>Phillip</td>
<td>Connoily</td>
<td>M</td>
<td>College Wynd</td>
<td>35</td>
<td>labourer</td>
<td>29-Nov-48 17 h</td>
<td>CA/O/D/W</td>
<td></td>
<td>D</td>
<td>Y</td>
<td>ch in house</td>
</tr>
<tr>
<td>242</td>
<td>Alex</td>
<td>Walker</td>
<td>M</td>
<td>10 Rose St</td>
<td>35</td>
<td>groom</td>
<td>29-Nov-48 41 h</td>
<td>CA/O/St?dSalines</td>
<td></td>
<td>D</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>243</td>
<td>Robert</td>
<td>Johnston</td>
<td>M</td>
<td>87 Cowgate</td>
<td>6</td>
<td>tinkers child</td>
<td>29-Nov-48 2 d</td>
<td>IV</td>
<td></td>
<td>D</td>
<td></td>
<td>unknown</td>
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<tr>
<td>244</td>
<td>Cath</td>
<td>Hart</td>
<td>F</td>
<td>17 St Mary's Wynd</td>
<td>36</td>
<td>?</td>
<td>28-Nov-48 33 d</td>
<td>CA/O/Wi/Oenema</td>
<td></td>
<td>D</td>
<td></td>
<td>not ch</td>
</tr>
<tr>
<td>245</td>
<td>Charlotte</td>
<td>Johnston</td>
<td>F</td>
<td>Morrison's Close 273 Cowgate</td>
<td>23</td>
<td>seamstress</td>
<td>25-Nov-48 5 d</td>
<td>D/IV/S/Heat</td>
<td></td>
<td>D</td>
<td>Y</td>
<td>ch in close; pregnant aborted</td>
</tr>
<tr>
<td>246</td>
<td>Mary</td>
<td>Byrne</td>
<td>F</td>
<td>Cameron Close Grassmarket</td>
<td>45</td>
<td>W labourer</td>
<td>30-Nov-48 28 d</td>
<td>D/IV/S/Heat</td>
<td></td>
<td>D</td>
<td>Y</td>
<td>ch in close IV produced great improvement chin slair</td>
</tr>
<tr>
<td>247</td>
<td>Colin</td>
<td>Fraser</td>
<td>M</td>
<td>Goods Entry College Wynd</td>
<td>44</td>
<td>silver plater</td>
<td>29-Nov-48 33 h</td>
<td>O/D/Friction/IV/CA</td>
<td>D</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>249</td>
<td>Thomas</td>
<td>Clelland</td>
<td>M</td>
<td>Curries Close</td>
<td>50</td>
<td>hawker</td>
<td>02-Dec-48 29 h</td>
<td>D/S/Salines/Heat/Fri ctons</td>
<td>D</td>
<td></td>
<td>ch in close</td>
<td></td>
</tr>
<tr>
<td>250</td>
<td>John</td>
<td>Cothill</td>
<td>M</td>
<td>6 East Rose St Lane</td>
<td>45</td>
<td>groom</td>
<td>30-Nov-48 2 d</td>
<td>CA/O/Ipeceh/O/Enema/Head Shaved and col applied</td>
<td>D</td>
<td>Y</td>
<td>in contact with patient</td>
<td></td>
</tr>
<tr>
<td>251</td>
<td>Agnes</td>
<td>Cilues</td>
<td>F</td>
<td>11 East Richmond Close</td>
<td>47</td>
<td>washenwoman</td>
<td>28-Nov-48 4 d</td>
<td>CA/O/D/SpBl</td>
<td></td>
<td>D</td>
<td></td>
<td>ch in house</td>
</tr>
<tr>
<td>252</td>
<td>Frederick</td>
<td>Ainsworth</td>
<td>M</td>
<td>Broxburn</td>
<td>27</td>
<td>mason</td>
<td>19-Nov-48 14 d</td>
<td>O/CA/Bled/Dovers/Ipeceh and opium enema/W</td>
<td>D</td>
<td></td>
<td>not ch</td>
<td></td>
</tr>
<tr>
<td>253</td>
<td>Jane</td>
<td>Flide</td>
<td>F</td>
<td>Bulls Land 303 Cowgate</td>
<td>30</td>
<td>dressmaker</td>
<td>02-Dec-48 32 h</td>
<td>CA/O/D/S/Iv/Turps Enema</td>
<td>D</td>
<td>Y</td>
<td>ch in house</td>
<td></td>
</tr>
<tr>
<td>254</td>
<td>Norah</td>
<td>Hogg</td>
<td>F</td>
<td></td>
<td>14</td>
<td>30 lately nurse in Ch H</td>
<td>30-Nov-48 4 d</td>
<td>CH inhalation/O/CA/Bled</td>
<td>C</td>
<td></td>
<td>Visited sick in Westport 28/11; Doubtful if ch</td>
<td></td>
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<tr>
<td>255</td>
<td>Margaret</td>
<td>Grant</td>
<td>F</td>
<td>Hastie's Close Cowgate</td>
<td>51</td>
<td>H shoemaker</td>
<td>02-Dec-48 4 d</td>
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<td>CA+ O/P/Sal/OR</td>
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<td>C</td>
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<td>01-Nov-48 36 h</td>
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<td>30 h</td>
<td>O/S/D</td>
<td>D</td>
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<td>36 d</td>
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<td>F</td>
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<td>F mason</td>
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<td>20 h</td>
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<td>30-Nov-48</td>
<td>15 h</td>
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<td>294</td>
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<td>11 d</td>
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<td>06-Dec-48</td>
<td>12 d</td>
<td>O/W/OR/O enemata</td>
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<td>296</td>
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<td>9 d</td>
<td>D/W/T</td>
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<td>19 d</td>
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<td>08-Dec-48</td>
<td>10 d</td>
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<td>11-Dec-48</td>
<td>6 d</td>
<td>CA+O/W</td>
<td>D</td>
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<td>ch in house; contact with ch</td>
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Cholera Returns 1848-49
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<th>ID</th>
<th>First Name</th>
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<th>Treatment</th>
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<th>PM</th>
<th>Contact ch and others</th>
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<td>Quin</td>
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<td>13-Dec-48</td>
<td>34 h</td>
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<td>C</td>
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<td>Graemus</td>
<td>M</td>
<td>21 College Wynd low door</td>
<td>56</td>
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<td>18 h</td>
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<td>Y</td>
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<td>36 h</td>
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<td>Y</td>
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<td>Jamieson</td>
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<td>12 h</td>
<td>Sal/D/Wi</td>
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<td>19 h</td>
<td>D/LA+O enema</td>
<td>D</td>
<td>Y</td>
<td>taking charge of a ch patient in Havelaw's Close ch in neighbourhood</td>
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<td>Watt</td>
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<td>cabinet maker</td>
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<td>F painter</td>
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<td>4 d</td>
<td>CA/Wi</td>
<td>D</td>
<td></td>
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<td>5 d</td>
<td>CA+O/VS/DW/P/Sal/3/Digital-squill and blue pill</td>
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<td>Collins</td>
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<td>O/VS</td>
<td>C</td>
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<td>27 Cowgate? Head</td>
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<td>02-Dec-48</td>
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<td>19-Dec-48</td>
<td>4 d</td>
<td>CO/T enema</td>
<td>D</td>
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<td>Contact ch and others</td>
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<td>W of porter</td>
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<td>26 h</td>
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<td>42</td>
<td>H mason hawker</td>
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<td>9 d</td>
<td>C/VS/O/BM/Mist.Pectoralis</td>
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<td>Connor</td>
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<td>7 d</td>
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<td>C</td>
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<td>D/W/CA/IV/CH/Laud anum/Wi</td>
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<td>Digital Squillandblue pill</td>
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<td>4 d</td>
<td>Sal/CA+O/Win/W/D</td>
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<td>not ch had been drinking all day on day of admission ch in stair</td>
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<td>27 h</td>
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<td>14-Dec-48</td>
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<td>VS/CA/Gin/P/OR/SoI Hydarg/Laudanum</td>
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<td>340</td>
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<td>M</td>
<td>Anchor Close</td>
<td>43</td>
<td>painter</td>
<td>28-Dec-48</td>
<td>28 h</td>
<td>D/W/CA/Laudanum</td>
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<td>341</td>
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<td>McLachlan</td>
<td>M</td>
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<td>Anchor Close</td>
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<td>H clerk</td>
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Cholera Returns 1848–49
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<th>Sex</th>
<th>Address</th>
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<th>Period</th>
<th>Treatment</th>
<th>D/C</th>
<th>PM</th>
<th>Contact ch and others</th>
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<td>31-Dec-48 17 h</td>
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<td>ch in stair attending the sick; VS of 12 ozs patient said she felt better; had been drinking for 8 days ch in stair above; drinking for two weeks ch in house</td>
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<td>30-Dec-48 2 d</td>
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<td></td>
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<td>F</td>
<td>Anchor Close</td>
<td>34</td>
<td>H painter</td>
<td>29-Dec-48 3 d</td>
<td></td>
<td>VS/OR/CH/D/Wi/CA</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>346</td>
<td>Jessie</td>
<td>Craig</td>
<td>F</td>
<td>16 Roxburgh Place</td>
<td>16</td>
<td>servant</td>
<td>20-Dec-48 13 d</td>
<td></td>
<td>VS/Sal/OR/CA/Laudanum/enema/CA/Wi</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>347</td>
<td>Francis</td>
<td>Brady</td>
<td>M</td>
<td>Havelaw's Close</td>
<td>6</td>
<td>F labourer</td>
<td>21-Dec-48 12 d</td>
<td></td>
<td>CA/Wi/CA/Or/Laudanum/Sol Hydranum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>348</td>
<td>Charlotte</td>
<td>McKay</td>
<td>F</td>
<td>26 Fleshmarket Close High St</td>
<td>28</td>
<td>seamstress</td>
<td>21-Dec-48 13 d</td>
<td></td>
<td>OR/D/VS/Sal/CA/Laudanum/enema/CA/Wi</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>349</td>
<td>Francis</td>
<td>Meehan</td>
<td>M</td>
<td>36 Cowgate</td>
<td>7</td>
<td>F labourer</td>
<td>12-Dec-48 23 d</td>
<td></td>
<td>OR/CA/Bismuth/</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>350</td>
<td>Agnes</td>
<td>Martin</td>
<td>F</td>
<td>Baillie Fyle's Close</td>
<td>20</td>
<td>servant</td>
<td>21-Dec-48 14 d</td>
<td></td>
<td>VS/CA/Or/Laudanum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>351</td>
<td>Rebecca</td>
<td>Robby</td>
<td>F</td>
<td>60 Thistle St</td>
<td>26</td>
<td>servant</td>
<td>03-Jan-49 23 h</td>
<td></td>
<td>CA/Gin/Capsicum/Camphor</td>
<td>Y</td>
<td></td>
<td>ch in same land</td>
</tr>
<tr>
<td>352</td>
<td>Helen</td>
<td>McNeill</td>
<td>F</td>
<td>Milne's Court</td>
<td>50</td>
<td>H tailor</td>
<td>24 h</td>
<td></td>
<td>D/Gin/CA/Camphor/Capsicum</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>353</td>
<td>John</td>
<td>Curtis</td>
<td>M</td>
<td>Quarantine House Lothian Rd prev in Havelaw's Close</td>
<td>8</td>
<td>F tailor</td>
<td>01-Jan-49 4 d</td>
<td></td>
<td>OR/Wi/T</td>
<td>Y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>354</td>
<td>Rose</td>
<td>Kibbins</td>
<td>F</td>
<td>Laurie's Lodgings West Port</td>
<td>35</td>
<td>fisherwoman</td>
<td>02-Jan-49 2 d</td>
<td></td>
<td>CH/OR/CA/Capsicum/Camphor</td>
<td>Y</td>
<td></td>
<td>ch in Havelaw's Close F died of ch&amp; sister and M are patients no ch in house</td>
</tr>
<tr>
<td>355</td>
<td>Isabella</td>
<td>Brown</td>
<td>F</td>
<td>Watts Close Fisherrow</td>
<td>35</td>
<td>fisherwoman</td>
<td>03-Jan-49 35 h</td>
<td></td>
<td>OR/CH/Cap/Camphor</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>356</td>
<td>John</td>
<td>Stewart</td>
<td>M</td>
<td>90 Grassmarket</td>
<td>80</td>
<td>gunmaker</td>
<td>04-Jan-49 12 h</td>
<td></td>
<td>Gin/ T friction</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>357</td>
<td>James</td>
<td>Affleck</td>
<td>M</td>
<td>Havelaw's Close</td>
<td>26</td>
<td>tailor</td>
<td>03-Jan-49 22 h</td>
<td></td>
<td>Gin/ T friction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>358</td>
<td>George</td>
<td>Smith</td>
<td>M</td>
<td>8 Nicolson</td>
<td>37</td>
<td>printer</td>
<td>04-Jan-49 3 d</td>
<td></td>
<td>VS/D/ST/CAO/CA/Laudanum</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>359</td>
<td>Helen</td>
<td>? Mulhry</td>
<td>F</td>
<td>272 Cowgate</td>
<td>40</td>
<td>washerwoman</td>
<td>28-Dec-48 11 d</td>
<td></td>
<td>OR/CA/</td>
<td>C</td>
<td></td>
<td>no ch in stair; doubtful case ch died of ch; ch in house</td>
</tr>
<tr>
<td>360</td>
<td>Anne</td>
<td>Curtis</td>
<td>F</td>
<td>Havelaw's Close</td>
<td>14</td>
<td>F tailor</td>
<td>31-Dec-48 18 d</td>
<td></td>
<td>VS/DA/HC/Laudanum</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>361</td>
<td>Robert</td>
<td>Curtis</td>
<td>M</td>
<td>Havelaw's Close</td>
<td>6</td>
<td>F tailor</td>
<td>31-Dec-48 18 d</td>
<td></td>
<td>Enema of L+O/Chalk mixture</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>362</td>
<td>Thomas</td>
<td>McCabe</td>
<td>M</td>
<td>2 Horse Wynd</td>
<td>19</td>
<td>labourer</td>
<td>29-Dec-48 18 d</td>
<td></td>
<td>D/OR/BL</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID</td>
<td>First Name</td>
<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
</tr>
<tr>
<td>-----</td>
<td>------------</td>
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<td>-----</td>
<td>-----------------</td>
<td>-----</td>
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<td>-----------------------------------------------</td>
<td>-----</td>
<td>----</td>
<td>------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>363</td>
<td>Mary</td>
<td>Johnston</td>
<td>F</td>
<td>32 Pleasance</td>
<td>8 M</td>
<td>washerwoman</td>
<td>05-Dec-49</td>
<td>5 d</td>
<td>CA/Wi/Digitalis</td>
<td>D</td>
<td>Y</td>
<td>playing with the children of a person who died of ch</td>
</tr>
<tr>
<td>364</td>
<td>Mary</td>
<td>Ross</td>
<td>F</td>
<td>Cants Close</td>
<td>30 H</td>
<td>cutter</td>
<td>06-Dec-48</td>
<td>10 d</td>
<td>VS/CA/Or/Laudanum</td>
<td>C</td>
<td></td>
<td>ch in close; dismissed from ch hospital 18 Dec</td>
</tr>
<tr>
<td>365</td>
<td>Andrew</td>
<td>McGouf</td>
<td>M</td>
<td>Hastie's Close Cowgate</td>
<td>24</td>
<td>hawker</td>
<td>05-Dec-49</td>
<td>5 d</td>
<td>OR</td>
<td>C</td>
<td></td>
<td>ch in house and mother in law died of it</td>
</tr>
<tr>
<td>366</td>
<td>Sarah</td>
<td>O Donnell</td>
<td>F</td>
<td>Fleshmarket</td>
<td>67 H</td>
<td>labourer</td>
<td>09-Jan-49</td>
<td>35 h</td>
<td>CA/ST</td>
<td>D</td>
<td>Y</td>
<td>ch in house and attended the sick</td>
</tr>
<tr>
<td>367</td>
<td>Elizabeth</td>
<td>Jenners</td>
<td>F</td>
<td>15 Thomson Court</td>
<td>22</td>
<td>servant</td>
<td>09-Jan-49</td>
<td>24 h</td>
<td>CH/mustard emetic/stomach pump</td>
<td>C</td>
<td></td>
<td>not ch stupor from alcohol</td>
</tr>
<tr>
<td>368</td>
<td>Elizabeth</td>
<td>Hamilton</td>
<td>F</td>
<td>5 Leith St Terrace</td>
<td>36</td>
<td>servant</td>
<td>09-Jan-49</td>
<td>2 d</td>
<td>CA/St/VS/Digitalis</td>
<td>D</td>
<td>Y</td>
<td>in Glasgow where ch v</td>
</tr>
<tr>
<td>369</td>
<td>Biddy</td>
<td>?</td>
<td>F</td>
<td>Cants Close</td>
<td>50</td>
<td>seamstress</td>
<td>11-Jan-49</td>
<td>2 d</td>
<td>Gin/CA/Digitalis</td>
<td>D</td>
<td></td>
<td>ch in close but saw no one</td>
</tr>
<tr>
<td>370</td>
<td>John</td>
<td>Moses</td>
<td>M</td>
<td>South Foulis Close</td>
<td>51</td>
<td>porter</td>
<td>14-Jan-49</td>
<td>16 h</td>
<td>VS/CA+O/Gin</td>
<td>D</td>
<td></td>
<td>ch in the house</td>
</tr>
<tr>
<td>371</td>
<td>Hugh</td>
<td>Smith or?</td>
<td>M</td>
<td>Cants Close</td>
<td>20</td>
<td>labourer</td>
<td>15-Jan-49</td>
<td>28 h</td>
<td>CH/St</td>
<td>D</td>
<td></td>
<td>ch in close</td>
</tr>
<tr>
<td>372</td>
<td>Elizabeth</td>
<td>Collins</td>
<td>F</td>
<td>Brodie's Close Cowgate</td>
<td>19 H</td>
<td>labourer</td>
<td>02-Jan-49</td>
<td>19 d</td>
<td>VS(24)CA+O/ST/or ema</td>
<td>C</td>
<td></td>
<td>ch in house</td>
</tr>
<tr>
<td>373</td>
<td>Marianne</td>
<td>Straiton</td>
<td>F</td>
<td>100 Lauriston Place</td>
<td>20</td>
<td>servant</td>
<td>18-Jan-49</td>
<td>2 d</td>
<td>VS(26)CA+O/ST/Dr Mustard</td>
<td>D</td>
<td></td>
<td>no contact; O +Ca given in small and frequent doses</td>
</tr>
<tr>
<td>374</td>
<td>Peter</td>
<td>Haigues</td>
<td>M</td>
<td>Brodie’s Close Cowgate</td>
<td>30</td>
<td>labourer</td>
<td>19-Jan-49</td>
<td>2 d</td>
<td>D/ST/CA+O/IV/mustard</td>
<td>D</td>
<td>Y</td>
<td>ineffectual ch in house; CA+O in small doses frequently given when condition</td>
</tr>
<tr>
<td>375</td>
<td>Colin</td>
<td>Henderson</td>
<td>M</td>
<td>Dunbar’s Close Canongate</td>
<td>22</td>
<td>smith</td>
<td>16-Jan-49</td>
<td>6 d</td>
<td>D/VS/Enemata/crotone</td>
<td>Y</td>
<td></td>
<td>seemed utterly hopeless up to 1Id given with transitory improvement</td>
</tr>
<tr>
<td>376</td>
<td>Luke</td>
<td>McDonald</td>
<td>M</td>
<td>Brodie's Close Cowgate</td>
<td>30</td>
<td>labourer</td>
<td>20-Jan-49</td>
<td>2 d</td>
<td>IV/ST/CA/CH</td>
<td>D</td>
<td>Y</td>
<td>no contact</td>
</tr>
<tr>
<td>377</td>
<td>Marianne</td>
<td>Robertson</td>
<td>F</td>
<td>Infirmary</td>
<td>23</td>
<td>servant</td>
<td>21-Jan-49</td>
<td>4 d</td>
<td>CA+O/ST/CH/BL/OR/VS</td>
<td>D</td>
<td>Y</td>
<td>ch in house; given homeopathic treatment in house; IV produced</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>temporary improvement a patient in ward in</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Infirmary for 4 mins with eczema of head had</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>examined a ch stool 36 hrs before attack</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID</td>
<td>First Name</td>
<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
</tr>
<tr>
<td>-----</td>
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<td>-----</td>
<td>--------------------------------</td>
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<td>---------------------------------------------------------------------------</td>
<td>-----</td>
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<td>-------------------------------------------</td>
</tr>
<tr>
<td>378</td>
<td>Elizabeth</td>
<td>Scott</td>
<td>F</td>
<td>Whitehorse Close Canongate</td>
<td>25</td>
<td>servant</td>
<td>30-Dec-48</td>
<td>35 d</td>
<td>Mustard emetic/capsicum gargle/Hydrarg/Hypt hths</td>
<td>C</td>
<td></td>
<td>ch in close; doubtful case; Syphilis in throat</td>
</tr>
<tr>
<td>379</td>
<td>Michael</td>
<td>Cain</td>
<td>M</td>
<td>46 South Foulis Close</td>
<td>12</td>
<td>hawker</td>
<td>17-Jan-49</td>
<td>13 d</td>
<td>W/w chalk mixture</td>
<td>C</td>
<td></td>
<td>ch in stair</td>
</tr>
<tr>
<td>380</td>
<td>Andrew</td>
<td>Clark</td>
<td>M</td>
<td>136 Cowgate</td>
<td>20</td>
<td>labourer</td>
<td>22-Jan-49</td>
<td>5 d</td>
<td>CA+O/ST/IV/Mist Potassium Digestives et Colchicum/pec/Ene ma/Stomach pump</td>
<td>D</td>
<td>Y</td>
<td>ch in house; 20 oz saline injected no reaction strong emetics not returned therefore stomach pump used no ch in ward there from 16 Jan no contact</td>
</tr>
<tr>
<td>381</td>
<td>Nancy</td>
<td>Brian</td>
<td>F</td>
<td>Ward 12 RIE previously McConachies Close Cowgate</td>
<td>5</td>
<td>F labourer</td>
<td>23-Jan-49</td>
<td>21 h</td>
<td>CA/St</td>
<td>D</td>
<td></td>
<td>attending a ch patient</td>
</tr>
<tr>
<td>382</td>
<td>Robert</td>
<td>Stewart</td>
<td>M</td>
<td>30 Heriot's Place Vennel</td>
<td>16</td>
<td>cowfeeder</td>
<td>26-Jan-49</td>
<td>4 d</td>
<td>CA/OR/</td>
<td>C</td>
<td></td>
<td>no contact; bloodletting before giving IV f/b; VS appeared to avert the collapse</td>
</tr>
<tr>
<td>383</td>
<td>Margaret</td>
<td>Muirhead</td>
<td>F</td>
<td>Chalmers Close</td>
<td>42</td>
<td>seamstress</td>
<td>24-Jan-49</td>
<td>11 h</td>
<td>VS/0/enema/Ca in small doses/IV/ST/CA in small doses/VS</td>
<td>D</td>
<td>Y</td>
<td>attending a ch patient</td>
</tr>
<tr>
<td>384</td>
<td>Christina</td>
<td>Cathcart</td>
<td>F</td>
<td>Weirs Close Canongate</td>
<td>45</td>
<td>W</td>
<td>25-Jan-49</td>
<td>34 h</td>
<td>ST/O/enema/IV/L+O</td>
<td>Y</td>
<td></td>
<td>ch in ward; lb 5 and 6 IV with marked temporary improvement attempt to repeat IV failed contact not known; attempt to bleed failed before failed attempt at IV M died of ch; hooping cough after recovery from ch ch in neighbourhood</td>
</tr>
<tr>
<td>385</td>
<td>Isabella</td>
<td>Sech</td>
<td>F</td>
<td>Ward 11 RIE</td>
<td>48</td>
<td>servant</td>
<td>30-Jan-49</td>
<td>32 h</td>
<td>ST/O/enema/IV/L+O</td>
<td>Y</td>
<td></td>
<td>attending a ch patient</td>
</tr>
<tr>
<td>386</td>
<td>David</td>
<td>Ross</td>
<td>M</td>
<td>Near the links Leith</td>
<td>26</td>
<td>labourer</td>
<td>31-Jan-49</td>
<td>16 h</td>
<td>VS(2)/St/CH/T enema</td>
<td>Y</td>
<td></td>
<td>attending a ch patient</td>
</tr>
<tr>
<td>387</td>
<td>James</td>
<td>Stewart</td>
<td>M</td>
<td>Quarantine House Forrest Rd</td>
<td>26</td>
<td>labourer</td>
<td>31-Jan-49</td>
<td>16 h</td>
<td>VS(2)/St/CH/T enema</td>
<td>Y</td>
<td></td>
<td>attending a ch patient</td>
</tr>
<tr>
<td>388</td>
<td>William</td>
<td>Lofthouse</td>
<td>M</td>
<td>162 Cowgate</td>
<td>24</td>
<td>tailor</td>
<td>30-Dec-48</td>
<td>35 d</td>
<td>VS(22)/CA+O/D</td>
<td>C</td>
<td></td>
<td>ch in ward; dysentery followed ch no contact</td>
</tr>
<tr>
<td>389</td>
<td>Grace</td>
<td>Silver</td>
<td>F</td>
<td>Ward 17 RIE previously 26 Richmond</td>
<td>19</td>
<td>servant</td>
<td>01-Jan-49</td>
<td>42 d</td>
<td>CH/P/CA+O/OR/BL/ C VS/pec+O St/VS/CA+O/BL/O C VS/P/L+O/C</td>
<td>C</td>
<td></td>
<td>ch in ward; dysentery followed ch no contact</td>
</tr>
<tr>
<td>390</td>
<td>Eliza</td>
<td>Nixon</td>
<td>F</td>
<td>8 Cowgatehead</td>
<td>15</td>
<td>servant</td>
<td>06-Jan-49</td>
<td>29 d</td>
<td>VS/CA+O/ST/L/Dov C er’s/Nux vomicar/L+O/chalk mixture</td>
<td>C</td>
<td></td>
<td>no ch in close for some considerable time</td>
</tr>
<tr>
<td>391</td>
<td>John</td>
<td>?Eigo</td>
<td>M</td>
<td>North Grays Close</td>
<td>50</td>
<td>labourer</td>
<td>10-Jan-49</td>
<td>64 d</td>
<td>OR/D/enema</td>
<td>C</td>
<td></td>
<td>probably not ch</td>
</tr>
<tr>
<td>392</td>
<td>Agnes</td>
<td>Watt</td>
<td>F</td>
<td>10 Low Calton</td>
<td>36</td>
<td>washerwoman</td>
<td>16-Jan-49</td>
<td>20 d</td>
<td>VS/CA+O/ST/L/Dov C er’s/Nux vomicar/L+O/chalk mixture</td>
<td>C</td>
<td></td>
<td>probably not ch</td>
</tr>
<tr>
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<td>Contact ch and others</td>
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<td>63</td>
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<td>55</td>
<td>H printer</td>
<td>10-Feb-49 12 h</td>
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<td>53</td>
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<td>Morrine</td>
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<td>16-Feb-49 19 d</td>
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<td>Contact ch and others</td>
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<td>ST/OR/D/VS(15)/L</td>
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<td>22</td>
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<td>CA+O/D/S/VS/914/ Bombay mixture and pills</td>
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<td>?</td>
<td>04-Mar-49</td>
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<td>VS(4)/S/CH/P/O/CA+O</td>
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<td>20-Feb-49</td>
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<td>OR</td>
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<td>Bain</td>
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<td>10-Mar-49</td>
<td>9 d</td>
<td>Or</td>
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<td>3 d</td>
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<td>F</td>
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<td>washerwoman</td>
<td>06-Sep-49</td>
<td>30 h</td>
<td>Potassium chlor/sodium chloride</td>
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<td>C</td>
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<td>23-Oct-49</td>
<td>18 h</td>
<td>hot wet sheet /cold water</td>
<td>D</td>
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<td>F</td>
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<td>F</td>
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<td>18 d</td>
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<td>Contact ch and others</td>
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<td>ch in land saw those ill 3 days ago no contact</td>
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<td>F</td>
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<td>15 h</td>
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<td>21-Oct-49</td>
<td>36 h</td>
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<td>D</td>
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<td>Sloane</td>
<td>F</td>
<td>48 St Mary's Wynd</td>
<td>27</td>
<td>W of shoemaker</td>
<td>21-Oct-49</td>
<td>6 d</td>
<td>St/O/hot wet sheet</td>
<td>D</td>
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</tr>
<tr>
<td>446</td>
<td>Janet</td>
<td>Rankin</td>
<td>F</td>
<td>27 Blackfriar's Wynd</td>
<td>43</td>
<td>charwoman</td>
<td>23-Oct-49</td>
<td>23 h</td>
<td></td>
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<td>ch in house attended those ill landlady died of ch did not see her ch in the square</td>
</tr>
<tr>
<td>447</td>
<td>David</td>
<td>Rule</td>
<td>M</td>
<td>Geddes Close</td>
<td>69</td>
<td>clerk in police</td>
<td>14-Oct-49</td>
<td>6 d</td>
<td>Hot wet sheet /St/purgatives/CO</td>
<td>D</td>
<td></td>
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<tr>
<td>448</td>
<td>John</td>
<td>Brash</td>
<td>M</td>
<td>7 Shakespeare Square</td>
<td>25</td>
<td>discharged private</td>
<td>17-Oct-49</td>
<td>2 d</td>
<td>Hot wet sheet /cold water</td>
<td>D</td>
<td></td>
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<tr>
<td>449</td>
<td>Anne</td>
<td>Queen</td>
<td>F</td>
<td>West Linton</td>
<td>40</td>
<td>servant</td>
<td>19-Oct-49</td>
<td>10 d</td>
<td>Pil Acetate Plumbi</td>
<td>C</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>450</td>
<td>Isabella</td>
<td>McKay</td>
<td>F</td>
<td>Havelaw's Close</td>
<td>41</td>
<td>H porter</td>
<td>05-Oct-49</td>
<td>10 d</td>
<td>CA+O</td>
<td>C</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>451</td>
<td>Michael</td>
<td>McBride</td>
<td>M</td>
<td>Baillie Fyfe's Close</td>
<td>42</td>
<td>hawker</td>
<td>07-Oct-49</td>
<td>2 d</td>
<td>Hot wet sheet /T/W/Mustard L+O/enema/O/Pil Acetate Plumbi</td>
<td>C</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>452</td>
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<td>Brown</td>
<td>F</td>
<td>Cholera Hospital</td>
<td>22</td>
<td>nurse</td>
<td>07-Oct-49</td>
<td>2 d</td>
<td>CA/CO/Pulv/jalap</td>
<td>C</td>
<td></td>
<td>yes contact</td>
</tr>
<tr>
<td>453</td>
<td>Agnes</td>
<td>McRae</td>
<td>F</td>
<td>12 Morton St</td>
<td>36</td>
<td>H smith</td>
<td>07-Oct-49</td>
<td>3 d</td>
<td></td>
<td></td>
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<td>daughter had ch 6 days ago in same house left Broxburn 2 days ago where she saw person with ch ch in close</td>
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<td>454</td>
<td>Ellen</td>
<td>Manuels</td>
<td>F</td>
<td>Little Hamilton's Close Grassmarket</td>
<td>27</td>
<td>H labourer</td>
<td>09-Oct-49</td>
<td>30 h</td>
<td>T/hot wet sheet</td>
<td>D</td>
<td></td>
<td></td>
</tr>
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<td>455</td>
<td>Anne</td>
<td>Smith</td>
<td>F</td>
<td>South Foulis Close</td>
<td>15</td>
<td>D of washerwoman</td>
<td>09-Oct-49</td>
<td>6 d</td>
<td>L+O</td>
<td>C</td>
<td></td>
<td>ch in close</td>
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<tr>
<td>456</td>
<td>Christina</td>
<td>Lane</td>
<td>F</td>
<td>135 North Grays Close</td>
<td>16</td>
<td>F weaver</td>
<td>07-Sep-49</td>
<td>5 d</td>
<td>D/purgatives/cola drinks/co?</td>
<td>D</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>457</td>
<td>John</td>
<td>Handyside</td>
<td>M</td>
<td>no address given</td>
<td>21</td>
<td>fireman on board Victoria London and Edinburgh steamer porter</td>
<td>07-Sep-49</td>
<td>5 d</td>
<td>T/D/BL/V/S(12)/Purgatives/hot wet sheet</td>
<td>D</td>
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<td>a passenger seized one hour previously</td>
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<td>458</td>
<td>Roderick</td>
<td>McKenzie</td>
<td>M</td>
<td>Cholera Hospital</td>
<td>27</td>
<td>porter</td>
<td>02-Sep-49</td>
<td>11 d</td>
<td>O/CA+O/Spec</td>
<td>C</td>
<td></td>
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</tr>
<tr>
<td>459</td>
<td>Owen</td>
<td>Laver</td>
<td>M</td>
<td>Craigmiliar</td>
<td>24</td>
<td>labourer</td>
<td>28-Aug-49</td>
<td>7 d</td>
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<td>D</td>
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<td>460</td>
<td>Janet</td>
<td>Gray</td>
<td>F</td>
<td>3 Haste's Close Cowgate</td>
<td>39</td>
<td>W of tailor</td>
<td>04-Sep-49</td>
<td>11 d</td>
<td>O/Spec</td>
<td>C</td>
<td></td>
<td>ch in house</td>
</tr>
<tr>
<td>461</td>
<td>Mary</td>
<td>Smith</td>
<td>F</td>
<td>South Foulis Close</td>
<td>16</td>
<td>F labourer</td>
<td>06-Sep-49</td>
<td>5 d</td>
<td>D/BL/purgatives/sedatives</td>
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<td>First Name</td>
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<td>Sex</td>
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<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
</tr>
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<td>462</td>
<td>Anne</td>
<td>Rice</td>
<td>F</td>
<td>1 Milne's Court</td>
<td>66</td>
<td>H shoemaker</td>
<td>06-Sep-49 8 d</td>
<td>T/CA+O/hot wet sheet</td>
<td>C</td>
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<td>463</td>
<td>Charlotte</td>
<td>McKay</td>
<td>F</td>
<td>80 West Bow</td>
<td>28</td>
<td>seamstress</td>
<td>07-Sep-49 5 d</td>
<td>T/hot wet sheet</td>
<td>C</td>
<td>no contact</td>
<td></td>
<td></td>
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<tr>
<td>464</td>
<td>John</td>
<td>Stewart</td>
<td>M</td>
<td>Whitehorse Close Canongate</td>
<td>49</td>
<td>mason</td>
<td>09-Sep-49 4 d</td>
<td>D/purgatives/hot wet sheet</td>
<td>D</td>
<td>ch in close</td>
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<td></td>
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<tr>
<td>465</td>
<td>Jessie</td>
<td>McDonald</td>
<td>F</td>
<td>Steven laws Close</td>
<td>22</td>
<td>staymaker</td>
<td>04-Oct-49 7 d</td>
<td>T/hot wet sheet/CO/Pot chol</td>
<td>D</td>
<td>ch in close</td>
<td></td>
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<tr>
<td>466</td>
<td>Janet</td>
<td>Sutherland</td>
<td>F</td>
<td>Brooks Land Stevenlaws Close</td>
<td>34</td>
<td>H slater</td>
<td>04-Oct-49 5 d</td>
<td>T/hot wet sheet/Wenema</td>
<td>C</td>
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<td></td>
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<td>467</td>
<td>Thomas</td>
<td>Mcguigan</td>
<td>M</td>
<td>Carubbers Close High St</td>
<td>48</td>
<td>porter</td>
<td>05-Oct-49 4 d</td>
<td>CH/hot wet sheet/cold water</td>
<td>D</td>
<td>no contact</td>
<td></td>
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</tr>
<tr>
<td>468</td>
<td>Mary</td>
<td>McGregor</td>
<td>F</td>
<td>Warristons Close</td>
<td>36</td>
<td>washerwoman</td>
<td>28-Sep-49 30 h</td>
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<td>Ferrier</td>
<td>M</td>
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<td>29-Sep-49 11 d</td>
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<td>Arnott</td>
<td>M</td>
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<td>21</td>
<td>cabinetmaker</td>
<td>29-Sep-49 21 h</td>
<td>Iodine/hot sheet</td>
<td>D</td>
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<td>Mack</td>
<td>F</td>
<td>?Rub Close Grassmarket</td>
<td>17</td>
<td>F labourer</td>
<td>07-Oct-49 3 d</td>
<td>O/ ipex</td>
<td>C</td>
<td>no contact</td>
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<td></td>
</tr>
<tr>
<td>472</td>
<td>Thomas</td>
<td>Grey</td>
<td>M</td>
<td>Middle Meal Market Stair</td>
<td>22</td>
<td>sweep</td>
<td>07-Oct-49 14 d</td>
<td>Hot wet sheet/cold water</td>
<td>C</td>
<td>ch in land, has seen those ill</td>
<td></td>
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<tr>
<td>473</td>
<td>Mary</td>
<td>Ross</td>
<td>F</td>
<td>Toddryck's Wynd/ Bailie Fyffe's</td>
<td>25</td>
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<td>07-Oct-49 4 d</td>
<td>O/ ipex</td>
<td>C</td>
<td>no contact</td>
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<td></td>
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<td>474</td>
<td>Janet</td>
<td>Arnott</td>
<td>F</td>
<td>Days Court East Crosscauseway</td>
<td>16</td>
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<td>05-Oct-49 24 h</td>
<td>T/hot wet sheet/cold water</td>
<td>D</td>
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<td></td>
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</tr>
<tr>
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<td>McDonald</td>
<td>F</td>
<td>26 India Place</td>
<td>54</td>
<td>W of coachman</td>
<td>06-Oct-49 27 h</td>
<td>St/hot wet sheet</td>
<td>D</td>
<td>ch in land saw th</td>
<td></td>
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<tr>
<td>476</td>
<td>Margaret</td>
<td>Stewart</td>
<td>F</td>
<td>256 Cowgate</td>
<td>49</td>
<td>H policeman</td>
<td>04-Oct-49 8 d</td>
<td>Pil Acetate Plumbli</td>
<td>C</td>
<td>no contact</td>
<td></td>
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<tr>
<td>477</td>
<td>John</td>
<td>Mclean</td>
<td>M</td>
<td>Middle Meal Market Stair</td>
<td>46</td>
<td>type founder</td>
<td>30-Sep-49 3 d</td>
<td>CH/T/hot wet sheet</td>
<td>D</td>
<td>no contact</td>
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<tr>
<td>478</td>
<td>William</td>
<td>Ross</td>
<td>M</td>
<td>Castle Hill</td>
<td>49</td>
<td>labourer</td>
<td>30-Sep-49 10 d</td>
<td>D/Purgatives/hot wet sheet</td>
<td>D</td>
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<td>479</td>
<td>John</td>
<td>Fraser</td>
<td>M</td>
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<td>28</td>
<td>type founder</td>
<td>01-Oct-49 4 d</td>
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<td>D</td>
<td>no contact</td>
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<td>480</td>
<td>Margaret</td>
<td>Smith</td>
<td>F</td>
<td>27 South Bridge</td>
<td>30</td>
<td>servant</td>
<td>02-Oct-49 24 h</td>
<td>CH/hot wet sheet/cold water</td>
<td>D</td>
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</tr>
<tr>
<td>481</td>
<td>Jessie</td>
<td>Gow</td>
<td>F</td>
<td>Fleshmarket Close</td>
<td>38</td>
<td>washerwoman</td>
<td>02-Oct-49 29 h</td>
<td>Hot wet sheet/cold water</td>
<td>D</td>
<td>ch in close</td>
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<tr>
<td>482</td>
<td>Widow</td>
<td>Wilson</td>
<td>F</td>
<td>Tolbooth Wynd</td>
<td>37</td>
<td>W of carter</td>
<td>03-Oct-49 15 h</td>
<td>W/hot wet sheet</td>
<td>D</td>
<td>no contact</td>
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<td>483</td>
<td>Janet</td>
<td>Ramsay</td>
<td>F</td>
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<td>19-Oct</td>
<td></td>
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<tr>
<td>484</td>
<td>William</td>
<td>Donachie</td>
<td>M</td>
<td>20 Cowgatehead</td>
<td>42</td>
<td>tinsmith</td>
<td>03-Oct-49 4 d</td>
<td>T/D/Hot wet sheet/cold water</td>
<td>D</td>
<td>ch in house seen those ill</td>
<td></td>
<td></td>
</tr>
<tr>
<td>leID</td>
<td>First Name</td>
<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
<td>PM</td>
<td>Contact ch and others</td>
</tr>
<tr>
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<tr>
<td>485</td>
<td>Sophia</td>
<td>Watt</td>
<td>F</td>
<td>23 Water Lane Leith</td>
<td>38</td>
<td>H porter</td>
<td>04-Oct-49</td>
<td>4 d</td>
<td>T/hot wet sheet/D/Wi/Sal</td>
<td>D</td>
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<td>ch in close has seen those ill</td>
</tr>
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<td>486</td>
<td>Isabella</td>
<td>McKay</td>
<td>F</td>
<td>Blair's Close Castle Hill</td>
<td>45</td>
<td>outworker</td>
<td>03-Oct-49</td>
<td>15 d</td>
<td>ST/T/hot wet sheet</td>
<td>C</td>
<td></td>
<td>ch in house saw no one</td>
</tr>
<tr>
<td>488</td>
<td>Anne</td>
<td>Simons</td>
<td>F</td>
<td>Muckles Close South Leith</td>
<td>50</td>
<td>W of tailor</td>
<td>04-Oct-49</td>
<td>2 d</td>
<td>CA/ST/Julap/hot wet sheet</td>
<td>D</td>
<td></td>
<td>no contact</td>
</tr>
<tr>
<td>489</td>
<td>Catherine</td>
<td>McNab</td>
<td>F</td>
<td>Hastie's Close Cowgate</td>
<td>55</td>
<td>W of?</td>
<td>30-Sep-49</td>
<td>2 d</td>
<td>D/Purgatives/St/hot wet sheet</td>
<td>D</td>
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<td>in attendance on ch patients</td>
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<tr>
<td>490</td>
<td>Jessie</td>
<td>Temple</td>
<td>F</td>
<td>105 High Street</td>
<td>15</td>
<td>F carpenter</td>
<td>26-Sep-49</td>
<td>9 d</td>
<td>Plumbi acel gr V</td>
<td>C</td>
<td></td>
<td>no contact</td>
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<tr>
<td>491</td>
<td>Charles</td>
<td>Wright</td>
<td>M</td>
<td>Fleshmarket Close</td>
<td>62</td>
<td>shoemaker</td>
<td>30-Sep-49</td>
<td>2 d</td>
<td>St/hot wet sheet</td>
<td>D</td>
<td></td>
<td>wife died of ch day before admission has seen ch patient</td>
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<tr>
<td>492</td>
<td>Mary</td>
<td>Macaulay</td>
<td>F</td>
<td>Campbell's Close Canongate</td>
<td>33</td>
<td>H hawker</td>
<td>21-Sep-49</td>
<td>6 d</td>
<td>D/St/Purgatives/Hot wet sheet</td>
<td>D</td>
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<tr>
<td>493</td>
<td>James</td>
<td>Brown</td>
<td>M</td>
<td>97 Grassmarket</td>
<td>?</td>
<td>carter</td>
<td>21-Sep-49</td>
<td>16 h</td>
<td>ST/T/hot wet sheet</td>
<td>D</td>
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<td>?</td>
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<td>494</td>
<td>William</td>
<td>Campbell</td>
<td>M</td>
<td>White Horse Close</td>
<td>37</td>
<td>labourer</td>
<td>22-Sep-49</td>
<td>32 h</td>
<td>ST/T/hot wet sheet</td>
<td>D</td>
<td></td>
<td>ch in close but saw no one</td>
</tr>
<tr>
<td>495</td>
<td>Margaret</td>
<td>Kerr</td>
<td>F</td>
<td>Boyd's Close</td>
<td>28</td>
<td>outworker</td>
<td>21-Sep-49</td>
<td>7 d</td>
<td>T/purgatives/hot wet sheet</td>
<td>C</td>
<td></td>
<td>ch in close</td>
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<td>496</td>
<td>Thomas</td>
<td>Maloney</td>
<td>M</td>
<td>Millers Close</td>
<td>11</td>
<td>F labourer</td>
<td>22-Sep-49</td>
<td>3 d</td>
<td>BL/D/purgatives/hot wet sheet</td>
<td>D</td>
<td></td>
<td>no contact</td>
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<td>Anne</td>
<td>Steel</td>
<td>F</td>
<td>White Horse Close</td>
<td>26</td>
<td>outworker</td>
<td>22-Sep-49</td>
<td>2 d</td>
<td>D/Sal/Purgatives/D hot wet sheet</td>
<td>D</td>
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<td>ch in close has seen persons ill with ch no contact</td>
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<td>498</td>
<td>Marianne</td>
<td>Sage</td>
<td>F</td>
<td>opposite Piershill Barracks</td>
<td>25</td>
<td>H soldier</td>
<td>18-Sep-49</td>
<td>7 d</td>
<td>T/D/Purgatives/hot wet sheet</td>
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<td>61</td>
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<td>36</td>
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<td>18 h</td>
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<td>Period</td>
<td>Treatment</td>
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<td>PM</td>
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<td>15-Sep-49 14 h</td>
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<td>Mono</td>
<td>F</td>
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<td>16-Sep-49 5 d</td>
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<td>D 16-Sep-49</td>
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<td>23-Sep-49 6 d</td>
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<td>Treatment</td>
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<td>PM</td>
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<td>27-Sep-49 24 h</td>
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<td>Wi/Tonics/warmth</td>
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<td>F</td>
<td>43 Rose St</td>
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<td>14-Sep-49 29 h</td>
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<td>14-Sep-49 15 h</td>
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<td>Keir or Shaw</td>
<td>F</td>
<td>199 Cowgate</td>
<td>21</td>
<td>H labourer</td>
<td>C 18-Sep-49</td>
<td>Wi/warmth</td>
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<td>F</td>
<td>35 Lothian Rd</td>
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<td>545</td>
<td>John</td>
<td>Leslie</td>
<td>M</td>
<td>5 Richmond Lane</td>
<td>28</td>
<td>fleshер</td>
<td>05-Sep-49 16 h</td>
<td>Sodil chlor, pot chlor/enema</td>
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<td>saw family member- ch in that house</td>
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Cholera Returns 1848-49
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<th>Address</th>
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<th>Period</th>
<th>Treatment</th>
<th>D/C</th>
<th>PM</th>
<th>Contact ch and others</th>
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<td>Robertson</td>
<td>F</td>
<td>Lumsden Court Leith</td>
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<td>?</td>
<td>05-Sep-49 16 h</td>
<td>Sodi chlor, pot chlor/enema</td>
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<td>Calder</td>
<td>F</td>
<td>8 Gibbs Entry Nicholson St</td>
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<td>?</td>
<td>04-Sep-49 19 h</td>
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<td>no contact</td>
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<td>M</td>
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<td>05-Sep-49 17 h</td>
<td>CA</td>
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<td>D</td>
<td>?</td>
<td>person in house ill with ch; died</td>
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<td>Mcconville</td>
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<td>with ch; attending a ch patient</td>
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<td>47</td>
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<td>? 5-Sep-49</td>
<td>CA/T/Hot wet sheet/enemas</td>
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Cholera Returns 1848–49
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<th>Treatment</th>
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<td>61 Cumberland St Lane West</td>
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<td>servant</td>
<td>09-Feb-49</td>
<td>CA+O/St/D/</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>665</td>
<td>William</td>
<td>Flint</td>
<td>M</td>
<td>9 Saunders St</td>
<td>48</td>
<td>street porter</td>
<td>04-Jan-49</td>
<td>O/Or/VS</td>
<td>D</td>
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</tr>
<tr>
<td>666</td>
<td>Mrs Wauchope</td>
<td>F</td>
<td>33 Brunswick</td>
<td>40+</td>
<td>F</td>
<td>27-Aug-48</td>
<td>St/O/CA+O</td>
<td>C</td>
<td>none Dr says that although before ch in town this a was a def. case</td>
<td>C</td>
</tr>
<tr>
<td>668</td>
<td>Dickson</td>
<td>F</td>
<td>Simpson's Court</td>
<td>69</td>
<td>19-Oct-48</td>
<td>C</td>
<td>none</td>
<td></td>
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<tr>
<td>669</td>
<td>Arthur</td>
<td>Gibson</td>
<td>M</td>
<td>4 Well Close Canongate</td>
<td>51</td>
<td>scavenger</td>
<td>20-Oct-48</td>
<td>O/Or/VS</td>
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<tr>
<td>670</td>
<td>Charles</td>
<td>Davidson</td>
<td>M</td>
<td>Hopes Land Canongate</td>
<td>25</td>
<td>smith</td>
<td>28-Oct-48</td>
<td>CA+O/St/Pot Nil</td>
<td>D</td>
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<tr>
<td>671</td>
<td>Henrietta</td>
<td>McKechnie</td>
<td>F</td>
<td>St Mary's Wynd</td>
<td>19</td>
<td>H chair maker</td>
<td>28-Oct-48</td>
<td>CA+O/Chalk</td>
<td>C</td>
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<tr>
<td>672</td>
<td>Mrs Docherty</td>
<td>F</td>
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<td>46</td>
<td>31-Oct-48</td>
<td>C</td>
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<td>none</td>
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<td>673</td>
<td>Francis</td>
<td>Ba</td>
<td>M</td>
<td>12 Leith Wynd</td>
<td>23</td>
<td>?</td>
<td>01-Nov-48</td>
<td>O/Or/VS</td>
<td>C</td>
<td></td>
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<td>674</td>
<td>John</td>
<td>Hollands</td>
<td>M</td>
<td>Hastie's Close Cowgate</td>
<td>4</td>
<td>?</td>
<td>01-Nov-48</td>
<td>CA+O/St</td>
<td>C</td>
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<td>675</td>
<td>Mrs Kemp</td>
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<td>50</td>
<td>01-Nov-48</td>
<td>D</td>
<td>St</td>
<td>C</td>
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<td>676</td>
<td>Margaret</td>
<td>Petrie</td>
<td>F</td>
<td>16 High St</td>
<td>12</td>
<td>milliner</td>
<td>26-Nov-48</td>
<td>CA+O/St</td>
<td>C</td>
<td></td>
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<tr>
<td>677</td>
<td>Alex</td>
<td>Petrie</td>
<td>M</td>
<td>16 High St</td>
<td>50</td>
<td>gilder</td>
<td>04-Nov-48</td>
<td>CA+O/St</td>
<td>C</td>
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<td>678</td>
<td>Helen</td>
<td>Kelly</td>
<td>F</td>
<td>Toddrick's Wynd</td>
<td>40</td>
<td>04-Nov-48</td>
<td>C</td>
<td>Ch in vicinity 8 people living in a small room with no window</td>
<td>C</td>
<td></td>
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<tr>
<td>679</td>
<td>John</td>
<td>Thomson</td>
<td>M</td>
<td>2 West Norton Place</td>
<td>52</td>
<td>shoemaker</td>
<td>04-Nov-48</td>
<td>CA+O/St</td>
<td>D</td>
<td></td>
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<tr>
<td>680</td>
<td>James</td>
<td>Anderson</td>
<td>M</td>
<td>2 West Norton Place</td>
<td>25</td>
<td>gardener</td>
<td>04-Nov-48</td>
<td>L+O/CA</td>
<td>C</td>
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<td>681</td>
<td>John</td>
<td>Sharp</td>
<td>M</td>
<td>3 Greenside Court</td>
<td>41</td>
<td>partner in stamp office</td>
<td>10-Oct-48</td>
<td>CA+O/St</td>
<td>D</td>
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<td>ID</td>
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<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
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<td>682</td>
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<td>Bishop</td>
<td>F</td>
<td>12 Clyde St</td>
<td>23</td>
<td>prostitute</td>
<td>05-Nov-48</td>
<td>4 d</td>
<td>CA+O/L+O</td>
<td>C</td>
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<td>683</td>
<td>Anne</td>
<td>Wood</td>
<td>F</td>
<td>12 Clyde St</td>
<td>22</td>
<td>prostitute</td>
<td>08-Nov-48</td>
<td>10 h</td>
<td>St</td>
<td>D</td>
</tr>
<tr>
<td>684</td>
<td>Matthew</td>
<td>Thomson</td>
<td>M</td>
<td>7 Shakespeare Square</td>
<td>50</td>
<td>carpenter</td>
<td>08-Nov-48</td>
<td>6 d</td>
<td>CA</td>
<td>C</td>
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<tr>
<td>685</td>
<td>Mrs</td>
<td>Marsden</td>
<td>F</td>
<td>13 Belis Wynd</td>
<td>36</td>
<td>chair maker</td>
<td>10-Nov-48</td>
<td>5 d</td>
<td>CA+O/St</td>
<td>C</td>
</tr>
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<td>686</td>
<td>Miss</td>
<td>Leslie</td>
<td>F</td>
<td>12 Clyde St</td>
<td>30</td>
<td>prostitute</td>
<td>10-Nov-48</td>
<td>16 h</td>
<td>CA+O/St</td>
<td>D</td>
</tr>
<tr>
<td>687</td>
<td>Mrs</td>
<td>McFadyean</td>
<td>F</td>
<td>St James St</td>
<td>36</td>
<td>H soldier</td>
<td>11-Nov-48</td>
<td>2 d</td>
<td>L+O</td>
<td>C</td>
</tr>
<tr>
<td>688</td>
<td>Catherine</td>
<td>Rielly</td>
<td>F</td>
<td>2 Horse Wynd</td>
<td>36</td>
<td>H labourer</td>
<td>12-Nov-48</td>
<td>3 d</td>
<td>St/ether</td>
<td>D</td>
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<tr>
<td>689</td>
<td>Janet</td>
<td>Orr</td>
<td>F</td>
<td>seen at 8 Richmond St</td>
<td>44</td>
<td>servant</td>
<td>13-Nov-48</td>
<td>7 h</td>
<td>Frictions/heat</td>
<td>D</td>
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<tr>
<td>690</td>
<td>Mrs</td>
<td>Mitchell</td>
<td>F</td>
<td>Potter Row</td>
<td>37</td>
<td>washerwoman</td>
<td>12-Nov-48</td>
<td>8 d</td>
<td>CA/Chalk</td>
<td>C</td>
</tr>
<tr>
<td>691</td>
<td>Richard</td>
<td>McGuire</td>
<td>M</td>
<td>105 Grassmarket</td>
<td>70</td>
<td>cobbler</td>
<td>09-Nov-48</td>
<td>2 d</td>
<td>L+O/creosole mixture/St/heat Creosole/O</td>
<td>D</td>
</tr>
<tr>
<td>692</td>
<td>Jane</td>
<td>Peffers</td>
<td>F</td>
<td>Burt's Close Grassmarket</td>
<td>47</td>
<td>hawker</td>
<td>09-Nov-48</td>
<td>14 h</td>
<td>Creosole/O</td>
<td>D</td>
</tr>
<tr>
<td>693</td>
<td>Margaret</td>
<td>Johnston</td>
<td>F</td>
<td>Hatters Land/ Burt's Close</td>
<td>67</td>
<td>bag maker for rope maker</td>
<td>13-Nov-48</td>
<td>10 h</td>
<td>CA+O/St</td>
<td>D</td>
</tr>
<tr>
<td>694</td>
<td>Matthew</td>
<td>Ferguson</td>
<td>M</td>
<td>Big Hamilton's Close Grassmarket</td>
<td>85</td>
<td>pauper</td>
<td>14-Nov-48</td>
<td>1 d</td>
<td>St</td>
<td>D</td>
</tr>
<tr>
<td>695</td>
<td>William</td>
<td>Saunders</td>
<td>M</td>
<td>Burt's Close Grassmarket</td>
<td>22</td>
<td>chimney sweep</td>
<td>15-Nov-48</td>
<td>9 d</td>
<td>Ca/St/saline orally</td>
<td>C</td>
</tr>
<tr>
<td>696</td>
<td>James</td>
<td>Burns</td>
<td>M</td>
<td>105 Grassmarket</td>
<td>20</td>
<td>Masons asst</td>
<td>18-Nov-48</td>
<td>5 d</td>
<td>Ca/St/saline orally/inj of ipecac/enemata hot bath/cardamoms/peppermint/caster oil</td>
<td>D</td>
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<tr>
<td>697</td>
<td>Abigail</td>
<td>McCarter</td>
<td>F</td>
<td>W Smiths Close Grassmarket</td>
<td>1</td>
<td>orphan</td>
<td>25-Nov-48</td>
<td>20 h</td>
<td>hot bath/cardamoms/peppermint/caster oil</td>
<td>D</td>
</tr>
<tr>
<td>698</td>
<td>Mrs</td>
<td>McDonald</td>
<td>F</td>
<td>Royal Mint Cowgate</td>
<td>52</td>
<td>Acting sick nurse</td>
<td>20-Nov-48</td>
<td>4 d</td>
<td>CA+O/Camphor/St/Pepper</td>
<td>D</td>
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<tr>
<td>699</td>
<td>?</td>
<td>Sheppard</td>
<td>F</td>
<td>Weirs Close Canongate</td>
<td>13</td>
<td></td>
<td>03-Oct-48</td>
<td>12 h</td>
<td>Astringents</td>
<td>D</td>
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<tr>
<td>700</td>
<td>William</td>
<td>McGregor</td>
<td>M</td>
<td>62 Thistle St</td>
<td>65</td>
<td>street porter</td>
<td>31-Dec-48</td>
<td>20 h</td>
<td>St</td>
<td>D</td>
</tr>
<tr>
<td>iID</td>
<td>First Name</td>
<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
</tr>
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<td>-----</td>
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</tr>
<tr>
<td>701</td>
<td>Elizabeth</td>
<td>Ross</td>
<td>F</td>
<td>Goods Entry College Wynd</td>
<td>33</td>
<td>labourer</td>
<td>29-Nov-48 2 d</td>
<td>Heat/CA+O/St</td>
<td>D</td>
<td>cases of ch in close</td>
</tr>
<tr>
<td>702</td>
<td>Edward</td>
<td>Daly</td>
<td>M</td>
<td>Hastie's Close Cowgate</td>
<td>25</td>
<td>hawker</td>
<td>05-Nov-48 14 h</td>
<td>Heat/CA+O/St/ether</td>
<td>D</td>
<td>all day in shop in Cowgate</td>
</tr>
<tr>
<td>703</td>
<td>Janet</td>
<td>Walls</td>
<td>F</td>
<td>309 Canongate</td>
<td>32</td>
<td>broker and grocer</td>
<td>24-Oct-48 6 d</td>
<td>O/St/Laudanum/her</td>
<td>C</td>
<td>slept with 2 children with scarlet fever</td>
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<tr>
<td>704</td>
<td>Anne</td>
<td>Ward</td>
<td>F</td>
<td>West Fishmarket Close</td>
<td>7 F carpenter</td>
<td>19-Nov-48 2 d</td>
<td>Heat</td>
<td>D</td>
<td>bought a bed of ch victim who nursed him took ch on morning of his death and died in Ch H on 22 Nov</td>
<td></td>
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<tr>
<td>705</td>
<td>Catherine</td>
<td>Dillon</td>
<td>F</td>
<td>Spirit Shop Cowgate</td>
<td>21</td>
<td>spirit dealer</td>
<td>09-Nov-48 2 d</td>
<td>CA+O/CH/Ether/Heal</td>
<td>C</td>
<td>Dr P Newbigging</td>
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<tr>
<td>706</td>
<td>Donald</td>
<td>McDougal</td>
<td>M</td>
<td>Bannermans Close Cowgate</td>
<td>40</td>
<td>furniture broker and undertaker</td>
<td>19-Nov-48 2 d</td>
<td>CA+O/Capsicum/Ether/morphine</td>
<td>D</td>
<td>none</td>
</tr>
<tr>
<td>707</td>
<td>A</td>
<td>B</td>
<td>F</td>
<td>24 Gayfield Square</td>
<td>68</td>
<td>?</td>
<td>12-Nov-48 12 h</td>
<td>CA+O/Sl</td>
<td>D</td>
<td>sister had ch name</td>
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<td>708</td>
<td>A</td>
<td>B</td>
<td>F</td>
<td>24 Gayfield Square</td>
<td>40</td>
<td>?</td>
<td>13-Nov-48 10 h</td>
<td>CA+O/Sl/Laudanum</td>
<td>D</td>
<td>attended William Larken</td>
</tr>
<tr>
<td>709</td>
<td>William</td>
<td>Larkin</td>
<td>M</td>
<td>18 Pleasance</td>
<td>40</td>
<td>coal carter</td>
<td>15-Nov-48 3 d</td>
<td>VS 16/O/caster oil enema</td>
<td>C</td>
<td>no contact</td>
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<tr>
<td>710</td>
<td>Mary Anne</td>
<td>Clark</td>
<td>F</td>
<td>21 Drummond St</td>
<td>38</td>
<td>mangle keeper</td>
<td>29-Nov-48 4 d</td>
<td>O/St</td>
<td>D</td>
<td>attended her husband having come from the country to do so ch in stair</td>
</tr>
<tr>
<td>711</td>
<td>Janet</td>
<td>Spence</td>
<td>F</td>
<td>?</td>
<td>36</td>
<td>Nurse</td>
<td>04-Dec-48 2 d</td>
<td>O/VS</td>
<td>C</td>
<td>ch in same fiat</td>
</tr>
<tr>
<td>712</td>
<td>John</td>
<td>?D</td>
<td>M</td>
<td>21 James Sq</td>
<td>30</td>
<td>gunsmith</td>
<td>26-Aug-49 3 d</td>
<td>CA+O/Chalk/Laudanum</td>
<td>C</td>
<td>no contact</td>
</tr>
<tr>
<td>713</td>
<td>Mrs</td>
<td>D?</td>
<td>F</td>
<td>21 James Sq</td>
<td>30</td>
<td>Wife of above</td>
<td>26-Aug-49 3 d</td>
<td>CA+O/Chalk/Laudanum</td>
<td>C</td>
<td>British cholera</td>
</tr>
<tr>
<td>714</td>
<td>John</td>
<td>Rogers</td>
<td>M</td>
<td>Old Fishmarket Close</td>
<td>45</td>
<td>shoemaker</td>
<td>01-Jan-49 3 d</td>
<td>CA/Dover's/Creosote</td>
<td>D</td>
<td>ch in neighbourhood</td>
</tr>
<tr>
<td>715</td>
<td>Mrs</td>
<td>Simpion</td>
<td>F</td>
<td>Craig's Close High St</td>
<td>34</td>
<td>servant</td>
<td>27-Dec-48 8 d</td>
<td>CA+O/D/Or/Creosote</td>
<td>C</td>
<td>ch in vicinity</td>
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<tr>
<td>716</td>
<td>George</td>
<td>Robertson</td>
<td>M</td>
<td>50 George St</td>
<td>32</td>
<td>writers clerk</td>
<td>27-Dec-48 3 d</td>
<td>D/St</td>
<td>D</td>
<td>case in close</td>
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<tr>
<td>717</td>
<td>Mrs</td>
<td>McKenna</td>
<td>F</td>
<td>57 Blackfriar's Wynd</td>
<td>30</td>
<td>labourer</td>
<td>01-Jan-49 8 d</td>
<td>CA+O/D/Or/Creosote</td>
<td>C</td>
<td>sent to H where she died</td>
</tr>
<tr>
<td>718</td>
<td>Eliza</td>
<td>Smith</td>
<td>F</td>
<td>1 Cumberland St</td>
<td>14</td>
<td>Woman's D</td>
<td>04-Jan-49 4 d</td>
<td>CA+O/D/Or/Creosote</td>
<td>C</td>
<td>case in close</td>
</tr>
<tr>
<td>719</td>
<td>Mrs</td>
<td>McDonald</td>
<td>F</td>
<td>Canongate</td>
<td>62</td>
<td>pauper</td>
<td>10-Nov-48 10 h</td>
<td>CA+O/Lrad acetate</td>
<td>?</td>
<td>case in close</td>
</tr>
<tr>
<td>720</td>
<td>Anne</td>
<td>Aitken</td>
<td>F</td>
<td>Bailiff Fyfe's Close</td>
<td>48</td>
<td>pauper</td>
<td>10-Nov-48</td>
<td>CA+O/Lrad acetate</td>
<td>?</td>
<td>case in close</td>
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<tr>
<td>721</td>
<td>Margaret</td>
<td>Johnston</td>
<td>F</td>
<td>Baron Grants Close</td>
<td>6 F hawker</td>
<td>12-Nov-48 24 h</td>
<td>CA+O</td>
<td>D</td>
<td>case in close</td>
<td></td>
</tr>
<tr>
<td>722</td>
<td>Mary</td>
<td>Cornwell</td>
<td>F</td>
<td></td>
<td>40</td>
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<td></td>
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<tr>
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<td>Surname</td>
<td>Sex</td>
<td>Address</td>
<td>Age</td>
<td>Occupation</td>
<td>Admitted</td>
<td>Period</td>
<td>Treatment</td>
<td>D/C</td>
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<tr>
<td>723</td>
<td>Lily</td>
<td>Law</td>
<td>F</td>
<td>N Grays Close</td>
<td>35</td>
<td>pauper</td>
<td>16-Nov-48</td>
<td>CA+O</td>
<td></td>
<td>C</td>
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<td>724</td>
<td>C</td>
<td>Dunsmure</td>
<td>F</td>
<td>Hyndfords Close High St</td>
<td>40</td>
<td>pauper</td>
<td>20-Nov-48</td>
<td>CA+O</td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>725</td>
<td>Mrs</td>
<td>Millar</td>
<td>F</td>
<td>Richmond Pend</td>
<td>52</td>
<td>Widow who wrought at hay making when able</td>
<td>23-Feb-49</td>
<td>CA/D/Dover's</td>
<td></td>
<td>D</td>
</tr>
<tr>
<td>726</td>
<td>Mrs</td>
<td>Reid</td>
<td>F</td>
<td>Cottage off Bonnington Rd</td>
<td>30</td>
<td>H gardener</td>
<td>18-Nov-48</td>
<td>O/D/St</td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>727</td>
<td>Isabella</td>
<td>Bennett</td>
<td>F</td>
<td>61 N Lane W Cumberland St</td>
<td>45</td>
<td>Cook</td>
<td>23-Dec-48</td>
<td>CA+O/D</td>
<td></td>
<td>D</td>
</tr>
<tr>
<td>728</td>
<td>Mrs</td>
<td>Riddle</td>
<td>F</td>
<td>4 Allan St</td>
<td>48</td>
<td>Potato gatherer at the time</td>
<td>04-Oct-48</td>
<td>O/D/St</td>
<td></td>
<td>C</td>
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<td>729</td>
<td>Isabella</td>
<td>Taylor</td>
<td>F</td>
<td>Ann's Court Canonmills</td>
<td>46</td>
<td>none</td>
<td>31-Oct-48</td>
<td>St</td>
<td></td>
<td>D</td>
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<td>730</td>
<td>Henry</td>
<td>Jackson</td>
<td>M</td>
<td>5 Saunders St</td>
<td>40</td>
<td>labourer</td>
<td>02-Nov-48</td>
<td>O/St</td>
<td></td>
<td>C</td>
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<tr>
<td>731</td>
<td>Eliza</td>
<td>Dow</td>
<td>F</td>
<td>3 Orchardfield Pl. Leith Walk</td>
<td>40</td>
<td>sewer</td>
<td>20-Nov-48</td>
<td>O/St</td>
<td></td>
<td>C</td>
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<tr>
<td>732</td>
<td>John</td>
<td>Roberton</td>
<td>M</td>
<td>Smiths Land Canonmills</td>
<td>69</td>
<td>porter</td>
<td>24-Nov-48</td>
<td>none too late</td>
<td></td>
<td>D</td>
</tr>
<tr>
<td>733</td>
<td>William</td>
<td>Ritchie</td>
<td>M</td>
<td>? Neils Land Canonmills</td>
<td>35</td>
<td>Masons</td>
<td>09-Jan-49</td>
<td>St/heat</td>
<td></td>
<td>D</td>
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<tr>
<td>734</td>
<td>Mrs</td>
<td>Hill</td>
<td>F</td>
<td>Broughton St</td>
<td>46</td>
<td>vegetable shop + keeps lodgers</td>
<td>10-Jan-49</td>
<td>O/St</td>
<td></td>
<td>C</td>
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<tr>
<td>735</td>
<td>John</td>
<td>Holland's</td>
<td>M</td>
<td>Hastie's Close Cowgate</td>
<td>4</td>
<td>F labourer</td>
<td>04-Nov-48</td>
<td>ST/O/</td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>736</td>
<td>Mrs</td>
<td>Kemp</td>
<td>F</td>
<td>Hastie's Close Cowgate</td>
<td>49</td>
<td>h mason</td>
<td>04-Nov-48</td>
<td>O/St/Laudanum</td>
<td></td>
<td>D</td>
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<tr>
<td>737</td>
<td>James</td>
<td>Sinclair</td>
<td>M</td>
<td>Fleshmarket Close</td>
<td>24</td>
<td>smith</td>
<td>14-Nov-48</td>
<td>O/St</td>
<td></td>
<td>D</td>
</tr>
<tr>
<td>738</td>
<td>Mrs</td>
<td>Clay</td>
<td>F</td>
<td>6 St Mary's Wynd</td>
<td>50</td>
<td>Married</td>
<td>16-Nov-48</td>
<td>CA+O/Dover's/St</td>
<td></td>
<td>C</td>
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<tr>
<td>739</td>
<td>Mrs</td>
<td>Wood</td>
<td>F</td>
<td>21 Carnryan</td>
<td>30</td>
<td>H sailor</td>
<td>12-Oct-48</td>
<td>CA+O/Capsicum</td>
<td></td>
<td>C</td>
</tr>
</tbody>
</table>
0
S and sometimes

Opium
Spirits

Sp

IV

Intra

W

Whisky

D

Diuretic

Bl

Blister

VS and sometimes VE

Venesection/

saline

venous

Bleeding/

Bled
P

Petroleum

CA+O

Calomel &

Opium

CH

Chloroform

B

Brandy

ST and sometimes S

Stimulants

L+O

Lead &

LA+O

basically same

as

Opium

Acetate of Lead and

L+O

Opium

Wi

Wine

LA

Acetate of lead/ lead
acetate

T

Turpentine

PN

Persian

N

Naphtha

CO

Colchicum

M

Morphia

OR

01. Ricini

CAO

Castor oil

H

Heat

BCLofM

Bichloride of mercury
Saline powders

Sal
A

naphtha

=

Castor oil

SQ

Astringents
Squill

B

Bismuth

CP

Capsicum

CM

Camphor
Spirit bath
ipecacuanha

Sp bath
1/ IP/

ipec

E

ether

F/Fr

friction

01

oleum

Dovers

Dover's Powders

Laud

laudanum

Pot. Chlor.

Potassium Chloride

Sodi. Chlor

Sodium Chloride

Pot. Nit.

Potassium Nitrate

Plumbi. Acet.

Lead Acetate

Cholera Database:

Explanation of Symbois

in Treatment Column.

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The column headed “Period” refers to the length of time from onset of cholera to final outcome which is listed in column headed “D/C”; D = Died; C = Cured.

Column headed “PM” indicates whether a post mortem was carried out by the symbol Y.

Column headed “Contact Ch and others” indicates whether the patient had been in contact with cholera and frequently there are comments as to where the contact took place and to the effects of treatment.