

THE IMPACT OF THE NEWER KNOWLEDGE OF NUTRITION:

Nutrition Science and Nutrition Policy, 1900-1939

A thesis submitted to the University of London
for the Degree of Doctor of Philosophy in the
Faculty of Medicine

by

Elizabeth Celia Petty

Department of Human Nutrition,
London School of Hygiene and Tropical Medicine,
Keppel Street,
London WC1E 7HT.

ABSTRACT

Ideas concerning relationships between diet and health in the UK are traced from the 1904 Committee on Physical Deterioration to the outbreak of World War II. Archive material is used to describe the often conflicting views of the Medical Research Council and the Ministry of Health and Board of Education concerning the public health applications of nutrition science. In particular, the work of the Ministry of Health's first Advisory Committee on Nutrition, which was appointed in 1931, is reviewed and evaluated. The debate among public health practitioners over the nature, cause and extent of the 'nutrition problem' is documented and the role in this debate of official dietary guidelines which appeared during the 1930s, is assessed.

The impact of the Newer Knowledge of Nutrition on welfare feeding policy is evaluated in the context of the official promotion of milk feeding in schools. In particular, Corry Mann's experimental evidence which was used to endorse this policy, is reconsidered, and it is shown that the MRC view that the trial was proof of the presence in milk of a "growth factor" which produced preferential growth efficiency in adequately fed children, was in error. From a re-evaluation of the evidence it is suggested that the experiment merely recorded catch-up growth in a group of poorly nourished children. The view that there existed an extensive nutritional problem due to poor quality diets is examined and challenged.

Both dietary survey data and anthropometric evidence are used to present the case that there was throughout the period studied a widespread problem of underfeeding among the poor and that intervention strategies based on the Newer Knowledge were not an appropriate method of dealing with this problem. This casts doubts on the widely held view that there was a need for nutrition education and suggests that the problem was one

of poverty rather than ignorance. Disaggregated anthropometric data located by the author are analysed according to NCHS standards to assess the prevalence of underfeeding. Significantly higher prevalences of stunting than low weight-for-age exist in all data sets; this phenomenon is considered in detail and low weight-for-age is proposed as the preferred index of malnutrition in 20th Century historical studies. Attention is drawn to the relevance of these studies for the current nutrition and public health debate.

TABLE OF CONTENTS

	Page
Abstract	2
Tables	6
Figures	9
Annexes	10
Acknowledgements	12
Introduction	14
Chronology	20
CHAPTER 1: NUTRITION AND PUBLIC HEALTH 1900-1914	21
CHAPTER 2: THE NEWER KNOWLEDGE OF NUTRITION	50
CHAPTER 3: PUBLIC HEALTH APPLICATIONS OF THE NEWER KNOWLEDGE OF NUTRITION	75
Part I: The Public Health Administration and the Medical Research Council.	75
Part II: The Ministry of Health's Advisory Committee on Nutrition, 1931-1934.	101
CHAPTER 4: THEORY INTO PRACTICE	121
Part I : Nutrition and the Public Health Profession, 1919-1929.	121
Part II: Nutrition and Public Health Practice, 1929-1939.	141
CHAPTER 5: NUTRITION INTERVENTION AND THE NEWER KNOWLEDGE OF NUTRITION: The Use of Milk in School Feeding, 1919-1939.	161
CHAPTER 6: AN EVALUATION OF NUTRITIONAL STATUS IN THE UK, 1900-1939.	187
CHAPTER 7: CONCLUDING REMARKS	267
Appendices	270
References	275

<u>TABLES</u>	<u>Page</u>
Table 0.1 Chronology	20
Table 1.1 Chronology of the discovery of major pathogenic organisms.	23
Table 1.2 Estimates of the extent of underfeeding, 1904-1905.	46
Table 1.3 Prevalence of malnutrition among Glasgow School children and among children entering LCC Poor Law Homes, 1906-1912.	48
Table 2.1 Energy intake and growth	60
Table 3.1 Official Malnutrition returns, London and LEAs, 1909-1916 and 1925-1938.	96
Table 4.1 Family income shortfall according to various standard of living criteria, as calculated by Rathbone, 1934.	148
Table 5.1 Estimated cost of increasing the provision of free and subsidised school milk, according to Independent Interdepartmental Committee, 1936.	162
Table 5.2 Number of meals supplied to each child selected for feeding, 1930-1937.	171
Table 5.3 Percentage of school meals provided as milk, 1930-1937.	171
Table 5.4 Nutritional value of conventional school meal compared with nutritional value of 1/3 pint whole milk.	172
Table 5.5 Calculated nutritional impact of school feeding.	172
Table 5.6 Cost of school feeding, 1930-1935.	173
Table 5.7 LEA income thresholds for the provision of free school meals, 1936.	174
Table 5.8 Carnegie Survey (1937): Income groups.	175
Table 5.9 Anthropometric evidence of undernutrition among elementary school children, Carnegie Survey, 1937.	177
Table 5.10 Height increments and nutritional status in Corry Mann's feeding trial.	182
Table 5.11 Weight increments and energy intake in Corry Mann's feeding trial.	184
Table 6.1 Recognition of specific nutrient deficiencies.	188
Table 6.2 Prevalence of poor dark adaptation according to Maitra and Harris, 1937.	193
Table 6.3 Effects of Vitamin A treatment on dark adaptation according to Harris and Abbasey, 1939.	195
Table 6.4 Prevalence of anaemia among the poor population of Aberdeen reported by Davidson et.al., (1933).	200
Table 6.5 Mean Haemoglobin levels recorded in the Carnegie Survey, 1937.	203

	<u>Page</u>	
Table 6.6	Prevalence of rickets recorded by School Medical Officers, 1927.	208
Table 6.7	To illustrate difficulties in assessing rates of stunting from mean height data.	212
Table 6.8	Prevalence of stunting in Glasgow, 1906.	218
Table 6.9	Prevalence of low weight-age in Glasgow, 1906.	219
Table 6.10	Ratio of prevalence of stunting to low weight-age according to class and age in Glasgow, 1906.	220
Table 6.11	Anthropometric assessment of nutritional status among entrants to LCC Poor Law Homes, 1908-1911.	223
Table 6.12	Prevalence of wasting in Glasgow, 1906.	225
Table 6.13	Prevalence of Height for Age over the 90th Centile, Glasgow 1906.	227
Table 6.14	Anthropometric evidence for undernutrition, 1937: Sex Ratios.	229
Table 6.15	Anthropometric evidence for undernutrition, 1937: Age Ratios.	230
Table 6.16	Anthropometric evidence for undernutrition, 1937. Prevalence of height for age and weight for age below the 3rd centile: all subjects.	232
Table 6.17	Anthropometric evidence for undernutrition, 1937. Prevalence of height for age and weight for age above the 75th Centile, all subjects.	232a
Table 6.18	Comparison of the prevalence of stunting and low weight age in populations surveyed in 1936 and 1937.	236
Table 6.19	Comparison of mean heights and weights of rachitic and non rachitic children.	239
Table 6.20	Comparison of Weight for Age, Height for Age and Weight for Height in rachitic and non rachitic children.	240
Table 6.21	Ratio of Stem Height to Total Height in rachitic and non rachitic children.	242
Table 6.22	Prevalence of stunting in Glasgow, 1906, assuming an increase in height of 5 cm in all subjects.	245
Table 6.23	Stunting in Glasgow, 1906, assuming various prevalence rates of rickets.	246
Table 6.24	Desirable nutrient densities calculated from adjusted energy requirement.	252
Table 6.25	Estimated nutrient intakes of 'labouring class' households studied by Rowntree, 1902	256
Table 6.26	Estimated Nutrient Densities of 'labouring class' diets studied by Rowntree, 1902	258

	<u>Page</u>
Table 6.27 Comparison of nutrient intakes in Group I and Group VI households studied in the Carnegie Survey, 1937 showing current RDAs.	260
Table 6.28 Comparison of Nutrient Densities in Group I and Group VI diets studied in the Carnegie Survey, 1937.	261
Table 6.29 Comparison of diets studied by Rowntree in 1902 and Boyd Orr in 1937.	264

FIGURES		<u>Page</u>
Figs 2.1a and 2.1b	Growth of rats in Hopkins's 1912 feeding trials	56
Fig 2.2	Growth of rats fed Vitamin B and Vitamin A deficient diets.	57
Fig 2.3	McCarrison's comparison of rats fed a typical Sikh diet with rats fed a typical poor European diet.	62
Fig 2.4	McCarrison's comparison of rats fed the diets of various Indian ethnic groups.	63
Fig 4.1	Relationship between energy intake and income in Mining families.	129
Fig 4.2	Relationship between income and calories purchased per penny in Mining families.	131
Fig 4.3	Relationship between income and fraction of income spent on food in Mining families.	132
Fig 4.4	Relationship between income and fraction of food expenditure spent on bread and flour in Mining families.	133
Fig 4.5	Average intake of Minerals and Vitamins expressed as percentage of standard requirements according to Boyd Orr, 1936.	146
Fig 5.1	Number of Local Authorities supplying food under the Education (Provision of Meals) Act, 1918 and 1921.	170
Fig 5.2	Milk industry propaganda based on Corry Mann's feeding trial.	179
Fig. 6.1	Mean Haemoglobin levels recorded by Mackay (1935) and Davidson et.al. (1933).	202
Fig 6.2	Correlation between the prevalence of stunting and low weight-age.	222

		<u>Page</u>
Annex 1a	Heights of rachitic and non rachitic children recorded by Tully, Rose, and Mackay and Rose.	247
Annex 1b	Weights of rachitic and non rachitic children recorded by Tully, Rose, and Mackay and Rose.	248

ACKNOWLEDGEMENTS

I am deeply indebted to John Rivers for his inspiring supervision of this work. I am also grateful to Charles Webster of the Wellcome Unit for the History of Medicine in Oxford, without whose initial support and encouragement this project would not have been possible, and to the Wellcome Trustees who funded it. It has been a great privilege to work within the Department of Human Nutrition at the London School of Hygiene and Tropical Medicine and I am extremely grateful to members of the Department for their readiness to share their knowledge and insights. I am particularly grateful to Philip Payne and Erica Wheeler for their interest in the development of historical studies; to Ron Behrens for his very generous help in resolving the many computer-related problems that have arisen in the course of this work; to Ron Page of the Department of Entomology at the LSHTM for his invaluable help with computers in the final stages; to Jane Schauerman and Madeleine Green who gave enormous assistance with word processing; I should also like to thank Philip James of the Rowett Research Institute for his invaluable cooperation in the collection of quantitative data held by the Rowett and Jean James for her kindness during my stay in Aberdeen. My understanding of the nutrition debate during the 1930s has been greatly enhanced by conversations and correspondence with David Lubbock, and I am very grateful to him for his courtesy in passing on his recollections of this period.

Historians of course need archivists and librarians to smooth the path of their research, and I should like to thank in particular the library staff of the London School of Hygiene and Tropical Medicine for their good humour and tolerance, Mary Nicholas of the MRC archives, Angela Raspin of the British Library of Political and Economic Science, and the staff of the Public Records Office.

Finally, I should like to thank my husband, Charles Barber, whose culinary skills ensured that the nutritional status of our two small children did not deteriorate during the course of my research. Without his strength and support this thesis could not have been written.

INTRODUCTION

This is a study of the debate about food and health in the UK during the period 1900-1939. It is a debate which raises important issues for both historians and nutritionists. For historians, the health of a population represents a largely unexplored index of its standard of living and the search for reliable indices of health, which would resolve some of the fundamental questions of economic history, has been the subject of a number of recent studies (1). Nutritionists have also become aware of the potential of historical data and it is now recognised that historical sources may provide important epidemiological material concerning the impact of dietary changes on patterns of disease (2).

Cross disciplinary interest in nutrition can be traced to Drummond's wide ranging historical study, *THE ENGLISHMAN'S FOOD* (3) and this interest has been sustained over the past two decades in seminars held at KQC, London. The KQC seminars have stimulated research into changing patterns of food consumption and drawn attention to the nutritional implications of developments in food production, manufacture and marketing (4); at the same time, studies have been undertaken on changing food habits and the history of the British diet (5) which complement this work. Finally the value of close collaboration between historians and nutritionists in the interpretation of historical data has been demonstrated in the work of Yudkin, Miller, Barker and Oddy (6).

Despite these important studies of the history of the British diet, there is still controversy among historians over crucial issues relating to diet and health. For example, the question of the "healthy or hungry '30s" (7) is unresolved. Yet, in contrast to the controversy which surrounds the 1930s, the view that the subsequent War years were "healthy" and that war time food policy was responsible for this,

remains undisputed (8) . This belief, which was first expounded by the authors of war time food policy themselves (9), helped to establish a view which is now fundamental to nutrition science: that the nutritionist has a vital role to play in the promotion of public health both in the industrial and non industrial world.

The present thesis developed from an interest in the alleged successes of war time food policy. However, it was clear at the outset that a study of the origins of that policy was needed before such an investigation could be undertaken. Work on the origins of war time food policy provided an opportunity to consider a number of questions which are relevant to both historians and nutritionists: for example, what are the best anthropometric indices of nutritional status in historical populations; what are the mechanisms through which laboratory science is translated into public health practice; and how appropriate were nutrition intervention strategies adopted during the period 1900-1939? The structure of this thesis is as follows:

Chapter 1

Ideas about the relationship between diet and disease have intrigued public health administrators as well as nutrition scientists, throughout the present century and although the public health applications of nutrition science are generally identified with the 'Newer Knowledge of Nutrition' which emerged during the period 1900-1920 (10), dietary change had already been proposed as a means of combatting problems of inferior nutritional status or 'physical degeneracy' by the Committee on Physical Deterioration (11) which presented its report in 1904. The dietary explanation for social class inequalities in health has remained popular with governments up to the present time. This approach, which defines much of the ill health in the community as a problem of ignorance and indifference rather than poverty and multiple deprivation,

suggests the comparatively cheap remedial strategy of health education and the origins of this particular form of intervention are discussed in Chapter 1.

Chapter 2

Discoveries in the field of Vitamin biochemistry during the period 1912-1918 seemed to corroborate the view that an 'incorrect' diet was a major cause of the chronic ill health which afflicted the working classes. Chapter 2 documents the experimental basis for these views, which shaped the nutrition debate during the inter war years, and describes their translation into public health goals. These goals were canvassed vigorously by Sir Walter Fletcher, Secretary of the Medical Research Council (MRC) from 1919-1933, and by his successor, Edward Mellanby.

Chapter 3

In 1919, Fletcher envisaged a close collaboration between the MRC and the newly established Ministry of Health in carrying out a programme of nutrition research and intervention, and in 1927 Mellanby set out a "policy for food and health", which he continued to advocate as MRC Secretary. The Ministry of Health accepted the view that changes in the national diet were an important goal for preventive medicine, and it did much to advance the new dietetics, based on the theory of the 'protective foods'. However, it refused to accept the MRC view that dietary deficiency was the sole cause of poor nutritional status. Despite the teachings of the 'Newer Knowledge', the public health administration held firmly to the traditional view of the aetiology of malnutrition, set out by the Committee on Physical Deterioration. According to this view, food was only one of many environmental factors which determined the outcome of the nutritional process. In Chapter 3, the constraints which influenced the Ministry's selective application of the Newer Knowledge are considered and the public health applications of nutrition science are discussed from the perspective of both the public

health administration and the MRC.

The First Advisory Committee on Nutrition, 1931-1934 Public health administrators saw the 'Newer Knowledge' as a means of improving health through self help and thus in 1931, at the height of the Economic Depression, the Ministry of Health convened an expert group, the Advisory Committee on Nutrition. The Committee was asked to produce guidelines which would enable the unemployed to eat a diet which was healthy but cheap. However, the Committee's guidelines were interpreted as minimal standards which in turn led to to accusations of 'widespread malnutrition' among sections of the working class whose low income prevented them from purchasing a 'minimal' diet. This first Nutrition Committee marks a turning point in the debate about food and health. Although it has so far escaped the notice of historians, it will be shown here that its impact was at least as great as that of the 1904 Committee on Physical Deterioration.

Chapter 4

The publications of this Advisory Committee provided a standard, approved by Whitehall, against which diets could be evaluated and many nutrition scientists, politicians, public health workers and welfare reformers subsequently argued that malnutrition could be inferred from family income. However, the public health administration did not accept this point of view and the issue remained highly controversial throughout the 1930s. Although the newer knowledge taught that nutrition was synonymous with food, the administration continued to argue that environmental factors also influenced nutritional status. The Advisory Committee's dietary guidelines and a subsequent BMA report on the minimum cost of a physiologically adequate diet prompted many Medical Officers to reconsider the official position. Chapter 4 considers the theoretical basis of the Ministry's case and traces shifts in opinion

among practicing Medical Officers, concerning the nature and extent of the nutrition problem.

Chapter 5

The public health administration applied the Newer Knowledge of Nutrition selectively. It had been committed to the view that working class diets were deficient in quality rather than in quantity since the pre 1914 years. In the inter-war period, the same view of qualitative deficiency was upheld, but now diets were described as being deficient in the micronutrients, rather than in protein and fat. As a result of this redefinition of the nutrition problem, the Chief Medical Officer encouraged the use of milk in school feeding and the traditional practice of providing high energy meals for malnourished school children increasingly gave way to a lower calorie supplement. The promotion of milk feeding was the cornerstone of official nutrition policy during the 1920s and 1930s and in Chapter 5 its appropriateness as an intervention strategy for the target population, which was made up of children from the lowest income groups, is evaluated. In this Chapter, Corry Mann's feeding trial which provided a scientific rationale for milk feeding is also reconsidered, and it is argued that, contrary to accepted opinion, his results did not demonstrate that milk feeding had a preferential effect on growth.

Chapter 6

A nutritional history of the debates which surrounded the application of the Newer Knowledge of Nutrition in public health practice would be incomplete without an assessment of the actual nature of the nutrition problem as it existed during the period under discussion. This thesis attempts to provide such an assessment and considers clinical observations, dietary survey evidence and anthropometric data. A number of problems which are peculiar to the assessment of nutritional status

in historical populations, limits the use of clinical evidence (and the Registrar General's mortality returns). However, reliable information does exist on dietary intakes and some nutritional analysis of this data has already been undertaken. Important anthropometric data has also survived in both published and unpublished form, which has not previously been subjected to nutritional analysis. Chapter 6 includes a more detailed micronutrient analysis of selected dietary surveys than has hitherto appeared, and an anthropometric assessment of the nutritional status of the members of the child population. Data sets are taken from the first decade of the century, and from the period which immediately preceded the outbreak of the Second World War.

The secular trend in growth is a phenomenon which has long been recognised, and changes in the average heights of populations have been used by Floud and others as a standard of living indicator (12). This work has produced some interesting insights. However, analyses of the secular trend in growth are based on aggregate data which yield no information on individual nutritional status. In the present thesis an analysis of individual children's heights and weights has been carried out, which suggests that the secular trend in height may not be as good an indicator of changes in the nutritional status of the population as the economic historians have generally supposed. This issue is also discussed in Chapter 6.

Table 0.1 CHRONOLOGY

1904	Report of the Committee on Physical Deterioration
1906	Education (Provision of Meals) Act
1907	Inauguration of the School Medical Service
1912	Gowland Hopkins' hypothesis concerning an 'accessory growth promoting factor' in milk published. Funk's vitamin hypothesis set out.
1914-18	World War I
1921	Coal Strike
1924	MRC Report on the Nutrition of Miners and their families
1926	General Strike
1926	MRC Report 'Poverty, Nutrition and Growth'
1926	MRC Report 'Diet for Boys During School Years' (the Corry mann report)
1929	Onset of World Economic Crisis
1931	Convening of Ministry of Health Advisory Committee on Nutrition
1932	Publication of Advisory Committee documents 'The Criticism and Improvement of Diets' and 'Diets for Children in Poor Law Homes'
1933	April: 'Hungry England' report
1933	November: BMA Nutrition Report
1934	'Nutrition' report produced by BMA and Ministry of Health Advisory Committee
1935	Convening of Interdepartmental Advisory Committee on Nutrition
1935	League of Nations report on the 'Physiological Basis of Nutrition'
1936	Boyd Orr's study 'Food, health and Income'
1936	Publication of Interdepartmental Advisory Committee on Nutrition's first report

CHAPTER 1

NUTRITION AND PUBLIC HEALTH, 1900-1914

1.1 Origins of the Public Health Movement

The evolution of species has been described as a process of 'punctuated equilibria', in which periods of stasis are interrupted by short periods of activity, characterised by outbursts of extinctions and new developments. The evolution of the the public health movement since the 1830s might be said to have followed a similar pattern.

In 1831 the United Kingdom was, according to THE TIMES, gripped by a 'great panic'(1) which was caused by the arrival of cholera, a new and deadly disease. Seventy years later another public health alarm broke out. This time , the fear was not of untimely death or sudden bereavement, but of an insidious racial deterioration among the British people. Although mortality rates from infectious disease had been uniformly high during the 18th Century, the cholera epidemic of 1831-2 was the first major epidemic since the 17th Century plagues (2) and it forced government to take action on public health. In 1832 a temporary Board of Health was established, which issued a series of sanitary regulations (3). These marked the beginning of the great public health reform movement of the 19th Century. Initially the Board of Health was concerned with domestic conditions. Local authorities were urged to appoint district inspectors, who were to report on

"the food, clothing and bedding of the poor, the ventilation of their dwellings , space, means of cleanliness, their habits of temperance" (4).

This anticipated by more than seventy years the preoccupations of the public health movement of the present century. In the intervening period, political exigency and developments in medical science combined to deflect the public health movement from an initial concern with problems of poverty and the domestic environment to questions of municipal engineering and the cleansing of public space. Successive

discoveries in the field of microbiology (Table 1.1) (5) provided a rationale for policies which sought to protect society as a whole from the disease organisms which flourished in filth. Yet although an association between poverty and disease was not at the centre of the public health debate during the period of sanitary reform, neither was it entirely neglected. In 1849 Sir John Simon, Principal Medical Officer to the Local Government Board, observed that District (Poor Law) Medical Officers were

"particularly competent to speak on the state of the population in that district, on their customary condition of health, on their liability to epidemic disease...these officers... can supply precisely the kind of detailed and precise information which is most serviceable to your officer of health" (6).

Similarly, Rumsey wrote in his ESSAYS ON STATE MEDICINE of 1856 that

"There are much higher functions of a preventive nature than those of a mere 'public informer' which the district medical officer ought to perform...The state of the apartments of the poor, their clothing and bedding, their choice and preparation of food, the physical management of their children, their nursing in sickness, would all come occasionally under his cognisance" (7).

However, the importance of individuals and their domestic environments was not generally recognised as a priority for the public health movement until the turn of the Century, when concern about the condition of national physique became a major political issue. The redefinition of public health goals can be traced to the Report of the Interdepartmental Committee on Physical Deterioration published in 1904 (8). This Committee, which was summoned to enquire into the nature and extent of the problem of poor physique among the working classes, blamed the ignorance and indifference of working class mothers for the current crisis in national physique and concluded that people, rather than places, were in need of reform. The report, which marks a major turning point in the public health debate is the starting point for the present study.

Table 1.1

CHRONOLOGY OF THE DISCOVERY OF MAJOR PATHOGENIC ORGANISMS

YEAR	DISEASE ORGANISM	INVESTIGATOR
1880	Typhoid (bacillus found on tissue)	Eberth
	Leprosy	Hansen
	Malaria	Laveran
1882	Tuberculosis	Koch
	Glanders	Loeffler and Schultz
1883	Cholera	Koch
	Streptococcus (erysipelas)	Fehleisen
1884	Diphtheria	Klebs and Loeffler
	Tetanus	Nicolaier
1885	Coli	Escherich
1886	Pneumococcus	A. Fraenkel
1892	Gas gangrene	Welch and Nuttal
1894	Plague	Yersi, Kitasato
	Bottulism	van Ermengem
1898	Dysentry bacillus	Shiga

1.2 The Report of the Committee on Physical Deterioration

The Committee on Physical Deterioration was summoned against a background of political and ideological debate that has been well documented (9). This debate reflected many anxieties which were often contradictory. Social investigators such as Charles Booth and B.S. Rowntree, and liberal reformers such as C.F.G. Masterman, were appalled by the overcrowding and filth they discovered in the slums and by the poor physique of children raised in these conditions. Masterman argued in his volume, HEART OF THE EMPIRE (10) that the British Empire had become sickly and degenerate at the core. At the same time, eugenicists and Social Darwinists such as Karl Pearson were concerned that the middle and upper classes were reproducing at a lower rate than the working classes and saw in this an inevitable formula for genetic decay (11). Their fears were compounded by the general decline in mortality which led them to believe that natural selection was becoming less severe (12). The absence of a similar decline in infant mortality caused anxiety in other quarters; despite medical advances infant death rates at the turn of the century were at levels which had remained unchanged since the 1850s (13).

The Boer War crisis of 1899-1901 caused the government to intervene in this debate. A military defeat, which would have had grave strategic consequences for the British Empire, had been only narrowly avoided and in the aftermath of the war, urgent questions were raised concerning Britain's future as a world power. The post-war debate was marked by a general agreement that if the 'decline and fall of the British Empire' was to be avoided, it would be necessary to re-invigorate the imperial race. Army recruiting statistics published in 1903 provided crucial evidence in this debate (14). They seemed to confirm the view of General Maurice, Director of Army recruiting, that

"no more than two out of five of the population below a certain standard of life are fit to bear arms" (15).

These allegations forced Balfour's Tory government to summon an inter-departmental committee to investigate the question of racial degeneration. The Committee on Physical Deterioration, which was convened in 1904 was instructed specifically :

"(1) To determine...the steps that should be taken to furnish the Government with periodical data for an accurate comparative estimate of the health and physique of the people;

(2) to indicate generally the cause of such physical deterioration as does exist in certain cases; and

(3) to point out the means by which it can be most effectually diminished" (16)

1.2.1. Witnesses to the Committee

Historians frequently refer to the general conclusions of the Committee, published in its "Report" (17). This has long been recognised as a landmark in welfare history; its recommendations led both to the feeding of poor children at public expense and to the establishment of the School Medical Service. These were measures which, as B.B. Gilbert observed in his study of the Origins of the Welfare State, challenged basic principals of Poor Law provision and set a precedent for later welfare state and national health services (18). However, quite apart from the Committee's recommendations, the evidence which is recorded in transcriptions of the examination of witnesses (19) is itself of major importance, although it has escaped the attention of scholars. This evidence consists of a vast collection of observations, facts, comments and opinions concerning the habits and domestic lives of the poor, which were described by witnesses as causes of 'physical degeneracy'. Obviously only a selectin of the evidence can be cited here, but every attempt has been made to make these quotations representative.

Members of the public health profession, voluntary workers, teachers and

school inspectors all identified the same range of problems when they were examined by the Committee. Its Report therefore reflects a consensus shared by both medical and non-medical observers, and by professionally qualified and lay workers alike; this represents a unique source of evidence concerning the aetiology of ill health at the turn of the century, which will be explored in succeeding sections of this study.

1.2.1.1. Professionals and the Poor: the growth of official contact

The public health profession had grown steadily during the second half of the 19th Century. Sanitary regulations required supervision and enforcement and from the 1860s local authorities were required by statute to undertake the inspection of their districts. In 1872 the appointment of Medical Officers of Health and of Inspectors of Nuisance was made obligatory (20) and changes in the medical curriculum during the 1880s brought improvements in the teaching of 'sanitary science'. Thus, by 1892 it was possible to introduce stricter regulations relating to the training and qualification of Medical Officers (21). One of the consequences of this process of professional expansion and development was the accumulation of knowledge concerning the domestic conditions of the poor.

During the last decades of the 19th Century, a similar expansion was taking place in the teaching profession. The introduction of compulsory elementary schooling in 1870 placed within a system of state education an entire class which had previously escaped official notice and at the same time produced a new body of professionals with intimate knowledge of the lives and domestic habits of the poor. These witnesses presented a view of the causes and cure for physical degeneracy which is remarkably similar to that presented by public health workers.

Finally, members of the growing band of health visitors and health

'missioners', who from the 1860s had been seeking to influence child rearing practices among the poor, were also questioned by the Committee (22). Evidence from these lay witnesses corroborated the views of the health and educational professionals.

The domestic lives of the slum dwellers appeared to these witnesses to be careless, slovenly and depraved. Thus, when they were asked why the 'lower strata' suffered from inferior physique, they argued that ignorance and degenerate habits rather than poverty or adverse conditions of life were responsible. They focused their analysis of the problem of physical deterioration (a term which was used interchangeably with physical degeneracy) on factors such as the working class diet; on the preparation of food and the regularity of mealtimes and on the alleged profligacy and laziness of the slum dwellers. The effect of this was to redirect the public health debate away from those issues which had dominated the great sanitary reform movement of the 19th century and to set an agenda for public health work which in many respects has remained unchanged to the present day. Subsequently, the behaviour of individuals- and especially of mothers - within the domestic environment, became one of the chief concerns of the public health movement.

The Committee did not deny the importance of the traditional public health issues . It referred in its Report to the particular problem of urbanisation with its

"attendant evils of uncleanness, foul air and bad sanitation"
(23)

and it saw a continuing need for municipal efforts to regulate and control water supply and sewerage. However, it reached the unanimous conclusion that the main causes of ill health and poor physique were to be found in the homes of the poor, and specifically that

"A striking consensus of opinion was elicited as to the effects of improper or insufficient food in determining physique ... this factor was acknowledged by every witness to be prominent among the causes to which degenerative tendencies might be assigned" (24).

The evidence which led the Committee to this conclusion provides an insight into relationships between food and health as they were understood at the turn of the century.

NOTE: In the discussion of physical degeneracy a number of terms were used interchangeably. These included 'physical degeneracy', 'poor nutrition', 'low physique' and 'poor physique'. Children were described as being 'badly nourished' or 'malnourished' or 'physically degenerate'.

1.2.2. Progressive Degeneration or Avoidable Ills?

The views which were presented to the Committee reflected a belief that physical deterioration was preventable and that if physical standards had indeed declined, as General Maurice and others argued, the process was nevertheless reversible. In 1904 this belief was still a matter of scientific dispute. The Committee on Physical Deterioration was convened at a time when a vociferous eugenic lobby argued that the racial stock was undergoing a process of progressive degeneration by the reversal of Darwinian natural selection because the weak, who in earlier ages would have been 'weeded out', were now allowed to survive and multiply (25). However, the Committee's final conclusions and recommendations represented an unqualified rejection of the eugenic case, a rejection which can be attributed to the strength of anti eugenic feeling within the public health movement.

Whilst the eugenic argument met with little support from the medical profession as a whole (26), it was complete anathema to public health practitioners. For example, Arthur Newsholme (a future Chief Medical Officer to the Local Government Board) argued in his extensive writings that physical degeneracy was the product of adverse environmental

conditions and that the main task of preventive medicine was to ensure that the developing child was given every assistance to grow to healthy maturity (27). During the early years of the century, Newsholme exercised considerable influence within the public health profession as editor of the JOURNAL OF HYGIENE and of PUBLIC HEALTH. His views were endorsed by all witnesses questioned by the Committee, for example, when D.J. Cunningham, Professor of Obstetrics and President of the Anthropometric Committee of the British Association was cross-examined by the Committee, he argued that inferior bodily characteristics resulting from

"poverty.... and which are therefore acquired during the lifetime of the individual, are not transmissible from one generation to another".

He cited the evidence of anthropologists in support of the view that there was a

"mean physical standard which is the inheritance of the people as a whole",

and asserted that in order to

"restore the classes in which this inferiority exists to the mean standard of national physique all that is required is to improve the conditions of living and in one or two generations the ground that has been lost will be recovered" (CPD Q.2210).

Dr Mackenzie, who was Medical Inspector to the Local Government Board of Scotland, claimed that the doctrine of the inheritability of character was now rejected by the 'majority of authorities' (CPD Q.6743) and Dr Eicholtz, a member of HM Inspectorate of Schools, maintained that

"the poorest and most ill-nurtured women bring forth as hale and strong looking children as those in the very best conditions" (CPD Q.556).

Whilst Eicholtz argued that the child

"fought strenuously for its own health at the expense of the mother" (CPD Q.556),

Mackenzie cited animal feeding experiments which suggested that in fact the foetus suffered rather than the mother. However, there was agreement over the central issue, namely that physical degeneracy among

the poor could be reduced by appropriate public health intervention and in its report, the Committee wrote of the 'encouragement' this view of physical degeneracy gave

"to working for the removal of the causes which are prejudicial to the health of each successive generation" (28).

Witnesses argued that only the poorest members of the slum population suffered from physical degeneracy, and that ignorance, intemperance and improvidence were the source of the problem. These vices led to bad domestic management, poor feeding and neglected children. The specific environmental insults to which children suffering from 'low physique and vitality' were subjected were described by Eichholtz as follows:

"[inadequate] feeding... bad clothing, bad boots, exposure, want of fresh air, overcrowding, filth, overstrain at work, and, to a less extent, smoking by boys" (CPD Q.475).

Of all these ills, a poor diet, which was synonymous with badly chosen or ill-prepared food, was identified by a majority of witnesses as the key factor. Thus, Eichholtz maintained that

"Food is at the base of all the evils of child degeneracy" (CPD Q.475)

and Dr Collie, a Medical Inspector for the London School Board, claimed that

"Malnutrition is the cause of most of the physical, and many of the mental, disabilities of children attending elementary schools" (CPD Q. 3926)

[It is clear from the context that by malnutrition Collie meant an incorrect or poor quality diet (CPD 3925-6).]

Statements such as these led the Committee report that there was a consensus over the damaging effect on physique of 'improper' or 'insufficient' food, which was

"prominent among the causes to which degenerative tendencies might be assigned" (29).

The Committee's short term solution was to feed those children whose

physique was being damaged by their mothers' indifference or incompetence. It recommended that the State should

"realize the necessity of ensuring adequate nourishment to children in attendance at school"

since it was

"the height of cruelty to subject half starved children to the process of education, besides being a short sighted policy" (30).

However, it went on to recommend that domestic education for women and girls, ignorant of the principals of correct diet, was the only long term cure for the nation's nutritional ills (31)*. This solution to what was perceived as a major cause of public ill health was not confined to the early years of the century but remained current throughout the entire period under discussion. The following section analyses evidence presented to the Committee concerning the relationship between diet and physical degeneracy. This evidence provides a description of the public health applications of nutrition science as they were understood in the first decade of the century. It illustrates the background of received ideas into which the 'Newer Knowledge of Nutrition' was launched and finally, it sheds light on the maternal inefficiency debate which runs through discussions of the cause and cure for poor nutrition fom 1900 to 1939 and beyond.

1.2.3. Defective Diets and Physical Deterioration

The Committee elicited from witnesses specific descriptions of the nutrition problem. The consensus was that:

- A poor quality diet, consisting largely of bread, tea and jam was common among the poor
- Home cooking skills had been lost.
- Rather than cook what was described as good, plain, wholesome food such as porridge and stew in the home, working class wives preferred to buy ready prepared meals such as fish and chips. It was believed that such food was not nourishing and that its use was a cause of physical

degeneracy.

-Ignorant infant feeding practice was seen to merge with improper child feeding practice. Both were condemned, on the basis of a comparison with middle class customs.

1.2.3.1. The 'Good Diet' and 'Good Dietary Practice': Medical Opinion

Definitions of a 'good diet' and 'good dietary practice' were provided by medical witnesses. This evidence can be illustrated by the testimony of Dr Hutchison, author of FOOD AND THE PRINCIPALS OF DIETETICS (32) who was nominated to give evidence by the Royal College of Physicians. Hutchison maintained that there was a broad agreement among physiologists as to protein and energy requirements. The average man, they were told, required

"3,500 units of energy for the upkeep of his body and 125 grammes of the chemical substance called proteid" (CPD Q.9957).

Hutchison stated that, according to dietary survey evidence, the diets of the labouring classes were

"rather deficient in energy [but] even more deficient in the amount of nitrogenous material [they] contain" (CPD Q.9958).

He maintained that

"Mere substitution of one meal a day of porridge and milk for bread and jam"

would bring the Edinburgh labourer's diet up to the 'physiological standard' (CPD Q.10028).

The importance of a correctly balanced diet was stressed by a number of medical witnesses. Experimental evidence which seemed to support the view that a 'balanced' diet was essential in nutrition was presented by Mackenzie, who cited work carried out in a Scottish poorhouse. This demonstrated that nitrogen waste was reduced when fat was added to the diet. According to modern nutritional views, these results show that prior to the change in diet, energy intake was insufficient for protein

metabolism (33). The opposite conclusion was drawn by Mackenzie, who interpreted them as proof that

"It is not so much the quantities of food as the proportion of the ingredients that make it up, that are important" (CPD Q.6815).

Dietary quality was also a question which concerned witnesses and protein was regarded as the most important nutrient in this respect. For example, Dr Niven, Medical Officer of Health for Manchester, stated that the proteins had a key role in nutrition since they were

"capable of repairing waste and of building up the body, which the other foods are not, except in so far as the salts are concerned" (CPD Q. 6516).

Hutchison's criticisms of working class diets and his recommendations for their improvement were shared by both medical and lay witnesses. He stated that

"If I were asked to state the chief fault of the diet of the working classes of this country I should say it is the excessive use of tea and bread" (CPD Q.9991)
and

"I should like to see less use of bread and jam and the substitution of porridge and milk, let us say" (CPD Q.9995)

He argued that these changes would increase the protein and fat content of the diet thereby 'improving' both its quality and its balance.

1.2.3.2. The 'Good Diet' and 'Good Dietary Practice': Non Medical Opinion

Non medical witnesses were also critical of working class diets. Mrs Bostock, who was a voluntary 'health missionary' in Glasgow, explained how she and her colleagues tried to encourage the use of porridge, soups and vegetable stews. Their particular preference was for

"the pot au feu of the French peasant- it is kept on the fire practically year in and year out" (CPD Q.7502).

They recommended for the children of the poor a modified version of the bland fare of the middle class nursery consisting of

"soup, skimmed milk and porridge" (CPD Q.7492).

A wider use of porridge and the adoption of French peasant cooking

methods were culinary ideals put forward by a number of witnesses. Their inappropriateness in British urban conditions escaped the notice of these observers of the poor. Maud Pember Reeves, author of ROUND ABOUT A POUND A WEEK commented on this phenomenon in her study of the daily lives of Lambeth women, written between 1909 and 1913. This includes a trenchant account of the activities of the charitable ladies who arrived bearing the

"gospel of porridge to the hard worked mothers and families of Lambeth" (34).

1.2.3.3. The definition of a 'Bad Diet'

Just as a 'good diet' meant plain home cooking - soups, stews and porridge- so a 'bad diet' included any form of ready cooked or 'convenience' food. The use of fried fish and chips was condemned as a matter of principle, since it was believed that this undermined the traditions of good home cooking. Witnesses repeatedly impressed upon the Committee the view that

"a knowledge of domestic cooking has gone out among the working classes" (CPD Q.7476)

Thus, Mrs Bostock complained that

"fried fish..and chipped potatoes .. are to be got very cheaply...It is very wrong of course" (ibid).

Mrs Bagot, who organised boys' clubs in London, also spoke of the lack of home cooking among the poor

"Few of them do much cooking; they get all their food at fried fish shops" (CPD Q.4554).

Miss Deverell, a junior inspector of schools at the Board of Education described a

"plentiful but unsuitable diet, taken at irregular intervals" (CPD Q.7985).

This 'unsuitable'(or 'incorrect') diet consisted of bread, jam and tea with occasional meals from fish and chip shops (CPDQ.7981-3) General Maurice, who had played a crucial role in arousing public concern over

standards of national physique (35), attributed the physical degeneracy which he observed among army recruits, to the use of 'improper food', rather than to a lack of food. He agreed with his questioners that this improper feeding was caused by the use of 'convenience' food.

"Mothers can get tinned food cheap and there is no trouble in cooking it and rather than prepare proper food for the child she uses this stuff" (CPD Q.332).

Maurice cited the use of

"raw herrings, pickles, fried fish and the like"

as examples of the bad child feeding which he believed resulted in physical degeneracy. (CPD Q.275) HM Inspector of Factories and Workshops, H.J. Wilson, described the poor physical condition of children in Dundee employed as half time mill workers, and attributed their stunted growth, pallor and wasted muscles to diets consisting largely of bread and strong tea taken without milk (CPD Q.1926).

Similarly Dr T.F. Young, President of the Association of Certifying Factory Surgeons, blamed cheap jam and white bread for the poor condition of children in Belfast and Liverpool. Many witnesses believed that the virtues and skills of home cooking which were absent in the towns could still be found in the countryside. Their loss was seen as a symptom of the corrupting influences of urban life. According to Dr Young

"In former times the children used to live on oatmeal and butter milk and potatoes, and the country children still live on that, but the town children live more on tea and this white bread and jam" (CPD Q.2093)

1.2.4. The Cause of the Nutrition Problem

Medical and non Medical observers agreed about the cause, as well as the nature, of the nutrition problem. Dr Collie attributed the poor condition of recruits to bad feeding. This he maintained, was caused by the fact that the 'lower strata' of the working class population were

"very improvident; they are extravagant; they could live very much more cheaply" (CPD Q.4042)

Every witness who was cross examined on this subject appeared to share Collie's view that

"The average working man's wife is lamentably ignorant of the value of the different foodstuffs and her unintelligent selection, and bad cooking, amount practically to underfeeding, even though the bulk to all appearances be sufficient" (CPD Q.3976).

Describing conditions in the homes of the London unemployed and casual labourers, Dr Eichholtz spoke of

"the laziness of the women. They do not get up to make the breakfast and when this is the case the children come to school without it. The laziness of the women, coupled with drink, is at the root of many of the evils of degeneracy." (CPDQ 436)

He went on to criticise the feeding of children in such families.

"First..there is the want of food...Then there is the irregularity in the way in which they get their meals...Then non-suitability of the food when they get it is a third factor" (CPD Q.436).

When Dr Niven was asked whether the poor diets he spoke of were the result of lack of money or improvident spending, he claimed that these diets were the product of both ignorance and intemperance, although he maintained that

"ignorance of the best diet [was a] complicated matter" (CPD Q.6291).

Dr Hutchison agreed with the view that defects in the diets of the poorer classes were caused by incorrect selection of food rather than by an inadequate food intake (CPD Q.9965).

Lay witnesses shared this view, and presented a spirited attack on the profligacy and declining standards of working class women. There was, the Committee noted in its report, universal agreement over the importance of 'bad' feeding as a cause of physical deterioration. 'Bad' feeding was synonymous with 'incorrect' feeding, or an 'improper diet' - the terms were used interchangeably (CPD Q.332).

The outstanding feature of the 'incorrect diet' consumed by the poor was described as the use of ready prepared or 'bought in' foods. The use of these food was believed to be symptomatic of the laziness and moral degeneracy of the urban working classes. For example Mrs Bostock, the Glasgow 'health missionary', declared that

"We tried to encourage porridge but they would not have it. It is too much trouble" (CPD Q.7409).

She asserted that cook shops, which sold ready prepared meals, were used by the "very ignorant" not because they were cheaper, but to "save themselves the trouble" (CPD Q.7542). Mrs Close, an expert on 'rural conditions' spoke of the

"ignorance and idleness and want of sense of duty of British mothers" (CPD Q.2635)

"The last thing they think of is duty and, therefore, they do not take the trouble to cook or get up in the morning and the children go to school without breakfast" (CPD Q.2558)

and Charles Booth, the author of the LIFE AND LABOUR OF THE PEOPLE OF LONDON (36) claimed that working class women

"do tend to neglect their homes more" (CPD Q.1087).

Finally, Mrs Lyttleton, another 'expert' on rural conditions, argued that

"The extraordinary facility of getting all kinds of food without much trouble in cooking has produced a habit of laziness among girls" (CPD Q.5458)

and that

"many modern tendencies need counteracting" (CPD Q.5498).

Thus the Committee wrote in its report that a major cause of physical degeneracy was the

"incurable distaste for the obligations of domestic life" (37) among working class housewives. The Committee accepted the view that working class women used prepared food, not because they were hard pressed and lacked adequate cooking facilities at home but because they were lazy.

The Committee was concerned to establish whether an inadequate diet was

caused by ignorance, neglect or poverty (CPD Q.11960). Sir John Gorst, MP, who was a prominent advocate of the provision of school meals by the State, agreed with other witnesses that where there was a problem of insufficient food this was due to the

"general ignorance of the women of the lower classes" (CPD Q.11962).

He asserted that

"Nothing is more deplorable than the impotence of the general English labourer or labourer's wife in the presence of food. Where a Frenchwoman would make an excellent dinner an Englishwoman would starve" (ibid).

Furthermore, although the Englishwoman spent a great deal more on feeding her family,

"the Frenchwoman gives them a great deal more food" (CPD Q.11963)

Similarly, B S Rowntree, author of POVERTY, A STUDY IN TOWN LIFE (38), maintained that

"A great number of children who are born into the world are affected because the mothers do not understand the choice of food for them or how to cook it [bad nutrition] is ignorance" (CPD Q.5315).

Just as there was a consensus of opinion about the cause of poor nutrition, there was also agreement about its cure. This was summarised by Mrs Lyttleton who claimed that

"The real remedy is an increase in intelligence throughout the country" (CPD Q.5521)

and that the problem of poor nutrition could only be solved by

"proper education and care (rather than giving free food) " (CPD Q.5459).

It was claimed that the remedy for poor feeding entailed not only a change in food habits but also the adoption of middle class behaviour. Thus Mr Libby, founder of the London 'Referee' Dinner Fund maintained that

"Bad nutrition arises in this way, that the mother of the family cooks for the father, she does not cook for the children, and the children all have to eat what the father eats" (CPD Q. 7891).

This view of 'wrong' eating was clearly based on a comparison with the organisation of the Victorian household, where nursery life and nursery food was clearly demarcated from that of the adult world. The notion that all members of the household should share the same food after infancy was alien to the middle class observers of the poor. Just as a diet of skimmed milk, soup and porridge, which was simply a cheaper version of middle class nursery food, was recommended by Mrs Bostock, so Miss Deverell deplored the fact that 'anything like a sit down meal' was unknown in many districts (CPD Q 7985). According to this view there was a need to teach the working classes not only what to eat and how to cook it, but how to organise the ritual of eating as well.

1.2.4.1. Domestic education as a cure for the nutrition problem

In its Report the Committee stated that the solution to the problem of poor nutrition - and therefore to the problem of physical degeneracy - required the education of working class girls in cooking, child rearing and household management (39). For example, Dr Niven argued that domestic education, including instruction in the basic principles of the science of nutrition should form the basis of the curriculum for girls in their final year at school. By this stage teaching subjects such as literature was, he maintained, a waste of time (CPD Q6526-8).

Lessons on the chemical constituents of food were also suggested by Mrs Smyth, whose particular interest was in the supply of milk to towns. Her argument that

"a woman may spend a heap of money on unsuitable food" (CPD Q.1290)

remained central in discussions about the cause of malnutrition throughout the inter-war period when it re-emerged under the guise of the Newer Knowledge of Nutrition.

1.2.5. The Nature of the Nutrition Problem

Although the State did not supply funds for school feeding until 1906 when the Education (Provision of Meals) Act was passed, poor children had been fed by charitable organisations throughout the 19th Century . This activity increased greatly after the introduction of compulsory elementary education in 1870 which, according to the School Board Chronicle of 1884, resulted in the

"bringing to light [of] the distress, destitution and underfeeding which formerly had escaped ... notice" (40).

By the turn of the Century voluntary feeding schemes, whose aim was to enable 'half starved' children to take advantage of the education offered by the State, were operating in London and many of the industrial cities and in 1905 an Interdepartmental Committee was convened both to investigate these schemes and also to enquire into existing arrangements for medical examination. As with the Committee on Physical Deterioration, the 'Report' of this Committee (41), has received greater attention than the Minutes of Evidence (42), although these Minutes provide further insights into perceptions of the nutrition problem at the turn of the Century. This section is based on evidence presented to the Committees of 1904 and 1905 and focusses on two issues which remained controversial during the inter war period: the criteria according to which children were selected for feeding, and the distinctions made between underfeeding and improper feeding as causes of poor physique. Contemporary estimates of the extent of the nutrition problem presented by various expert witnesses are also set out.

The Selection of Children for Feeding

According to data gathered by the Committee on the Medical Inspection and Feeding of School Children, the selection of children for feeding was generally carried out by teachers. Their reasons for selecting children included the following criteria (43):

1. Known destitution. It was assumed that in cases of extreme poverty children must necessarily be underfed.
2. Clinical signs. Children were recommended for feeding when they looked thin, pale and generally ill-nourished.
3. Self selection. Meals were sometimes offered on a deterrent principal. They were made so unappealing that it was believed only the half starved would take them up.
4. Cost of living criteria. In Manchester a systematic attempt was made to feed children according to a scale of needs based on Rowntree's calculations, which included minimum food requirements derived from Atwater's (44) standard. The use of 'scientific' cost of living estimates as a test for malnutrition received little support at this time but was widely canvassed during the 1930s.

In its summary of this evidence, the Committee acknowledged that the selection of children for feeding was problematic.

"We do not disguise from ourselves that this matter of the selection of children is replete with every kind of difficulty" (45).

However, it recommended that meals should be made available primarily for those children whose parents had temporarily and unavoidably fallen upon hard times (46). It was thought to be unwise to 'reward' families suffering from long term destitution since this might act as a disincentive to work; the actual condition of the child was considered to be less important than fostering the work ethic.

The Committee noted that medical opinion had not, to date, been sought in the selection of children.

"The question of malnutrition and underfeeding has attracted very little attention in connection with medical inspection" (47).

However, the prevailing view was that medical advice could do little to better the judgement of the conscientious teacher, who was generally aware of changes in family circumstances and could monitor the child's

behaviour and physical condition. The medical view was less comprehensive. For example, in his evidence to the Committee on Physical Deterioration, Sir John Gorst, a leading Parliamentary advocate of a system of school feeding, stated

"There is no definite test of an underfed child. It is only the opinion of the headmaster, or the class teacher as to what is an underfed child" (CPD Q.11991).

Gorst did not believe that medical diagnosis was a necessary element in the process of selecting children for feeding.

Although malnutrition was seen as a social and economic problem by the teaching profession, the Board of Education was increasingly anxious that it should be defined as a medical problem. This issue became highly controversial in the early 1930s when the adequacy of income was viewed by many nutrition scientists and welfare campaigners as the best test for malnutrition; it is discussed fully in Chapter 4.

1.2.5.1. Malnutrition: improper feeding or underfeeding?

In evidence presented to both the Committee on Physical Deterioration and to the Committee on the Medical Inspection and Feeding of School Children children were variously described as being 'malnourished', 'improperly fed' and 'under fed'.

The Committee on the Medical Inspection and Feeding of School Children questioned witnesses on their understanding of these terms which implied that some children were badly nourished owing to the poor quality of their diets whilst others did not get enough to eat. This suggested a need for different forms of remedial action and once again the debate foreshadows discussions which took place during the inter war period. Some witnesses were clearly confused by these distinctions (for example Mrs Grant, (MIFS Q.6173) and Mr le Shovelier (MIFS Q. 5182)) and denied that children might be 'underfed', due to an 'unsuitable' diet. However,

these witnesses believed that all children who were 'improperly' fed at home would benefit from 'wholesome' meals at school. Others, such as T.E. Harvey, who was deputy warden of Toynbee Hall and a member of the London County Council (LCC) Joint Committee on Underfed Children, grasped the distinction between qualitative and quantitative deficiency. Harvey argued that underfeeding might be caused either by insufficient food or by a poor quality diet and that these conditions called for different forms of remedial action. He believed that all school children should receive half a pint of milk per day, since this would help the 'improperly fed' (MIFS Q.1706), and that more substantial meals should be available to the underfed.

Harvey distinguished between 'malnutrition', caused by bad home conditions and unsuitable food, and under feeding where there was an insufficiency of food (MIFS Q.1769-1771); his views were echoed by many Medical Officers commenting on the nature of the nutrition problem during the 1920s, whose opinions are discussed in Chapter IV. These fine distinctions were condemned by Dr Macnamara, a Member of Parliament who had previously been headmaster of a London elementary school. MacNamara insisted in his evidence to the Committee on Physical Deterioration that any child who was not properly fed was 'underfed' (CPD Q.12454). There was no need, he argued, to distinguish between insufficient, improper and underfeeding. Moreover, he claimed that malnourished children could easily be recognised without seeking medical advice

"the pasty, worn and wan look and pinched appearance of a child is eloquent testimony to his condition" (CPD Q.12422).

Dr Hall of Leeds, who gave evidence to the Committee on the Medical Inspection and Feeding of School Children, was equally emphatic in the definition of the nutrition problem he encountered and also maintained that there was no difficulty in identifying an underfed child

"You have the ill-nourished skin, the ill nourished hair, the stunted growth, the light weight" (MIFS Q.5646).

In his view, the nutrition problem was

"a question of underfeeding" (MIFS Q.5649).

The Committee on the Medical Inspection and Feeding of School Children did not make any recommendations for the future definition of terms in its Report. However, the view that improper feeding (or a 'poor quality diet') amounted to 'underfeeding' re emerged in the inter war period when the assessment of dietary quality was based on the discoveries of the 'newer knowledge of nutrition'.

1.2.6. The Extent of the Nutrition Problem

Both Committees asked witnesses to estimate the extent of the problem of 'poor feeding'. (This term was used interchangeably with 'underfeeding', 'improper feeding' and 'malnutrition'). The estimates which were produced are marked by wide discrepancies; since malnutrition was as yet ill defined, the criteria by which children were assessed as being poorly fed were highly subjective and frequently idiosyncratic.

Estimates of the extent of underfeeding were based both on clinical assessments and on estimates of the prevalence of extreme poverty.

Dr Eichholtz, HM Inspector of Schools, offered a clinical description of the children he classified as underfed:

"They suffer from every physical symptom of anaemia, with pale faces and lustreless eyes..A good many children suffer from blight in the eyes and sore eyelids. The hair is badly nourished and wispy and the skin is rough, dry, pale and shrivelled, giving a very old look very early in life" (CPD Q.437).

On this basis he estimated that, in the poorer areas of London, 25% to 30% of children were underfed and that the figure for London as a whole was around 16% In the very worst schools, at bad times of the year, up to 90% of children might be underfed (CPD Q.435-7). Dr Collie adopted similar criteria for his definition of improperly fed children:

"They are pale - anaemic. They have a half starved look and have very little resistance to disease, especially zymotic disease..children who survive the first three years of their lives are to be found..suffering from tubercular affections of the hip and other joints, from spinal and other disease" (CPD Q.3933).

His prevalence rates for improperly fed children were similar to those of Eichholtz.

The London School Board presented the far lower estimate of around 10% of children as being improperly fed. This was dismissed by Eichholtz on the grounds that it was based on numbers of meals provided rather than on the number of individual children fed or on a clinical examination.

In contrast to the London figures, Dr Airy estimated that in Birmingham only 2,000 to 2,500 children were 'requiring food'. This amounted to approximately 5% of the elementary school population. However, his figures included only those children whom he defined as 'starving'. He drew a distinction between children who failed to get 'ample ' food and those who were 'starving'(CPD Q.13284) . A starving child would accept the unappetising bread and lentil soup provided. A child who was merely underfed would not (CPD Q.13300). Estimates of the extent of the problem in Scotland, provided by Dr MacKenzie are far closer to those of Eichholtz and Collie. In the extremely poor Cannongate district of Edinburgh, he estimated that 38% of children were 'half starved' and that the figure for the city as a whole was around 30% (CPD Q.6977). Sir John Gorst, MP, claimed that one third of the elementary school population was 'insufficiently or improperly fed' (CPD Q 11841) and Dr MacNamara, MP, was 'sure ' that 10% to 15% of children in the industrial cities were underfed (CPD Q.12450). Mr Libby estimated that 25% to 30% of children in London's poorer schools were in need of feeding (CPD Q.7835). Finally, a Salford clergyman thought that only 2% of children in this extremely poor town town were malnourished (CPD Q.4384).

Table 1.2

ESTIMATES OF THE PREVALENCE OF UNDERFEEDING, 1904*

WITNESS	TOWN	% MALNOURSHED
Macnamara (CPD Q.12374)	London	20%
Eichholtz (CPD Q.436-443)	London	16%
"	Manchester	40%-60%
"	Salford	10%
"	Leeds	25%
Clergyman (CPD Q.4384)	Salford	2%
Libby (CPD Q.7835-7836)	London	12%-30%
Gorst (CPD Q.11841)	UK	33%
Mackenzie (CPD Q.6939)	Edinburgh	38%-30%
	Aberdeen	9%
Airy (CPD Q.13246)	Birmingham	5%

Source: Evidence presented to the Committee on Physical Deterioration

Table 1.3

ANTHROPOMETRIC EVIDENCE OF UNDERNUTRIION AMONG GLASGOW ELEMENTARY SCHOOL CHILDREN AND ENTRANTS TO LCC POOR LAW HOMES AGED 8-9 YEARS, 1906-1912

	(number)	Stunted %	Low weight age %
Glasgow boys (a)	(4009)	64	27
LCC boys (b)	(43)	79	35
Glasgow girls (a)	(3817)	57	24
LCC girls (b)	(37)	57	14

A full discussion of anthropometric methods and an analysis of anthropometric survey data appears in Chapter 6. The purpose of this Table is simply to give an indication of the possible extent of the problem of underfeeding at the beginning of the period of study. The threshold for stunting is the 3rd centile of Height for Age as defined by NCHS, 1983 (51). The threshold for low weight-age is the 3rd centile of Weight for Age as defined by NCHS, 1983. The prevalence of wasting (weight for height below the 3rd centile of the NCHS standard) was too low for meaningful analysis. 2 children from the LCC data set and 46 boys and 16 girls from the Glasgow data set fell within this category.

These data were recalculated by this author from raw data presented in reports a: Elderton, 1914 (52); b: Unpublished LCC Poor Law Registers.

* NOTE: a recommendation which gave rise, inter alia, to the establishment of the Department of Household and Social Science of King's College, London.

CHAPTER 2

THE NEWER KNOWLEDGE OF NUTRITION

2.1. The implications of the 'Newer Knowledge'

In the same period as the traditional preoccupations of the Victorian sanitary movement were being dropped and replaced by a new emphasis on problems of individual health, domestic hygiene and working class diet, developments were taking place in nutrition science which, in the inter war years, introduced an exclusively nutritional explanation for major public health problems. This was known throughout the 1920s and 1930s as the "Newer Knowledge of Nutrition". The Newer Knowledge complemented the public health priorities set out by the Committee on Physical Deterioration in some respects and challenged them in others. Although the Newer Knowledge denied that improvements in domestic hygiene would produce the physiological improvements which the public health movement claimed, it reinforced the view that a badly chosen and ill prepared diet was a major cause of public health problems. Thus, the Newer Knowledge dovetailed with existing public health concerns and fuelled interest in the importance of dietary quality in the aetiology of ill health.

The origins of the Newer Knowledge of Nutrition are generally seen in the accumulation of evidence from clinical observations and from animal feeding experiments involving the omission of various substances from the diet, which led to the discovery of vitamin deficiency disease. However, the newer knowledge also introduced the conceptually distinct belief that the recently discovered micronutrients were involved in states of marginal deficiency which produced non specific 'sub-optimal' health; and ultimately, it led to the notion that by perfecting the organism's internal or 'chemical' environment new levels of 'super health' and enhanced vitality could be achieved (1). These distinctions are not generally recognised, although Hopkins drew attention to them in

the MRC's first publication on the subject of the vitamins, the 'Report on the Present state of Knowledge concerning Accessory Food Factors' (2), which appeared in 1919.

2.2. The Vitamin Hypothesis and Deficiency Disease

The experimental and clinical evidence from which the vitamin hypothesis was derived is well documented and need only be mentioned briefly here. The prolonged period during which dietary deficiency was not recognised as a cause of disease has occupied the attention of both nutrition scientists and historians of science for many years. This has resulted in a very thorough appraisal of work which might have given rise to the general acceptance of the deficiency theory of disease at an earlier date but failed to do so (3). It is widely recognised that, from the last decade of the 19th century, observations were being made which seemed to challenge not only accepted theories concerning the essential components of diet, but the very nature of disease and its aetiology. These observations concerned the possibility that disease could be caused by dietary deficiency as well as by the invasion of the organism by an external pathogen. The idea of deficiency disease had been postulated during the 18th Century by the naval hygienists Blane and Lind, who produced experimental evidence which demonstrated that scurvy could be induced by the omission of known "anti scorbutic remedies" from the diet (4).

In the course of the 19th Century, further experimental and clinical evidence which demonstrated the role of diet in the aetiology of various diseases accumulated. For example, the French clinician Trousseau showed in 1849 that butter and cod liver oil both had anti rachitic qualities, and that those of cod liver oil were far greater than those of butter (5). In 1881 Lunin published the results of a series of experiments which compared the effects on growth of milk and purified

diets (6). However, the experimental use of purified diets was not taken up again until 1905 when Pekelharing adopted it to investigate Eijkman's observations on polyneuritis (7). His results led Pekelharing to propose the existence of hitherto unrecognised accessory nutritional elements. This same hypothesis was independently suggested by Hopkins in 1906 when he postulated the existence of "minimal qualitative factors" (8), which he later termed "accessory food factors". In 1912, Hopkins published results which demonstrated that, in order to remain in good health, the organism (ie the laboratory rodent) required substances in the diet other than the previously accepted basic nutrients (9). Shortly before this, Funk announced the isolation of the chemical substance that appeared to possess the anti neuritic vitamin and at the same time proposed the hypothesis that beri beri, scurvy, pellagra and possibly rickets were caused by

"special substances which are of the nature of organic bases, which we will call vitamins" (10).

The emergence of the dietary deficiency theory of disease coincided with a period of major discoveries in the field of microbiology. Between 1880 and 1910 pathogenic organisms were identified which were responsible for many of the most common fatal diseases of Western Europe (11). Since the first deficiency diseases to be identified, beri beri and scurvy, were not major public health problems in the UK, the USA or Europe, it is hardly surprising that attention remained focussed on the germ theory of disease. In these circumstances, the climate of opinion was not receptive to an untested theory which attributed primary importance in the disease process to the absence of a nutrient rather than the presence of a pathogenic micro organism. The conceptual difficulties presented by the vitamin hypothesis were confronted directly by Hopkins in his Huxley Lecture of 1920. The new science of nutrition, based on the assertion that disease could be attributed to a

'negative cause' was met in many quarters by what he described as

"the obsession that some positive agent -some intruder so to speak-must always dominate aetiology" (12).

Yet despite this initial indifference and even hostility, the theory of micronutrient deficiency was soon accepted by the medical profession as being not merely correct, but also of major public health importance. A growing body of evidence which suggested that contemporary descriptions of nutritional need were hopelessly inadequate, combined with the dramatic results of animal feeding trials, were responsible for this change of view.

2.3. Nutrition, Vitamins and Growth

At the turn of the Century two of the axioms of 19th Century nutrition - that nutritional need was dominated by protein and that protein, carbohydrate, fat and ash were the only compounds involved in nutrition - were already crumbling. The view that nutritional need was dominated by protein, championed by the German school, (whose most notable exponents included Voit and Rubner) was criticised by Chittenden, who showed in a series of experiments published in 1904 (13), that students, athletes and University professors all thrived for long periods on a diet containing what was then seen as absurdly low levels of protein. By suggesting that the protein requirement was in fact much lower than the estimates of the German school, Chittenden implicitly undermined the basis for such notions of dietary quality as already existed. However, neither he nor his critics took note of this inference.

In the UK, the existing view of the range of compounds necessary for adequate nutrition came under attack from Gowland Hopkins. Hopkins inferred from his work on protein biochemistry that, in addition to the known nutrients, many unknown substances were also essential to support life (14). By the late 1890s it was recognised that proteins differed in

amino acid composition (15). However, differences in protein quality remained unexplored until 1906 when Willcock and Hopkins showed that the level of the amino acid tryptophan determined the quality of zein (16). Using purified rations they discovered that rats could survive for only 14 days on a diet of carbohydrate, fat inorganic salts and zein, but for 28 days when a small amount of tryptophan was added to the diet. This was the earliest animal experiment in which the indispensability of a specific amino acid was demonstrated (17) and it showed that dietary quality was a micronutrient effect - the paradigm which, it could be argued, launched the newer knowledge of nutrition. In the same year as these results appeared, Hopkins argued that there were many unknown "minimal qualitative factors in the diet", which were as essential as the proteins, carbohydrates and fats, and that these "unknown dietetic factors" operated in the aetiology of scurvy and rickets (18). However, he went still further than this and suggested that 'nutritive errors' were also responsible for many disorders which did not constitute recognised disease syndromes, but were nevertheless debilitating to the individual. Thus, by 1906 Hopkins already held the view that the 'accessory food factors' were necessary to promote 'optimal health' as well as to prevent frank deficiency disease.

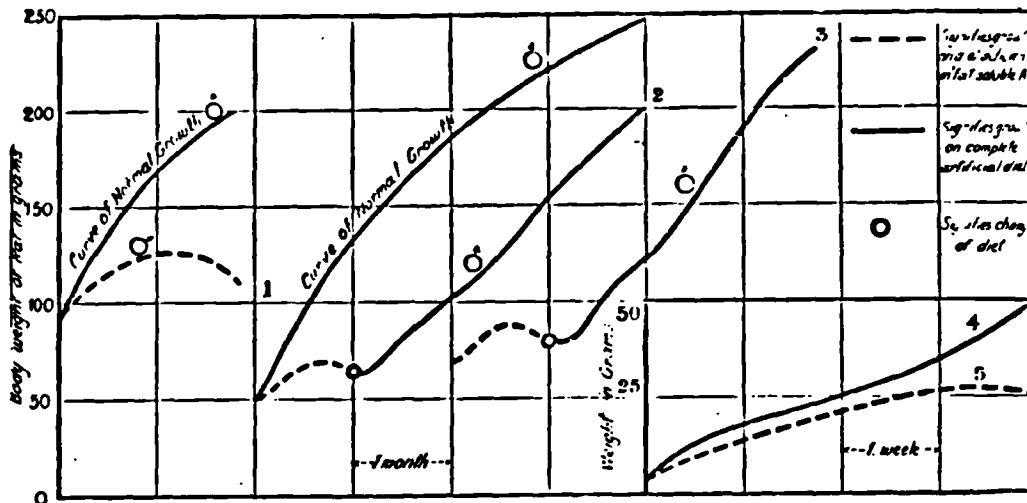
In 1912 he produced experimental evidence which seemed to support this case (19). Hopkins's 1912 results showed that young rats fed an artificial ration containing purified casein, starch, cane sugar lard and inorganic salts failed to grow - although he estimated that they were apparently consuming a more than sufficient quantity of food to maintain normal growth - and that growth was only resumed when a small quantity (3cc) of milk was added to the diet. Thus, in his discussion of results Hopkins hypothesised the existence of a 'catalytic or stimulative' substance in milk and tissue extracts which was required to maintain growth; subsequently, growth itself was used as a

measure of health. During the period 1913-1918, a great many feeding experiments were carried out which extended Hopkins's work* and identified the main food sources of the vitamins. Using the same protocol, which involved adding small quantities of natural foods such as butter, yeast and raw vegetables to a purified artificial diet, a mass of evidence was produced which demonstrated the dramatic effects of making minor adjustments in the diets of laboratory animals (21). The growth curves in Figures 2.1a and 2.1b show Hopkins's famous results published in 1912 (22) and those in Figures 2.2a and 2.2b show results published by Drummond in 1917 (23); these record the growth of rats fed vitamin B deficient diets and vitamin A deficient diets. Summarising this work in the MRC's Vitamin Report of 1919, Hopkins maintained that it provided evidence that "at least two substances of the vitamin type are concerned with growth" (24). He described these substances as 'probably'- rather than necessarily -identical with the anti neuritic, the anti rachitic and anti scorbutic vitamins. The distinction is significant though inevitable; with the exception of scurvy in the guinea pig, it proved exceptionally difficult to induce human diseases in animals fed artificial diets.

Although human diseases were not established in the early animal experiments, the animal model did produce an idea of the sensitivity of growth to vitamin deficiencies and therefore to health; thus, by the early 1920s scientists were arguing that the newer knowledge of nutrition had major implications for preventive medicine. However, these claims were based on a misinterpretation by Hopkins of the cause of growth failure. Neither Hopkins nor subsequent researchers measured the food intake of their animals - the belief that growth failure was due to a lack of qualitative factors in the diet made this unnecessary- and although Hopkins 1912 results are difficult to replicate they

Figure 2.2a

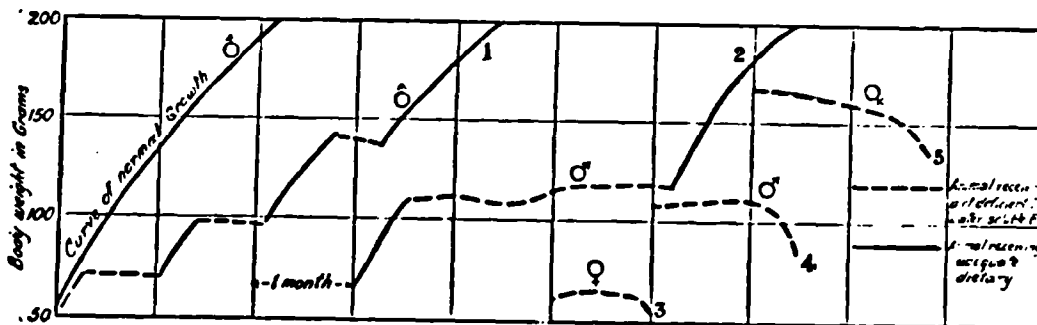
Growth of rats fed vitamin B and vitamin A deficient rats



Showing preliminary growth and eventual decline on diet deficient in Fat-soluble A. Also recovery on addition of a source of Fat-soluble A.

These curves illustrate how the absence of the fat-soluble accessory substance ultimately inhibits the growth processes of young animals. The broken curves represent the changes in body weight of rats fed upon a diet freed as far as possible from all traces of that accessory factor. The small amount of growth, which usually occurs at first, probably indicates the utilization of reserve stores of the indispensable unit, but failure ultimately sets in. At the points marked on the curves by (o) the deficiency of the fat-soluble was made good by the addition of butter fat. This change was at once followed by a resumption of growth. In the small figure are given the weight curves of young rats born from females receiving artificial diets of this nature. Curve 4 indicates the normal growth of young rats nursed by mothers receiving an adequate artificial ration similar to that used in the experiments illustrated in Figure 3. Curve 5 shows the development of young nursed by females receiving a dietary deficient in the fat-soluble factor. These two curves represent the average of a considerable number. The young nursed by the females on the inadequate ration were for some time not much below the normal standard as regards body weight, although they were at all times markedly undernourished.

Figure 2.2b



Showing failure to grow on diet deficient in water-soluble 'B', and recovery on adding that factor.

These curves illustrate how a deficiency of the water-soluble growth factor immediately causes a cessation of growth, and show the equally prompt resumption when an extract containing the active substance is added to the dietary of the experimental animal.

Curves 3, 4, and 5 demonstrate that the more mature the animal, the longer can it survive against the deleterious effects of a deficiency of water-soluble A.

Source: MRC Special Report Series No.38 (20).

almost certainly reflect a reduced food intake in the rats fed the purified, deficient diet, rather than a lack of a 'catalytic, qualitative growth factor'. This assertion can be made with confidence since growth is an energy requiring process and in order NOT to grow animals eating normal amounts of energy must somehow dissipate this energy- which they can only do by increasing their metabolic rate. However, no such increase in metabolic rate is associated with Vitamin deficiency (with the exception of Essential Fatty Acid (EFA) deficiency, (25) and EFAs were available in the artificial ration). [Sodium deficiency (26) and under some circumstances protein deficiency (27) can also increase metabolic rate.]

The reason for loss of appetite in animals fed purified artificial rations and recovery on the addition of milk or other 'natural' foods, is obscure. Increased palatability or possibly a physiological response to the deficient diet may have been responsible. However a deficiency in total food intake, rather than Vitamin intake was unquestionably the cause of growth inhibition. A degree of anorexia induced by feeding rats a deficient diet is still a common finding in laboratory experiments. The onset of the anorexia is rapid - it is often detectable in the first day of the experiment.

Subsequent research, including that of Harriet Chick and Elsie Widdowson acknowledged the problem of food intake variations but their attempts to allow for this again produced confusing results. In 1942, Chick published results which showed that rats fed brown bread grew faster than those fed white bread. (28). These rats also ate more food, but Chick showed that their weight gain per gram of food or protein consumed was higher - and therefore, presumably that brown bread, with its higher micronutrient content, was better than white bread. Widdowson and McCance investigated this question further (29) and Widdowson recently

wrote of Chick's findings

"the results showed clearly that the increase in weight per gram of protein eaten was greatest for the wholemeal flour. Something in the wholemeal flour made it a better food than white flour for weanling rats." (30)

However, this assertion is fallacious, and is based on a simple arithmetical misunderstanding. Animals have a basic daily maintenance requirement of protein and energy which is needed simply to avoid losing weight and growth can only occur when intake is above this level. A consequence of this is that the more that is consumed, the greater is the gain per unit of consumption. In the terms of classic animal nutrition, the gross efficiency of the diet rises with intake, even though the net efficiency remains constant, because the importance of the maintenance quotient is reduced as intake rises. Thus if 30g of protein are needed to maintain weight and gain is 0.5g per gram of protein consumed above this (ie the Net Protein Ratio, $NPR=0.5$), then it is clear that weight gain per gram of protein (the Protein Efficiency Ratio, PER) increases with intake. PER is zero when protein intake is 30g; 0.125 when protein intake is 40g; 0.25 when protein intake is 60g; and 0.35 when it is 100g. Chick's results can be explained by such an effect. The failure to recognise this is a reflection of the schism between biochemical nutrition and the nutrition which preceded the advent of the 'Newer Knowledge' and was concerned chiefly with animal production.

The data shown in the first four columns of Table 2.1 are taken from Chick's experiments.

The data shown in Table 2.1a are taken from Chick's experiment

Table 2.1

Extraction rate of flour % (a)	Protein intake (g) (a)	Wt gain (g) (a)	Growth promoting value of Protein (a) (b)	Protein intake above maintenance (c)	Net Protein Ratio (c)(d)
100	39.9	71	1.77	3.9	3.0
85	34.5	57	1.67	18.5	3.1
75	30.9	45	1.48	14.9	3.0

(a) Data of Chick as cited by Widdowson, 1975.

(b) Equivalent to PER.

(c) Recalculated by present author assuming a maintenance protein requirement of 16g.

(d) Net protein ratio is weight gain/protein intake above maintenance.

In the last two columns of Table 2.1 these results have been re-analysed by the present author to demonstrate that NPR was unchanged and that all effects on growth were merely due to differences in protein intake. Thus, in contrast to the claims of Chick and Widdowson there is no evidence for a difference in the nutritional value of protein at different extraction rates. The apparent effects being a reflection of variation in food intake.

Widdowson's claim that the white flour had a smaller 'growth promoting' value in its protein is an error resulting from the effect on PER of lower protein intake. In fact, the three data points suggest that growth rose linearly with increasing protein intake and that an intake of 16g protein would result in no growth.

During the inter-war period, inferences for human diets continued to be drawn from animal feeding experiments. For example, during the 1920s McCarrison, head of Nutrition research in India, compared the effects of feeding rats a typical Sikh diet, with the effects of feeding a poor European diet (31). Rats fed the Sikh diet, which consisted of whole wheat, butter, milk, legumes, raw vegetables and meat, grew well. Those fed the poor European diet of white bread, margarine, tinned meat,

boiled vegetables, tinned jam, tea and sugar with a little milk, failed to thrive. The dramatic contrasts in growth are shown in Figure 2.3. The inference drawn from this experiment, in which food intakes were not measured, was that differences in growth were caused by qualitative differences in the diet.

It is possible that this was in fact the case, but not for the reasons McCarrison believed. McCarrison's 'poor European' diet was lower in protein than the Sikh diet. The differences would be immaterial to the slow growing human species which needs a low concentration of protein for even maximal growth. However, the rat grows much faster than the human relative to its size, and therefore requires a higher concentration of protein in its diet. The rats fed McCarrison's 'Sikh' diet would have received this higher concentration of protein.

In the same way, McCarrison compared what he saw as similarities in the effects of diet on the growth of rats and on the body size of members of various Indian ethnic groups, when fed the same diet (See Figure 2.4).

Boyd Orr repeated these experiments using as his model poor Peterhead diets and diets to which small quantities of milk and green vegetables had been added (32). Once again, the animals thrived only when the milk and vegetable supplements were added, which seemed to prove that the diets of certain sections of the British working classes would not even keep a rat alive. Boyd Orr's results cannot be explained by the effect discussed above, but they may reflect an increased intake of the diets containing milk and green vegetables.

Figure 2.3

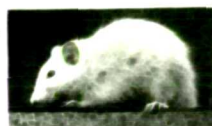
SIKH DIET VERSUS DIET OF POORER CLASS EUROPEAN.

The former diet consisted of whole wheat flour chapatties, butter, whole milk, dhal (legume), fresh raw vegetables ad libitum and fresh meat with bone once a week.

The latter diet consisted of white bread and margarine, tinned meat, boiled vegetables, tinned jam, tea and sugar with a little milk.



Two rats of the same age and initial body-weight: the one (left) fed on the Sikh and the other (right) on the poor European diet.



Two rats of the same age and initial body-weight: the one (left) fed on the Sikh and the other (right) on the poor European diet.



Two rats of the same age and initial body-weight: the one (left) fed on the Sikh and the other (right) on the poor European diet.

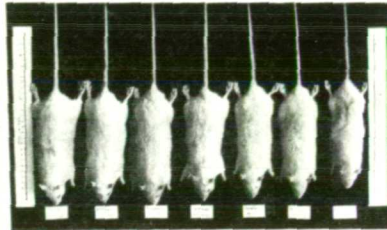
The rats shown above are representative of 20 in each group. Duration of experiment 187 days. Average initial body-weight both groups 125 grams; average final body-weight: Sikh, 188 gms; poor European, 118 gms. Common diseases in the latter group were pneumonia and gastro-intestinal ailments.

Source: McCarrison's 'Nutrition and National Health' (31)

DIET AND PHYSIQUE OF INDIAN RACES.



Hunza Hillman: Diet: whole cereal grains (mainly wheat), milk, vegetables and abundant fruits-apricots, etc.; meat occasionally.



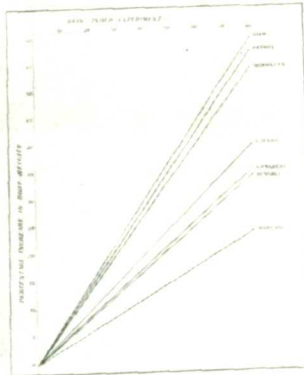
Average representatives showing weight in grams of 7 groups of rats fed from the same early age on certain national diets of India. The best of these diets (Sikh) was composed of whole wheat, butter, milk, legumes, vegetables with meat occasionally. The worst (Bengalis and Madrassies) is one composed mainly of rice.



Tibetan Hillman - representative of dandy carriers, rickshaw-men, etc. Very hard worked. Average protein intake 175 grams daily, of which over 60% is derived from animal sources. The heat value of their diet may be as much as 6,000 calories daily (M^cCa₉).



East coast cultivator: Diet: rice with dhal and vegetables and a small amount of fish, milk, and butter. Protein from 50 to 70 grams daily; calories 2,400 to 2,750 (M^cCa₉).



Percentage increase in body-weight of 7 groups of young rats, of the same initial aggregate weight fed on certain national diets of India (vide photograph above).



Nepalese Hillman (Goorkhal) Protein 120 to 150 grams, of which less than one third is derived from animal sources. Calories 3,000 to 3,200. Such people eat largely the better class cereals-wheat, maize and good millets (M^cCa₉).



Bengali: Diet: rice, dhal, vegetables, oil with a little fish and perhaps a little milk. Protein, 50 grams daily; Calories 2,300 to 2,500 (M^cCa₉).



Mahraffa **Sikh** (M^cCa₉) **Pathan**



Typical of rice-eating Madrassi. Diet contains little or no animal protein. Calories low (M^cCa₉).

deficiency disease which could be prevented as well as cured by nutritional intervention, and it seemed to offer an immediate remedy for a disease which in some areas of the UK had reported prevalence rates of over 50% of the elementary school population (36).

The elimination of rickets was described by protagonists of the Newer Knowledge as being both important in itself and also as providing a means of preventing many other conditions which were believed to be caused by rachitic malformations. For example, the prophylactic use of the anti rachitic vitamin was described by the American nutritionist McCollum, in 1922, as a method of eradicating

"the universal prevalence of under-developed and feeble jaws, and inferior and decayed teeth, poorly developed nose and throat, adenoids, weak chest and indigestion".

These conditions were thought to

"undermine the constitution, dwarf the body and pre dispose to disease" (37).

Similarly in 1921, Fletcher, Secretary of the MRC, described dietary deficiency as the cause of the

"stunting and deformity one sees in every city crowd" (38).

2.4.2. Sub Optimal Health and Vitamin Deficiency

Experimental evidence suggested that changes in the diet would not only eradicate diseases and malformations associated with vitamin deficiency but that these changes would also lead to general improvements in standards of health and 'vitality' in the community. During the 1920s and 1930s, experimental evidence from which these inferences were drawn was exhaustively documented by E V McCollum in his book, the NEWER KNOWLEDGE OF NUTRITION (39) which first appeared in 1918 and was regularly revised. This book provides an important record of the public health applications of the 'newer knowledge' as they were perceived by contemporary researchers. Although McCollum's writing is unmistakably

that of an enthusiast he is set apart from his colleagues only by the prolific nature of his literary output. His views were widely shared, notably by the Secretary of the Medical Research Council, as well as by other leading scientists working on problems of human and animal nutrition. McCollum argued that the study of nutrition as a branch of preventive medicine had not been sufficiently appreciated in the past and he presented evidence which suggested that 'new and surprising relations between food and health' had recently become apparent (40). In particular, he claimed that research in the field of nutrition had

"a greater value in preventive medicine in relation to raising the vitality of mankind...than they have to the prevention of the occurrence of the deficiency diseases. This fact has never been sufficiently appreciated" (41).

This view was based on the belief that

"by far the most important effect of faulty nutrition in man is the result of errors...which are not sufficient to cause prompt and spectacular failure...it is the gradual operation of more or less constant, but unperceived causes..which in most cases are responsible for undermining the health of the individual" (42).

McCollum maintained that an improperly constituted diet was

"one of the causes of inferiority in physical development, instability in the nervous system, lack of recuperative power and endurance and consequent cumulative fatigue, and lack of resistance to infections such as tuberculosis and other types where specific immunity is not easily developed by the body" (43).

The *Committee on Physical Deterioration* had been summoned to inquire into problems of precisely this nature and the *Newer Knowledge*, which seemed to hold the promise of a more vigorous and disease resistant population, was therefore welcomed by the public health movement. The belief that new levels of health and vitality could be produced by changes in the diet was a driving force in nutrition science during the inter war years (as it is at the present time) and it was used by Fletcher to justify the allocation of a large proportion MRC funds to nutrition. Thus, in his Annual Report for 1927 he wrote

"who can guess what new relations of diet to disease or liability to disease are waiting to be discovered" (44).

2.4.3. Marginal Malnutrition

An essential feature of the "sub optimal health" from which large sections of the population were thought to suffer, was the gradual operation of 'unperceived causes' (45). It was argued by protagonists of the 'newer knowledge' that

"Physical deterioration.... generally is unnoticed or not admitted until it is pronounced.. it is borderline states of malnutrition, characterised by a condition of nutritional instability, that is the greatest menace to the individual when his health and efficiency over the life period is considered" (46).

This belief was to have an important influence on the perception of the nutrition problem that existed among the population of the depressed areas of the United Kingdom in the late 1920s and early 1930s. Observers from the Ministry of Health and Board of Education reported that although child health appeared to be standing up well to adverse conditions, the population was nevertheless at the brink of serious deterioration (47). This same view, which was expressed by a number of Local Authority Medical Officers and in the public health press (48), is discussed more fully in Chapter IV. It was believed that many children in the depressed areas had entered what McCollum referred to as the "twilight zone'of nutritional instability " (49).

The notion of marginal malnutrition influenced the Ministry of Health's decision to convene an Advisory Committee on Nutrition in 1931 and fuelled the public health debates of the 1930s. However, during this period, the continuing absence of clinical evidence of 'nutritional instability' led the public health administration to take an increasingly sceptical view of this alleged condition; these issues are discussed in Chapters III and IV.

2.4.4. The Protective Foods and Nutrition Education

The development of the idea of the protective foods and its acceptance by the public health movement is so well known that it has escaped comment, although its impact, particularly in the field of nutrition education, was immense.

McCollum recognised at an early date that the modern nutritionist could not confine his activities to the laboratory ; it was necessary for nutrition scientists to communicate their experimental work in three important ways. Firstly, they had to translate their findings into dietary advice for human populations. Secondly, they needed to convince the public health profession that experimental work had immediate implications for human populations. Finally, they had to enlist the support of the public health profession in changing dietary habits. McCollum can claim a personal success with regard to the first objective. In 1918 he coined the memorable phrase "the protective foods" and subsequently outlined a scheme of popular dietetics which remained essentially unchanged for the next fifty years (50). In McCollum's scheme there were

"two classes of foodstuffs, of peculiar value in human and animal nutrition. These tend strongly to correct the deficiencies in a cereal, legume seed, tuber and fleshy root diet, or one in which in addition to these muscle meats are included. These are milk and the leafy vegetables...the protective foods" (51).

During the inter war period public health workers were extremely receptive to this view of dietary quality, which was based on ideas which had emerged from the animal feeding laboratories and their enthusiastic response to the call for dietary re-education is considered more fully in Chapter IV. The concept of the protective foods carried a powerful crusading message which was readily adopted by the public health movement, not least because the new dietetics continued to blame the poor for an incorrect choice of food. Although the details had changed, 'science' still taught that some foods had a special merit

and that ignorance of the relative 'value' of foods was a major cause of inferior physique. Just as the Committee on Physical Deterioration had argued in 1904 so McCollum wrote in successive editions of the NEWER KNOWLEDGE OF NUTRITION that

"More can be achieved through dietary reform than through any other agency" (52).

The assertion that

"The specific dietary properties of the food mixtures which enter into the diet are of paramount importance" (53)

would have been accepted by the expert witnesses summoned by the Committee in 1904 and indeed many of the foods recommended to the ignorant poor remained unchanged - the only difference was in the scientific justification for this advice. Despite these fundamental continuities which assisted the passage of the Newer Knowledge from the laboratory into public health practice, the discontinuities are also striking. Before the advent of the Newer Knowledge of Nutrition, essentially the ratio between protein and other energy yielding nutrients had formed the criteria for judging dietary quality; according to the Newer Knowledge, neither protein nor energy but vitamins were of paramount importance. This revised definition of dietary quality changed the emphasis from the macro nutrients to the micronutrients and the full implications of this change in terms of nutrition intervention strategies and their appropriateness for the target population are discussed in Chapter 5.

2.4.5. Food or Housing? Optimising the chemical environment

Although the public health administration took up the new dietetics based on the philosophy of the 'protective foods', with great enthusiasm, it parted company with the scientists over their interpretation of the range of factors which influenced nutritional status. The Committee on Physical Deterioration had claimed that poor

nutrition was the product of social, dietary and environmental influences and during the 1920s and 1930s the public health administration continued to argue this case. However, according to the newer knowledge of nutrition the external environment was of negligible importance; the efficiency of physiological processes was determined exclusively by the 'internal chemical environment'. In his summary of experimental evidence, McCollum wrote that in bad hygienic conditions, a "highly satisfactory diet" was capable of protecting young animals to a "surprising degree"(54). The message for the public health administration was clear. Dietary reform should take priority over further improvements in sanitation, housing and water supply. It will be shown that Fletcher, who was a key figure in transmitting scientific ideas from the laboratory to the political forum in the UK, was deeply committed to this point of view. He complained bitterly that the Ministry of Health was more concerned with housing than with nutrition (55) and supported the findings of the experimentalists, which suggested that

"our most effective defense barriers against most of the dangers to which our environment exposes us lie within us" (56).

Nutritionists recognised that this implied the adoption of new priorities in public health work. For example, McCollum wrote in 1922 that

"Without minimizing the importance of bacteriology, pathology or parasitology .. the time has now arrived when greater attention should be given to the most fundamental problem of all viz: the supervision of the conditions under which the development of the normal individual takes place" (57).

It was argued that by perfecting the 'chemical environment, new standards of 'positive' health could be achieved. Just as animal experiments demonstrated that many 'degrees of gradation' of malnutrition could be induced by the adjustment of the diet, so it was claimed that degrees of gradation of health could also be achieved by

dietary means. Thus, nutritionists argued that freedom of choice and a variety of food sources could no longer be seen as a basis for securing dietary adequacy. According to McCollum

"It is not enough for the diet to yield a proper calorific value, furnish protein according to the long accepted standards, and afford variety and palatability.. The specific dietary properties of the food mixtures which enter into the diet are of paramount importance and must be given due and weighty consideration" (58).

In the same way he argued that the priority in child nutrition was

"that of securing.. a wisely planned diet calculated to raise their vitality to a higher plane" (59).

The definition of the precise levels of nutrient intake required to achieve these results subsequently became a major priority in nutrition research; and by the mid 1930s the income necessary to secure these nutrients had become a central issue in the public health debate.

2.5. Opposing Views: The Older Knowledge of Nutrition

In 1922 McCollum described his vision of human progress through the application of nutrition science.

"The ultimate aim of scientific investigations is to give man control over the forces of nature. The science of nutrition gives promise of making possible the realisation of the optimal condition of physical well being" (60).

The belief that this goal could be achieved by perfecting the dietary intake of human populations was a hallmark of the Newer Knowledge of nutrition and it was accepted by leading figures in the medical research establishment from the early 1920s . However, this view was not shared by all members of the scientific community and dissenting voices were raised in the debate about food and health throughout the inter war years. It is beyond the scope of this thesis to discuss the internal politics and philosophies of British science during the 1920s and 1930s, and a comprehensive account of the individuals and University faculties which resisted the newer knowledge of nutrition would be out of place. Instead, the views of the two leading dissenters, D. Noel Paton and E.P.

Cathcart, who were also admitted to the Council and Committees of the MRC, are outlined briefly in the following section, and their work representative of the rest is discussed at relevant points in the text. Paton and Cathcart were protagonists of an older view of nutrition. Paton was Regius Professor of Physiology at the University of Glasgow from 1906 until his death in 1928 and Cathcart was also a Glasgow physiologist. Although he was appointed to the Gardiner Chair of Biochemistry in 1919, Cathcart proceeded to change this to a second Chair in Physiology (61).

Like J.S. Haldane, Paton and Cathcart championed an 'holistic' approach to physiology (62) and argued that the nutritional process could only be understood by looking at the wide range of social and environmental factors which they believed influenced its outcome. This of course was in sharp contrast to the newer knowledge, which taught the primacy of the 'chemical' environment. Cathcart wrote extensively on this subject during the 1920s and 1930s. For example, in 1928 (63) he attacked the biochemists whom, he claimed saw organisms as 'chemical factories' and wrote that

"The whole mechanistic outlook is to me anathema..our methods may be muddy, may be amateurish, may be childish, but we at least do not shut our eyes to the fact that we are dealing with a living organism and that it is no use pottering about with isolated fragments".

Their concern with the living organism extended to an interest in the social traditions and psychological constraints which they believed influenced physiological functioning and ironically Paton's work on the quantification of these factors produced a concept which was compatible with the Newer Knowledge. He argued that 'maternal efficiency', by which he meant good domestic management, was the key variable in determining child health as assessed by height and weight. The study which gave rise to this view is discussed in Chapter IV.

The MRC Secretaries of the inter war years, Walter Fletcher and Edward Mellanby, were deeply committed to the Newer Knowledge of nutrition, and strongly opposed to 'environmental' interpretations of the aetiology of malnutrition. It might therefore seem paradoxical that Paton and Cathcart should have held prominent positions on the Council; Paton was Secretary of the Scottish Child Life Committee which carried out extensive studies into environmental factors associated with rickets between 1916 and 1921 (64) and Cathcart was Chairman of the Committee on Quantitative Problems of Human Nutrition from 1921 to 1926 and of the subsequent Nutrition Committee from 1926 to 1933. This is partly explained by the facts of historical change: they were eminent in their respective fields before the implications of the newer knowledge were recognised. However, their opposition did not concern the powers within the MRC unduly. For example, in the period 1918-1921 Paton and the Glasgow physiologists led a rearguard action against clinical and experimental work on the dietary aetiology of rickets and continued to argue that social, environmental and hygienic factors were responsible for this disease. Shortly before Mellanby's MRC monograph on experimental rickets was due to appear, their own study of rickets in Glasgow was completed. However, Hopkins and Fletcher ensured that the opposition view, if not suppressed, received the minimum attention and discussions took place at this time which considered how best to 'pave the way' for Mellanby (65). During the inter war period, the MRC dealt with scientific opposition to the newer knowledge of nutrition by a policy of 'repressive tolerance'.

NOTE* Although Hopkins was awarded the Nobel Prize for his work on the Vitamins, he was not primarily interested in nutrition and dropped nutrition research altogether after 1918. This was a source of considerable aggravation to the Secretary of the Medical Research Council, Sir Walter Fletcher. Fletcher placed great emphasis on the importance a strong biochemical basis for medical research and in 1924 he secured the Dunn bequest for Biochemistry (and Hopkins) at Cambridge. Nutrition was the outstanding field in which biochemistry had an applied role in medicine and Hopkins's neglect of the subject led Fletcher to write in 1927 "I told Hopkins that, having somehow bagged the credit for inventing vitamins, he spends all his time collecting gold medals on the strength of it, and yet in the past ten years has neither done, nor got others to do, a hand's turn of work on the subject" (MRC 1349, Fletcher to Hill, 20 Jan, 1927).

CHAPTER 3 - PART I

PUBLIC HEALTH APPLICATIONS OF THE NEWER KNOWLEDGE OF NUTRITION

The Public Health Administration and the Medical Research Council

3.1. Preventive Medicine in the Aftermath of the 1914-18 War

In 1919 legislation establishing a Ministry of Health was enacted and a specific obligation to advance preventive health was written into its terms of reference (1). By this date, the medical research establishment believed that nutrition science offered great opportunities for preventive medicine which could be used for the purpose of national physical reconstruction. The Newer Knowledge of Nutrition seemed to offer solutions to precisely those problems which had concerned the Committee on Physical Deterioration; problems of poor physique, low vitality, growth failure and lack of resistance to disease. In the atmosphere of renewal and regeneration which was a feature of the immediate post war years, nutrition scientists and the medical research establishment looked to the application of their discoveries in public health practice with great optimism. This Chapter deals with the translation of the Newer Knowledge of Nutrition into public health objectives.

Nutritionists and the Medical Research Council (MRC) argued that the public health possibilities of the Newer Knowledge called for action from both local and central government; the framework for the shaping of policy during the inter-war period was binary, involving both the Ministry of Health and, in an advisory capacity, the MRC. However, controversy raged between the administration and the MRC over the precise inferences of nutrition science for public policy.

3.1.1. The Ministry of Health

Established in 1919, the Ministry of Health consisted of 10 sections which had responsibility for areas including Maternal and Child Welfare,

General Health and Epidemiology, Sanitary Administration and the supervision of Food Supplies (2). A key figure at the Ministry of Health was Sir George Newman, who was appointed Chief Medical Officer in 1919. At the time of his appointment, Newman already held the post of Chief Medical Officer at the Board of Education and he kept both positions until his retirement in 1933. Newman wrote extensively on public health topics and used his annual reports as a vehicle for expounding his views both on priorities for the public health profession, and on the nature and cause of ill health in the community. Nutrition was a topic to which he regularly returned in his reports. "On the State of Public Health" (OSPH) was presented to the Minister of Health and was based on the annual returns of Local Authority Medical Officers; "The Health of the School Child" (HOSC) was presented to the President of the Board of Education, and was a summary of the annual returns of Local Education Authority School Medical Officers. These reports are an important primary source which shed light on the official view the public health administration wished to disseminate among its Medical Officers. In 1933 Newman was replaced by Sir Arthur MacNalty; however the change in leadership did not produce any major change in the official approach to nutrition.

3.1.2. The Medical Research Council (MRC) and Nutrition

The MRC was wholly independent of the Ministry of Health. Medical research first received public funding in the UK in 1911, when it was introduced as part of a workingmen's health insurance scheme. Contributions towards Medical research were set at the annual rate of one penny for every insured person and yielded £53,000 in the first year (3). A committee was set up under the National Health Insurance Commissioners to administer the fund and its first Secretary, Walter Morely Fletcher, was appointed in 1914. Fletcher remained Secretary of

the new Medical Research Council which was formed in 1919 under the provisions of the Ministry of Health Act. The Act made available an annual Exchequer grant for Medical research and transferred the general administrative control of this research to a ministerial committee of the Privy Council. This ensured the Council's autonomy; it was free to pursue a programme of primary biomedical research without the constraints of short term Departmental pressures.

The Secretaries of the MRC during the inter war period, Walter Fletcher, who held the post from 1919-1933 and his successor, Edward Mellanby, both gave high priority to research in nutrition. During the 1920s and 1930s, nutrition regularly received more than a tenth of the MRC's total research budget which remained at a level of around £60,000 throughout this period. (4). Fletcher's commitment to the 'Newer Knowledge of Nutrition' played a crucial role in the development of nutrition science in the UK. Before his appointment to the Medical Research Committee in 1914, he had been engaged in medical research as a Fellow of Trinity College Cambridge and was elected to the Royal Society for work on muscle physiology carried out in collaboration with Hopkins. Fletcher was a keen admirer of Hopkins's biochemical research and was anxious for the MRC to promote the study of nutrition, the medical applications of which had been apparent to him from an early date (5).

Mellanby, who was MRC Secretary from 1933-1949 was a leading nutrition scientist, who had been a student and later research associate of Hopkins at Cambridge. This scientific association, which began in 1907 continued until Hopkins's death in 1948 (6). Mellanby's work on experimental rickets, undertaken during the 1914-18 War whilst he held the Chair of Physiology at King's College for Women, contributed to the understanding of the role of vitamin D in the aetiology of this disease and his subsequent research carried out at the University of Sheffield,

where he held a Clinical Chair, was chiefly concerned with the possible role of Vitamin A as an anti infective agency (7).

3.1.3. The Fletcher Years: Strengthening the Research Basis

The annual report of the Medical Research Council which covered the period 1920-1921 described nutrition research as an example of

"work which it is highly proper to support from public funds" (8).

In the case of nutrition, laboratory research had produced results which were of immediate relevance to public health practice; it had proved to be a sound investment. Fletcher drew attention in his reports of the early 1920s to the fact that advances in nutrition science, made under the auspices of the MRC, were in precisely those fields of preventive medicine which had been identified during the setting up of the new Ministry of Health, as priorities for national health. Thus, in 1924 he argued that the elucidation of the aetiology of rickets provided a means of curing a major cause of physical degeneracy (9) and in subsequent years he was able to record a series of further advances in the preventive applications of nutrition science. For example in 1925 he claimed that work on dental malformation provided an index of 'hidden deficiency', and 'sub optimal nutrition' the importance of which, from the point of view of preventive medicine, could "hardly be exaggerated" (10). These observations recall the descriptions of 'marginal malnutrition' and 'sub optimal health' discussed in Chapter 2. Fletcher shared the experimentalists' belief that the importance of nutrition in preventive medicine had not been sufficiently recognised by the public health administration. He used his annual reports to rectify this view and at the same time to strengthen the case for the public funding of primary research.

His views were endorsed by the public health press; for example, in 1926 an editorial in the MEDICAL OFFICER commented

"No department of medicine is in a more healthy state than the study of dietetics. In no other sphere has the laboratory and the clinic worked in such close communion; in none has the theoretical worker demonstrated such sound practical sense nor the clinician absorbed and utilised so much pure science. All fields of study have come together and the result is a steadily progressing improvement in curative and preventive medicine" (11).

Fletcher also believed that nutrition science strengthened the MRC's case for generous Exchequer funding since it enhanced the position of the UK as a leader in the international scientific community. He drew attention to this in the MRC's annual report for 1920-21, when he claimed that vitamin research had been 'originated' in the United Kingdom, and stressed that the initiative should not be allowed to pass to development in other nations (12). Ten years later he expressed the same sentiments in correspondence with Lord Dawson, President of the Royal College of Physicians

"The Government funds of the MRC from 1913 for the first time gave a chance for the proper development of this vital subject in its native home. The result is that we have not only caught up with the Americans but have taken the chief lead at most of the vital points of advance. The Germans are only just beginning to realise that they have missed the 'bus by about fifteen years" (13).

Fletcher recognised that a close collaboration between the Ministry of Health and MRC was necessary if the discoveries of nutrition science were to be translated into public health practice and in 1919 he set out detailed proposals for relations between the Ministry and the Council with respect to nutrition policy and research. His memorandum anticipated collaboration in field-research and cooperation from the Ministry, through its network of Medical Officers and Maternal and Child Welfare services, in the application of scientific discoveries (14). Fletcher's proposals initially met with a favourable response from the Ministry. For example, in 1920 the MRC received a request for investigations into dental decay and the 'relation of milk to nutrition', both of which were undertaken (15). Work on 'predisposing dietary factors' in the causation of dental caries became a research

priority in the 1920s and the inquiry concerning nutritive value of milk eventually produced Corry Mann's report, "Diets for Boys during the School Years" (16). However, after this initial dialogue, relations between the Ministry and MRC became increasingly strained and later proposals for collaborative work in nutrition, particularly in relation to Maternal and Child Welfare, were unsuccessful (17). These worsening relations were partly a reflection of conflicts over the demarcation of legitimate spheres of interest between the MRC and the Ministry.

Fletcher was suspicious of the initiation of biomedical research programmes by bodies other than the MRC and although a concordat (18) defining respective research responsibilities was drawn up in 1924, there was little collaboration in studies relating to problems of human nutrition during Newman's tenure of office.

Thus, in the course of the 1920s, Fletcher's initial vision of a united attack on the problem of dietary deficiency remained unfulfilled and he became increasingly outspoken in his criticism of the Ministry's attitude to nutrition. This criticism was not confined to the public health administration, but extended to Medical Officers of Health who, Fletcher claimed, had not yet grasped the significance of discoveries in nutrition science. For example in 1928 he delivered a lecture at the Public Health Congress and Exhibition in which he urged Medical Officers to go into the laboratories and familiarise themselves with experimental studies in nutrition and then to apply these scientific discoveries in their practical work (19). Similarly, in one of his last printed works, which appeared in the first volume of NUTRITION ABSTRACTS AND REVIEWS, Fletcher wrote that the public health worker was

"at the opening of a new chapter in preventive medicine, comparable indeed with that opened to his predecessors at the dawn of bacteriology. He has again to learn as they did, the philosophy of the 'infinitely little'".

Fletcher argued that contact with experimental work was essential for these workers, since it brought

"conviction of the 'threshold' for adequate nutrition, of the actuality of a veiled malnutrition" (20).

3.1.3.1. The Case Against the Ministry

Fletcher's anger and frustration over the official attitude to nutrition reached a climax in 1931 when Newman asserted in his annual report that the nutrition of the pregnant woman was a matter of "commonsense" (21).

"This opinion",

Fletcher wrote in a furious reply

"expressed in spite of so much brilliant recent British work, seems to me particularly unfortunate."

He reminded Newman that the MRC needed the help of the Ministry in

"convincing the Treasury and the taxpayer that this kind of research ..is of vital and immediate practical value to this crowded island kingdom"

and pointed out that his suggestion that correct diet was merely a question of "commonsense" severely undermined this case; it suggested that nutrition did not belong to medicine and was therefore not a proper part of medical research.

Fletcher argued that scientific work had shown at "point after point" that improving dietary quality was far more important than

"increasing quantity ignorantly (or by common sense) as wages rise and far more important than improvement in housing" (22).

In arriving at this view, Fletcher not only extrapolated from animal experiments showing the beneficial effects of diet, but animal experiments which failed to demonstrate environment had an effect.

Fletcher interpreted Newman's report as evidence that, despite assurances to the contrary, Newman condoned the Ministry's policy of putting housing first, which led effectively to the withholding of action on nutrition. These issues were discussed at length in an

exchange of letters with Major Greenwood, Chairman of the Ministry's Advisory Committee on Nutrition (23) and a member of the MRC's own Nutrition Committee. Similar complaints can also be found in earlier correspondence with the Ministry, with Lord Dawson, and with Robinson, Secretary to the Minister of Health (24). Fletcher's case against the Ministry included the specific charges that it had failed to undertake a vigorous nutrition education campaign; that it had refused to press for changes in the food processing and manufacturing industries, in relation to the fortification of foods such as margarine and the preservation of the vitamin content of canned foods; and that the Ministry had given higher priority to rehousing than to nutrition.

3.1.3.1.1. Nutrition Education

As early as 1921, Fletcher criticised the Ministry's approach to nutrition education when he wrote to MacFadden, a senior official in the Food Division that

"already there is abundant knowledge which would justify the Ministry of Health giving strong and clear guidance to the producing and consuming public".

He argued that in a recent official report, DIET IN RELATION TO NORMAL NUTRITION there was

"not much appeal made or help given, to the mother of a family, to tell her what to buy and what not to do in the kitchen...this is the very kind of of knowledge which the Ministry of Health ought to disseminate as from the housetop" (25).

In the same way, he maintained that the MRC's report on the "Nutrition of Miners and their Families", published in 1924 (26), was further evidence of the need for a

"really big campaign in this fundamental business of Nutrition and the MRC nutrition and the right use of money and of cooking methods" (27).

This report contained detailed analyses of family budgets, from which it was inferred that improvements could be made in the "economical use of food" which provided a rationale for much of the nutrition education

work carried out during the inter war period. A re-analysis of this data appears in Chapter 4.

3.1.3.1.2. Food or Housing

Fletcher's complaints that the Ministry had not done enough to promote nutrition education could be challenged - the Ministry DID support the 'new dietetics' based on the theory of the protective foods, and this was rapidly adopted by the public health movement. However, it is true that there were fundamental differences between the Ministry and the MRC over the relative importance of housing (or the external environment) and diet (or the internal environment). The 'Newer Knowledge of Nutrition' taught that the internal chemical environment rather than the external physical environment determined the success of the organisms's physiological functioning. Fletcher expressed his agreement with this view in correspondence with MacFadden, Dawson and M'Gonigle as well as with Newman.

Thus in 1921 he told MacFadden that

"personally I believe [dietetic deficiency] is overwhelmingly more important than the housing question" (28).

Ten years later he made the same comment to Lord Dawson, who had recently accepted a seat on the Medical Research Council. He wrote on the subject of nutrition that

"..the medical administrators have delayed inexcusably to use the abundant new knowledge that has been pouring out of the laboratories in the last fifteen years...Physiology teaches that faulty nutrition is far worse for our population than faulty housing. We waste untold millions a year by not using the new knowledge we have"(29).

Similarly, in correspondence with M'Gonigle, MOH for Stockton on Tees, he stated that

"I have been maintaining for at least ten years that the whole point of modern physiological knowledge was to the importance of diet, rather than that of better housing, in the interest of national health" (30).

M'Gonigle had undertaken a study which seemed to show that there was a correlation between rehousing and increased mortality and he argued that the higher rents charged for new properties led to a decline in standards of nutrition and higher death rates (31). Responding to a grant application to further his enquiries, Fletcher wrote

"..On purely physiological grounds it has seemed to me to be wholly wrong to squander money on housing without either (1) studying the indirect effects this may have upon the diet of the people concerned and (2) or at least an equally active policy and expenditure in bringing to the people themselves the benefits of our new and rapidly growing knowledge of nutrition " (32).

Fletcher failed to convert the public health administration to the view that dietary change would in itself revolutionise the nation's health. However, under his guidance the MRC developed a strong research basis for nutrition science in the UK and during the 1920's MRC funded projects were initiated in four areas:

- a) The determination of the chemical nature of the vitamins. Work in this area was carried out principally at the Lister Institute (33).
- b) The elucidation of functional failure caused by vitamin deficiency. The Dunn Nutrition laboratories were established to undertake work in this field (34).
- c) The determination of the vitamin content of foods. McCance and Widdowson made a comprehensive survey of the vitamin content of common items of the British diet. This work was essential for the translation of experimental work into dietary guidelines for the British population (35).
- d) The determination of the nature of the normal British diet. Work in this area was dominated by E P Cathcart. Cathcart was chairman of the MRC's Nutrition Committee from its inception in 1926 until 1933 and a was a member of both the Ministry of Health's Advisory Committee (1931-34) and the subsequent Interdepartmental Advisory Committee on Nutrition (36).

This comprehensive research programme bears witness to Fletcher's success in achieving one of his prime objectives: throughout the inter war period the United Kingdom remained a world leader in nutrition research.

3.1.4. Mellanby's Policy for Food and Health

Following Fletcher's death in 1933, Edward Mellanby was appointed to the post of MRC Secretary. Although this did not lead to a break in the priority given to nutrition research, Mellanby's tactics in dealing with the Ministry of Health were very different from Fletcher's. He translated Fletcher's appeals for a more 'positive approach' to nutrition into proposals for a national nutrition policy and at the same time, adopted a new strategy in his relations with the Ministry of Health. Mellanby believed that the most effective way of influencing government was through officially appointed expert advisory committees. Fletcher, by contrast, had preferred to restrict the discussion of scientific matters to the MRC's own Committee structure. Thus, whilst Fletcher reluctantly tolerated the appointment of the Ministry of Health's first Advisory Committee on Nutrition in 1931 (37), Mellanby had campaigned vigorously for the appointment of such a Committee (38).

In 1927 Mellanby outlined a programme for State action in relation to food (39) which put forward four linked arguments. Firstly, that efforts by the State and local government in relation to nutrition would bring about improvements in health equal to those of the sanitary revolution of the 19th Century. Secondly, that 'correct feeding' was of even greater importance than good hygiene in combatting the problem of disease, since it appeared that nutritional factors increased individual resistance to infection. (This was a reference to his own work on Vitamin A as anti infective agent (40).) Thirdly, that vitamin and mineral deficiencies were responsible for various disorders including

bone deformities, dental disease, scurvy, goitre and "nervous symptoms". Finally that "stunted growth" was caused by "deficient intake of biologically good protein".

Mellanby asserted that knowledge of dietetics was "greatly in advance of the application of this knowledge ... by official bodies" and that a Board of Nutrition, composed of expert experimental scientists should therefore be convened by the Ministry of Health in order to advise the Ministry as to the 'relative importance of new discoveries'. He suggested that this body should also guide the public through the morass of conflicting statements which were current, such as those which concerned the relative merits of white and wholemeal bread.

The role which Mellanby allotted to experimental scientists in defining the public health applications of their work and in transmitting this directly to the public in the form of "really authoritative statements" signalled a new and very important departure. Fletcher's view, which is documented in correspondence with both Lord Dawson and Ministry of Health officials (41), was that the place for scientists was in the laboratory and not in Committee rooms, especially when these were the Ministry's rather than those of the Medical Research Council. Mellanby preferred to work directly with Whitehall and his enthusiasm for official Nutrition committees was revealed again in 1934 when he joined the Cabinet's Economic Advisory Committee (EAC), in his capacity as Secretary of the MRC. He used his seat on this Committee, which had direct access to the Prime Minister, to press the case for an Interdepartmental Advisory Committee on Nutrition (42).

Mellanby's success was due in part to the assistance of Sir Maurice Hankey, Secretary of the Committee on Imperial Defence and Clerk of the Privy Council, who had become convinced during Fletcher's term of office that changes in the diet were a precondition for improvements in

national health (43). Hankey gave Mellanby both advice and support in presenting the case for a new Nutrition Committee. In May 1934, he addressed the Scientific Research Sub Committee of the EAC on the subject of poor standards of national physique as indicated by recruiting statistics (44); this is reminiscent of the arguments which prompted Balfour's government to convene the Committee on Physical Deterioration in 1904. Hankey's address was followed by a statement from Mellanby in which he argued that if "real progress" was to be made in the matter of poor physique "a carefully thought out [nutrition] policy was required" (45) and recommended that a strong committee of the EAC should be convened to investigate all aspects of the problem. As a result of this meeting, Mellanby won the Scientific Research Committee's support. Hankey further assisted Mellanby by submitting a personal memorandum to the Lord President, Stanley Baldwin, in which he stressed the importance of improving the national diet in the interests of national security (46). This prepared the way for the official EAC proposal (47), in which it was argued that a thorough inquiry into national nutrition was urgently needed. Following Hankey's advice, Mellanby stressed the defence aspects of the problem; he quoted the high rates of rejection of army recruits, but avoided mention of the cost to the Ministry of Health which his policy of "feeding the mothers" implied (48). In this proposal, Mellanby set out his views on the associations between nutrition and ill health. He argued the following case:

"Benefits which, on the basis of existing scientific knowledge, it appears certain would accrue from a food policy

- i) an increased average stature of the population. It is probable that this might result in an average increase in 2 or 3 inches in the adult;
- ii) a great reduction in dental caries, amounting possibly to 60% or more of the present incidence;
- iii) the elimination of rickets and its sequelae;
- iv) a great reduction in the incidence of pyorrhoea;
- v) the elimination of simple anaemia;
- vi) the elimination of simple goitre;
- vii) a general increase in the physical activity and the mental alertness of the average individual".

Benefits which would probably but not certainly accrue from a national food policy were described as

- "i) a reduction in middle ear disease with its sequelae of discharging ears and deafness;
- ii) a reduction in broncho-pneumonia and bronchitis in early life and in catarrhal conditions of the respiratory tract;
- iii) a reduction in septic tonsils and adenoids and septic nasal sinuses;
- iv) a reduction in all forms of tuberculosis;
- v) a reduction in acute rheumatism and its disabling result, rheumatic endocarditis".

[Rheumatic endocarditis was at this time the commonest form of heart disease.]

Mellanby argued that the main dietary defects to which a national food policy should address itself were the over-consumption of cereals in the form of bread and similar foodstuffs and the under-consumption of protective foodstuffs, such as milk, cheese, eggs and green vegetables. His memorandum set out a series of dietary standards for specific groups and maintained that ante-natal clinics, child welfare centres, the school medical service and the national health insurance scheme formed

"a chain of organisations..well fitted to carry out any scheme that might be adopted for the improved feeding of the population" (49).

During the period October 1934-January 1935 Mellanby also advised the Political and Economic Planning group (PEP), which produced a nutrition policy based largely on these views (50). Although these policy documents put the problem of poor physique in the context of national defence, as it had been in 1904, an exclusively dietary solution was now proposed. Mellanby believed that the problem could only be remedied by extensive nutritional intervention, and he hoped that an inquiry into the nation's food habits would convince government of the need to take such action.

These lobbying tactics were successful and in February 1935, the Prime Minister announced the appointment of an interdepartmental Advisory

Committee on Nutrition (51). Mellanby clearly felt he had won over the real decision makers; thus, in October 1935 he wrote to Macnalty, the Chief Medical Officer, that

"There is not only a great deal of public interest in Nutrition but both of us know there a number of people of high political standing who think it is of first importance and intend to drive ahead with it" (52).

However, Major Greenwood, who had chaired the Ministry of Health's first Advisory Committee on Nutrition which sat from 1931-1934 was scathing of Mellanby's faith in his influence over people "of high political standing" (53). Greenwood's scepticism proved to be well founded for the appointment of another Nutrition committee did not convert the Ministry of Health to the view that, by supplementing the diets of the poor, major problems of national ill health could be solved. Until the outbreak of World War II, the official approach to the nutrition problem remained essentially that of the 1904 Committee on Physical Deterioration: that is, that it was caused by a range of social and environmental factors. This was at the root of the complaints raised against the Ministry by both Fletcher and Mellanby. In the following section, the official understanding of the nutrition question is explored more fully and the Newer Knowledge of Nutrition placed in the context of an existing approach to preventive public health work.

3.7.5. Nutrition and the Public Health Administration

3.1.5.1. Preventive goals in Public Health work, 1919-1939

Preventive goals were written into the Ministry of Health Act of 1919 which required the newly established Ministry to

"secure the preparation, effective carrying out and coordination of measures conducive to the health of the people, including... prevention and cure of disease...initiation and direction of research...collection, preparation, publication and dissemination of information and statistics.." (54)

These objectives reflected fiscal realism as well as post war idealism,

for it had already been accepted that money spent on preventing ill-health was money well spent. In the course of the debates which accompanied the Education (Provision of Meals) Act of 1906, the preventive principal was widely canvassed and the view that

"it will be found ultimately that it is a great deal cheaper to spend pennies on children than pounds on paupers" (55)

helped to secure a Parliamentary majority for school feeding at public expense (56). These same arguments were current in the years which immediately followed the Armistice and were expounded at great length by the Ministry's newly appointed Chief Medical Officer, Sir George Newman (57).

The philosophy of "prevention rather than cure" was re-iterated by officials in public pronouncements throughout the inter war years. For example, in 1931 Parlance Kinloch, the Chief Medical Officer for Scotland, wrote that

"One of the fundamental aims of statecraft must be the promotion of the health of the individual to the end that as a race we may grow in health and vigour.... both as medical administrators and as medical men - or... biologists, we are more concerned with life than with disease." (58)

During this period, the "promotion of health and vigour" was seen by the Ministry as an exercise in social education rather than in welfare intervention and nutrition played a central role in this policy. Despite MRC criticisms, the public health administration was in fact highly effective in securing the rapid dissemination of the 'new dietetics' among the ranks of the public health movement; this is demonstrated very clearly in the nutrition education literature produced by local authority Medical Officers during the 1920s, and in reports of health education campaigns (59). The official agenda for health education which these documents reflect is very similar to that set by Committee on Physical Deterioration and described in Chapter I. Improving the quality of working class diets, which had been identified in 1904 as a priority

for the public health movement, remained one of the main tasks of preventive medicine, and as in 1904 the official view continued to stress the importance of raising standards of personal and domestic hygiene. Thus, in 1923, PUBLIC HEALTH described the objectives of preventive medicine as follows:

"It is a commonplace to say that preventive medicine, which half a century ago was concerned very largely with the improvement of the grossly unhealthy circumstances of the environment has now, after largely mastering its problems in that sphere, advanced towards a more intimate concern in the conditions of health as they concern the individual" (60).

The article went on to discuss the importance of diet in securing health and recommended a programme of both public education and agricultural reforms similar to those proposed in the mid 1930s by the League of Nations, under the banner of the 'Marriage of Health and Agriculture' (61).

The importance of nutrition in preventive medicine was stressed by a delegation of public health workers and Medical practitioners which in 1921 called on the Ministry to distribute official information

"directing attention to cheap supplies of vitamins and other essential food substitutes (sic) which can be secured by utilising whole cereals and pulse, fruits salads and vegetables" (62).

Improving dietary quality was already seen in terms of increasing the intake of the vitamin rich 'protective foods'.

The Chief Medical Officer, Sir George Newman, acknowledged the importance of these foods in his report to the Board of Education in 1920 (63) and in subsequent official reports throughout the decade (64). He maintained that dietary defects were linked with a high incidence of tuberculosis (65) and increased susceptibility to disease (66) as well as being a direct cause of rickets and dental disease (67). The public health administration could not therefore be accused of ignoring the

Newer Knowledge of Nutrition, and indeed Newman asserted that

"sound nutrition [was] at the foundation of national health" (68).

However, Newman and the Ministry dissented from the teachings of the Newer Knowledge over a crucial issue: according to the official view, diet was only one of many factors which influenced the outcome of the nutrition process. It was for this reason that both Newman and his successor MacNalty denied that improvements in health could be secured simply by dietary change, and it was for this same reason that the Ministry would not meet Fletcher's and Mellanby's demands that nutrition should be given precedence over housing. Their refusal derives from the public health administration's explanation of the cause of poor nutrition and its understanding of the role of diet in the aetiology of malnutrition, all of which are dealt with in the next section.

3.1.5.2. The 'Official' Public Health Meaning of Malnutrition

In 1912 Newman described nutrition as:

".. a general physiological term which connotes a complex condition of things. It is true that for the nourishment of the body there must be food [but] healthy and complete nutrition is something infinitely more than mere feeding...it connotes a healthy body in all respects" (69).

This view was sustained throughout the inter war period, despite the discoveries of the newer knowledge of nutrition. Thus, in his report for 1928, Newman wrote:

"Nutrition is not an alternative term for 'food'. The malnourished child may lack fresh air; it may be in need of exercise or rest; it may be sickening with gastro intestinal trouble, rickets, tuberculosis or rheumatism, or it may have dental disease or adenoids. Lack of food is but one of the causes of malnutrition..." (70).

Environmental factors affecting nutrition were described as overcrowding, lack of fresh air and exercise and fatigue. This view was not revised by Newman's successor, MacNalty, who asserted in 1935 that

"the efficiency [of the nutritional process] depends on many factors besides food...upon adequate sleep, proper and uncrowded housing, sunlight, fresh air, exercise and even happiness" (71).

This view essentially endorsed the 'older knowledge' of nutrition, described in Chapter 2. Thus, in 1937 Cathcart (an arch proponent of the 'older knowledge') wrote in the British Medical Journal that malnutrition did not depend solely on the adequacy of food intake, but that

"lack of sleep, of play, of happiness, absence of worry and so on play a part in the determination of the condition...food alone cannot work miracles" (72).

3.1.5.3. The Cause of Poor Nutrition: A Continuing Story of Maternal Inefficiency

Under the heading 'Nutrition and Malnutrition', Newman wrote in 1910 that

"Defective nutrition stands in the forefront as the most important of all physical defects from which school children suffer"

and he called on Medical Officers to undertake research into its causes.

The practical value of such research was described as follows:

"there is no subject the elucidation of which is more baffling to the medical inspector, no condition more difficult accurately to estimate, with causes more complex and interwoven" (73).

However, in subsequent reports Newman did not hold back from making very precise statements about the root cause of poor nutrition which he consistently ascribed to a combination of ignorance and maternal inefficiency. Thus, in 1914 he wrote that

"the three chief causes of malnutrition in children are poverty, ignorance and the neglect of their parents and the greatest of these is probably ignorance" (74).

Newman described how poverty operated in the causality of malnutrition in his report for 1926 when he claimed that poverty was only an 'indirect' cause of malnutrition

"in that poverty may bring with it ignorance, unsatisfactory home conditions, lack of parental control..more often it is careless mothering, ignorance of upbringing and lack of nurture, than actual shortage of food which results in a malnourished child" (75).

Table 3.1

**MALNUTRITION RETURNS LONDON AND LOCAL EDUCATION AUTHORITIES 1909-1916
AND 1925-1938**

YEAR	PREVALENCE OF MALNUTRITION %	
	London	LEAs
1909	12.8	
1910	11.5	13.1
1911		11.8
1912		11.6
1913		
1914	9.4	12.9
1915	6.6	16.3
1916	7.6	
1925	6.3	9.5
1926	6.2	9.6
1927	5.3	9.0
1928	5.1	9.1
1929	5.2	9.5
1930	4.8	10.6
1931	4.8	11.2
1932	4.9	10.7
1933	4.7	11.1
1934	4.6	12.0
1935	5.7	11.3
1936	6.0	11.2
1937	6.1	11.2
1938	6.6	11.3

'Malnutrition' was assessed by School Medical Officers according to subjective clinical criteria.
Source: Annual Reports of the Chief Medical Officer to the Board of Education, 1909-1938. Table compiled by the present author.

"better medical care and supervision, better nurture, the remedy of infective conditions,..school meals, physical training, more fresh air and improved sanitation..and.. a great reform in the social life of the people" (81)

and in his last report, written in 1933, Newman explained that the period of mass unemployment had not caused a deterioration in child health due to the

"increased care and devotion of the mothers and teachers, to manifold forms of voluntary service and to the public provision of insurance benefit, school feeding and medical supervision" (82).

Where results were less than optimal, the blame was placed firmly on the shoulders of the victims of poor nutrition. Thus, Newman stated in his annual report to the President of the Board of Education for 1930 that, although in most parts of the depressed South Wales coalfield the physique of children had not been adversely affected, malnutrition was to be found in

"more or less isolated pockets of a socially inferior population" (83).

In a confidential report on conditions in South Wales, it was claimed that

"Parental inefficiency combined with the stress due to industrial depression has produced signs of definite deterioration in the school children" (83)

and that

"if the 'social' factor could be eliminated, the 'economic' factor can be overcome by wise and prudent management on the part of the parents, more especially if they are assisted by arrangements for the provision of meals to school children" (84).

Surveys conducted in South Wales and in the North East during the late 1920s were also cited by the CMO as evidence that diet was not the key factor in the aetiology of malnutrition and that, in addition to food,

"..much else is needed to secure nutrition - fresh air, warmth, exercise, rest, cleanliness, and above all domestic nurture" (85).

Similarly, a study carried out by the LCC in 1932 (86) was interpreted as evidence that malnutrition was not necessarily associated with

unemployment. Despite the crude nature of this study , which failed to give the family incomes of children studied, it was received with great enthusiasm by the Ministry and was reported to the Minister of Health as evidence that malnutrition was

"ignorance quite as much as insufficient resources" (87).

The official view aroused little critical comment until the late 1920s; thereafter, the CMO's reassurances that economic conditions were not reflected in children's nutritional status were increasingly interpreted as official complacency. Criticisms levelled against the Board of Education were based on the discoveries of the Newer Knowledge of Nutrition, according to which children whose diets failed to reach a given standard were by definition malnourished. (The arguments which were presented are set out in greater detail in Chapter 4 Section 4.5). Public debate involving outspoken criticism of the administration (88) led to an increasingly firm insistence from the Board that its Medical Officers should not to deviate from the traditional criteria for nutritional assessment (89) and the CMO's reports of the 1930s are characterised by the repetition of definitions and directives which first appeared in the pre-1914 period (90). In 1935, this provoked an extremely strong letter from Mellanby to MacNalty in which MacNalty was advised to review his Department's approach to nutrition in accordance with modern scientific knowledge. Mellanby argued that:

"Malnutrition to you appears to mean the condition of gross underfeeding, marked ill development and real ill health ...What I suggest it should mean is 'any condition where present knowledge indicates that better physical health and development of the individual would have been obtained had feeding been along different lines" (91).

Using the same criteria as Mellanby, Boyd Orr took as his standard for health

"a state of physical well being such that no improvement can be effected by a change in diet" (92).

Many similar statements can be found in the writings of

nutritionists commenting on the health of the nation during the 1930s and a full account of these views appears in Chapter IV. Their definitions of health and malnutrition were based on exclusively dietary criteria; environmental factors were excluded from the analysis. Indeed, it has been shown that environmental improvement in the shape of better housing was seen by Fletcher to be positively detrimental to the cause of preventive medicine and the improvement of national health since this deflected public funds away from the 'physiologically' more important nutrition question.

3.1.5.5. The Cost of Change: Finance and Physiology

The question of nutritional assessment was closely associated with the debate about the adequacy of supplementary feeding arrangements. Although Local Education Authorities (LEAs) were not obliged by statute to feed all poor children who were diagnosed as malnourished, they were strongly encouraged to do so by the Board of Education (93) and the two issues were often linked in the debate about malnutrition. The Board recognised that a more liberal approach to the diagnosis of malnutrition would have required a vast increase in its feeding grants to LEAs (94); thus by maintaining the old criteria for nutritional assessment, it kept the obligation of the State to provide meals for school children within 'reasonable' bounds.

For this reason, the Board stressed throughout the inter-war period that supplementary feeding was not intended as a form of poor relief: other departments were responsible for this (95) and it frequently invoked the Education (Provision of Meals Acts) of 1906 and 1921, according to which LEAs were only empowered to feed children who, "through lack of nourishment" were unable to take advantage of the education offered them. Not all poor children, the Board claimed, could be classified as malnourished in this sense; it was argued that other factors-notably

maternal efficiency - in many cases offset the effects of poverty. However, the Board's refusal to feed on an economic basis met with mounting criticism from the public health movement. For example, a delegation from the Society of Medical Officers of Health accused the Board in 1935 of flouting the basic principals of preventive medicine by refusing to feed on an income basis (96) and in 1938 Hopkins described the Board's approach to supplementary feeding as 'illogical and grudging' for the same reason (97). The source of many of these criticisms can be traced to the Ministry of Health's own expert Advisory Committee on Nutrition, which was convened in 1931. The deliberations of this Committee are the subject of the following Section.

CHAPTER 3 - PART II

3.2. The Ministry of Health's First Advisory Committee on Nutrition, 1931-1934.

3.2.1. The Convening of the Advisory Committee

In October 1930 the Labour Minister of Health, Arthur Greenwood, agreed to convene an Advisory Committee on Nutrition. This Committee was set up by W.A. Robinson, Secretary to the Minister of Health, and its purpose was to combat the adverse effects of unemployment on health by dietary education. By the late 1920s the Ministry took the view that a low consumption of the "vitamin rich" protective foods, especially among the unemployed, represented a major public health problem and confidential surveys of the state of health in the South Wales coalfields reported that prolonged economic recession had brought certain communities to the brink of a nutritional catastrophe (1). It was argued that by improving the quality of the diet in these areas of high unemployment, susceptibility to disease could be lowered and resistance to the 'ravages' of disease improved (2).

Although the perceived problem of a poor quality diet among the unemployed prompted the Ministry to 'take action on the nutrition question' it was also argued at this time that freely chosen diets were not necessarily adequate for health and that 'optimum nutrition' could only be ensured on the basis of expert advice; it was widely believed that an "incorrect diet", that is one which was lacking in the required levels of nutrients, lowered both vitality and resistance to disease irrespective of social class (3). Thus, Robinson explained to the Minister of Health that the object of the proposed Nutrition Committee was to "secure adjustments in our dietaries and way of living" in accordance with the discoveries of the new science of nutrition. Changes of this nature were always a slow process, he told the Labour Minister in "an old and conservative country like this" (4). Robinson

told Fletcher that his specific aim in convening the Committee to disseminate knowledge and improve practice with respect to food and that the Ministry wanted to know:

"what are the new factors in dietetics resulting from your (MRC) work which we should try to get into people's ordinary food in order of health importance?".

It intended to pass on this information to the people

"through the public health machine, health visitors and all the rest of it, press propaganda etc" (5).

This was precisely the course Fletcher had been urging the Ministry to adopt since 1921, and he was outraged that a decade later it should see fit to waste the time of the nation's leading experimental scientists on a committee which, he maintained ought to consist of

"really enthusiastic Medical Officers, lady visitors and the like".

The convening of a committee of experts amounted, in his view, to a window dressing exercise on Newman's part, to deflect attention away from his earlier neglect of the problem (6).

Robinson's belief that there was a relationship between dietary quality and health was apparently shared by his senior administrators. Thus the CMO, Newman, was in favour of a Committee to

"tackle this problem of nutrition which we are all agreed is of outstanding importance to the health of the nation" (7).

However, their concern about the national diet was not incompatible with the Ministry's underlying nutritional philosophy - that food alone could not offset the effects of a bad domestic environment.

3.2.2. Membership and Terms of Reference

The initial membership of the Committee included F.G. Hopkins and Edward Mellanby whose work in the fields of vitamin biochemistry and physiology has been mentioned; E.P. Cathcart, Professor of Physiology at the University of Glasgow and Chairman of the MRC's Nutrition

Committee, V.H. Mottram, Professor of Physiology at King's College for Women and a former pupil of Fletcher's, Dr Buchan, President of the Society of Medical Officers of Health, and Miss J. Lindsay, a domestic economist. The Committee was chaired by Major Greenwood, Professor of Epidemiology and Vital Statistics at the London School of Hygiene and Tropical Medicine who was a member of the MRC's Nutrition Committee and had been associated with the MRC's work on quantitative aspects of human nutrition since the pre 1918 period.

At the inaugural meeting, W.A. Robinson explained on behalf of the Minister that the objective of the committee, was to provide:

"authoritative information on food questions which could be passed on to the public through suitable publicity channels"

as the Ministry's advice on diet would, he explained, be

"enhanced when it is known that it emanates from a body of specialists familiar with modern advances in the knowledge of nutrition" (8).

This reflected the Ministry's concern about the more spectacular claims of the manufacturers of Vitamin enriched proprietary foods. The public, which had shown enormous interest in the question of food and health, was being bombarded with conflicting advice and information and it was felt that they needed authoritative advice to dispel the new myths and promote sound dietary practice.

There was a broad consensus among both scientists and administrators over the appropriate strategy and the Committee undertook to advise the Ministry's Food Section on the "physiological characteristics of food and its relation to nutrition", on dietaries for public institutions and on public statements and propaganda relating to the choice of food (9). At an early date, the Committee recorded its agreement over the root cause of poor nutrition. This, it was stated, was due to the ignorance, laziness and lack of thrift of the British housewife (10).

It set its own agenda and identified the following priorities:

- to offer food advice to the public
- to provide information on the preparation of food and cooking
- to suggest dietaries for poor law institutions
- to produce short memoranda on the prevention of rickets and dental caries and general infections for Maternal and Child Welfare workers (11).

Although this first Advisory Committee failed to meet its initial brief, it succeeded in raising a political storm, embarrassing the Ministry of Health and challenging the integrity of the British Medical Association.

3.2.3. Raising the Storm: the setting of dietary standards

Early in 1932, the Committee produced two documents, THE CRITICISM AND IMPROVEMENT OF DIETS and DIETS FOR CHILDREN IN POOR LAW HOMES (12). These were intended as general guidelines for public health officers responsible for institutional feeding and for use in dietary education among the population as a whole. It deliberately did not put a price on the diets recommended in these documents since it was felt that this would raise matters which were outside its terms of reference:

"in view of its wide and possibly embarrassing repercussions the Committee had agreed to restrict themselves to general principles" (13).

Nevertheless, E P Cathcart, who was the Committee's most persistent dissenting voice, did anticipate political repercussions. Cathcart pointed out that dietary standards, which included for children a recommended daily intake of:

"1 pint of milk, cheese 'freely' partaken, 1 orange or 1 tomato or helping of raw salad, 1 oz butter or vitaminised margarine, fat fish once a week 1/2 teaspoon cod liver oil"

was beyond the means of the average working man. He feared that if these recommendations were embodied in an official document they might be

"seized upon by the transitional beneficiaries and others as a yardstick to measure what their allowances might be" (14).

Officials discounted Cathcart's anxiety on the grounds that the document was merely

"suggestive and not intended in any sense to standardise dietetic practice" (15).

However the document did have far reaching effects which were to alter public perceptions of the very nature and extent of malnutrition. The first Advisory Committee on Nutrition demonstrated that nutrition science could be a potent political weapon when it was translated into official statements about dietary adequacy and dietary requirements. Despite its members' explicit attempts to confine themselves to purely scientific aspects of the problem, the publication of THE CRITICISM AND IMPROVEMENT OF DIETS and DIETS FOR CHILDREN IN POOR LAW HOMES drew the Committee inexorably into a political minefield. Their dietary guidelines provided standards against which nutritional adequacy could be measured and so inspired a series of inquiries which sought to define on the basis of family income, the extent of malnutrition in the UK. This approach rested on the premise that if the intake of specific nutrients fell below prescribed levels, the organism was necessarily malnourished. The Cost of a "Physiologically Adequate Diet" The first of a series of major inquiries into the cost of a 'physiologically adequate diet' was published by the WEEK END REVIEW in April 1933 under the headline 'Hungry England'. This report was written by a committee of inquiry which was chaired by A.L. Bowley, Professor of Statistics, at the London School of Economics, and included W.H. Mottram, Mary Nicholson of the Octavia Hill Housing Trust and R.C. Davidson, an authority on unemployment and Public Assistance. Mottram was a prominent figure on public platforms throughout the 1930s and was a particularly active member of the Committee Against Malnutrition which was founded in 1934 (See Section 4.2).

The 'Hungry England' report calculated the cost of a physiologically adequate diet, as defined by the Committee, at current London and provincial prices (16). This report was described by Carnwarth, a Medical Officer in the Ministry's Food Division as

"admirable...an attempt to express in practical terms (in terms of quantities of the common foods and their prices) the principals of the Ministry's memorandum" (17).

However, Robinson recognised the importance of the report in the light of current discussions over proposed national scales of public assistance: in a minute to the Minister he wrote that the Week End Review and other press organs had

"bestirred themselves in the production of dietaries suitable for the workers and the cost of them" (18).

He suggested to the Food Division that the Advisory Committee should consider the report since

"We shall be called on to express pretty clear and definite opinions as to the soundness or otherwise of such conclusions as those of 'Hungry England' and I hope we shall have our preparations ready" (19).

The Committee was therefore requested to

"state what is the sum of money necessary for the adequate nutrition of adults and children of different ages. Does the Report in fact give a basis for a sound and reasonable calculation for administrative purposes?" (20).

The responses which this request prompted reflect the polarisation of opinion within the Committee on fundamental matters of principle.

Cathcart, in a written reply, stated that

"On account of (1) the widely varying degree of housewifely intelligence and (2) the...differences in food customs...it is impossible to lay down a standard diet for Britain....shortage of housewifely intelligence (bad cooking, bad marketing, bad household economy) plays a bigger part than shortage of cash in the majority of cases of malnutrition."

He added that

"The problem we are up against is not a simple nutritional one but a tough psycho-sociological conundrum which no mild enactments from Whitehall could resolve" (21).

Mellanby, on the other hand, fully endorsed the approach adopted by the Committee and recommended that dietary standards of a more comprehensive nature should now be agreed.

Mellanby was to play a key role on subsequent committees which were convened during the course of the 1930s to set nutrient requirements and dietary standards: he was a member of the special Committee on Nutrition set up by the Minister of Health in 1934 to settle a dispute with the BMA over protein and calorie requirements; in 1935 he chaired the League of Nation's Committee on the Physiological Bases of Nutrition and he was a member of the 1936 Interdepartmental Advisory Committee on Nutrition which he had proposed to the Cabinet's Economic Advisory Committee.

In response to the WEEK END REVIEW's report, he advised that

"it would be well to set down specifically what, from a physiological point of view, is considered an adequate diet for different ages and what would be the estimated cost" (22).

Mellanby and Mottram suggested that the cost of feeding infants set out in the report was artificially low since breast feeding had been assumed. They also recommended higher milk allowances for children under 14 and presented schedules of 'adequate' diets (23).

Despite these differences, the appearance of agreement among experts was sustained. In its official response to the Ministry's enquiry, the Committee reported that

"Professor Bowley's committee have based their calculations on physiologically adequate diets and see no reason to dissent from the financial calculations drawn" (24).

3.2.4. The BMA Report

In November 1933, seven months after the Hungry England article had appeared, the BMA published a report which was to become a touchstone in the nutrition debate (25). The approach adopted by the BMA study was

not unlike that which had been used in the 'Hungry England' investigation; it defined an adequate diet in terms of nutritional requirements and proceeded to demonstrate the cost of meeting these requirements in families of various sizes and with children of various ages. However this report, which endorsed the view that that families whose weekly expenditure on food fell below approximately 5s 11d per head per week were malnourished, had a far greater impact than the Hungry England inquiry; when such an assertion was made by the BMA it obviously carried a great deal more weight than a similar statement made in a left wing periodical. The report also raised a storm because, for reasons explained fully in Section 3.2.6., its authors broke ranks and set protein and calorie requirements at higher levels than had been set by the Ministry's Advisory Committee in THE CRITICISM AND IMPROVEMENT OF DIETS. This provoked an extremely acrimonious correspondence, carried out in the columns of the TIMES in January 1934, between Greenwood, Chairman of the Advisory Committee and Sir Henry Brackenbury, Chairman of the BMA. The public nature of the dispute was potentially extremely damaging for the medical profession and it caused the Minister of Health, Hilton Young, to appeal personally to members of the Advisory Committee to draw up a compromise agreement with the BMA Committee; this was considered necessary in the interests of professional credibility (26).

Because it was issued by an authoritative committee of the BMA, the report sent shock waves across the entire political spectrum. The Labour Party used the report to launch an attack on government welfare policy, which it outlined in a Party document, 'Notes for Speakers' (27). This document pointed out that after food expenditure at BMA levels had been deducted from public assistance rates only 6s 2d was left for other essential items and concluded that

"Independent medical testimony is now available proving that the present scales of unemployment pay are inadequate to meet the bare minimum food requirements of an average unemployed family".

In 1931, the Labour Government had resigned over the issue of public expenditure cuts demanded by the May Committee. The May report raised fundamental doubts over the soundness of the Labour Party's financial management and called for substantial cuts in public spending which included a reduction in unemployment benefit (28). These cuts were implemented by the subsequent National Government. Not surprisingly, therefore, the Labour Party document, which touched on a highly sensitive political nerve, rapidly found its way to the Secretary of the Minister of Health, who alerted the Minister of the

"use for political purposes which the Labour Party are making of the BMA committee's report" (29).

The immediate effect of the Report was indeed to sharpen the debate about the impact of mass unemployment on national health. Shortly after its publication, G Shakespeare, a government front bench spokesman, maintained in an address to the Society of the Chemical Industry that there had been

"no general increase in physical impairment, or in sickness, or in mortality, as a result of the economic depression or unemployment".

Defending the record of the Ministry, he asserted that this could be attributed to the

"efficiency of our public health services, our schemes of national health and unemployment insurance and the growing attention paid to the treatment, care and nourishment of children of tender years" (30).

Questions were nevertheless raised the House of Commons concerning the adequacy of allowances for the unemployed, in the light of the BMA report (31). This new political dimension in the nutrition debate caused Major Greenwood, in an offer of resignation prompted by the controversy to reflect that he should have considered earlier that

"political capital might be made out of this field of scientific enquiry" (32).

After the appearance of the BMA report, official attitudes to the malnutrition question came under increasing attack. In the absence of clinical evidence of widespread malnutrition it was argued that hidden hunger and marginal malnutrition was prevalent among groups which could not afford a 'physiologically adequate' diet. Similarly the Board of Education's malnutrition return, the reliability of which had already been questioned, was further undermined as demands for a more sensitive and objective method of assessing nutritional status were made, issues are that discussed in Chapter 4.

3.2.5. The Making of the Nutrition Scandal

Despite the Advisory Committee's objections, the BMA refused to retract its report which was, at least until the appearance of Boyd Orr's study FOOD HEALTH AND INCOME in 1936, the most frequently quoted document in the debate about the impact of unemployment on the health of the nation and in response to the report, a number of campaigning organisations were established which aimed to raise public awareness of the problem of 'widespread malnutrition' (See Chapter IV). In view of its very great political impact, it is ironic that, according to enquiries made by the Ministry, the BMA document had been read by very few members of the Committee which produced it (33). R.M.F. Picken of the Welsh National School of Medicine, who sat on the Committee, maintained that they had been "unable to control the experts on their own Committee" and that, since few of the members were physiologists, no real debate had taken place over the controversial scientific aspects of the Report (34). The Advisory Committee, which had set lower standards for Calorie and Protein intakes than the BMA Committee was understandably scathing in its appraisal of the document. It was, they concluded

"highly unscientific...unconvincing...valueless as a scientific document...not in conformity with the evidence" (35).

3.2.6. Spiralling Standards

Two members of the Advisory Committee, Mottram and Buchan, had signed the BMA document, yet neither resigned from the Advisory Committee. Mottram's explanation for his support of the BMA's higher requirements illustrates a problem fundamental to the setting of dietary standards. He argued that, since the Advisory Committee's standards had been set on the basis of Cathcart's dietary surveys, which included a disproportionate number of unemployed families and since all surveys showed that protein intakes rose with income, the real 'requirement' for protein was probably closer to the freely chosen diet than the standard derived from a non representative sample (36). In fact neither side could support its case with objective evidence; consequently arguments about the 'correct' requirement for protein reflected political as much as scientific points of view.

The standards adopted by the BMA Committee were rejected by Cathcart, who maintained that

"large families were being reared to a healthy physical and mental maturity on a diet which is hopelessly inadequate according to BMA standards" (37)

and Carnwarth noted that there seemed to be an irresistible tendency for Nutrition committees to inflate dietary requirements. He observed that if the BMA scale was to be accepted it was unlikely whether there was, or ever had been, enough food in the country to satisfy the standard (38). This argument might equally have been applied to the 'world food crisis' which dominated the nutrition debate in the aftermath of World War II. Again anticipating post war preoccupations, Carnwarth commented that

"no doubt the next committee that sits will boost the figure a bit higher 'just for the fun of the thing'" (39).

Greenwood, too, was concerned by developments which had taken place under his Chairmanship of the Advisory Committee. After this was

disbanded in 1934 (pending the new Interdepartmental Advisory Committee on Nutrition), he confided in Robinson that he thought that the practice of fixing dietary norms or standards was a foolish one - even though he had presided over the institutionalisation of this procedure. Greenwood maintained that

"The needs of the living organism are infinitely variable and wherever we fix [the standard], it will be too little for some."

In the light of the BMA report, he predicted that by fixing the norm so high (sic) that the number going short was 'vanishingly small', they would ultimately be faced by headlines reading "Ministry pampers the workshy" (40). (Greenwood's argument was apparently that high normative standards for diets would be interpreted as advocacy of an overgenerous standard of living for the unemployed.)

He also found himself increasingly isolated from the views of the majority in discussions of the practical applications of nutrition science and revealed that the compromise strategy adopted to settle the BMA controversy was apparently typical of the practice which had been adopted on many occasions in the course of the Advisory Committee's own deliberations. Thus, Greenwood wrote to Hudson, the Secretary of the Committee that

"the democratic method of settling matters of dispute by voting does not seem applicable to scientific questions..there is not at present sufficient agreement among those whose experience gives them the right of judgement for it to be possible to advise the Ministry on many of the problems submitted" (41).

Greenwood had identified a problem which continues to beset the expert advisory committee system. If government wishes a clear and positive policy it must restrict the committee's membership to experts who agree in their interpretation of the evidence. If it wants information on the current state of knowledge, it must include experts whose interpretation of the evidence will almost certainly differ.

3.2.7. Matters Arising: Measuring Malnutrition

Having raised a storm over dietary standards and the prevalence of malnutrition in the community, the Ministry pressed the Committee to produce

"a more definite and objective connotation for the term 'Malnutrition'" (42).

The Board of Education's annual nutrition returns had become the object of considerable public interest during the period of mass unemployment and the discovery of enormous discrepancies between the reported rate of malnutrition in adjacent industrial towns undermined confidence in the government's regular assurances that the economic depression was not taking a toll on child health. (This issue is discussed in Chapter 4.)

The Committee was could not, however, reach agreement over the best method of measuring malnutrition. Mellanby maintained that a sufficiently skilled observer could always detect "the concrete morbid stigmata of 'malnutrition'" and proposed further research to identify these; he planned to bring together experienced researchers familiar with nutritional diseases such as rickets and anaemia for this purpose. Buchan, a practicing Medical Officer, held a different view.

Malnutrition to him meant "some bodily state short of actual disease" (43); it was a condition which necessarily preceded the appearance of 'morbid stigmata'. The distinction was an important one, as Buchan argued in a delegation from the Society of Medical Officers of Health which approached the Board of Education in 1935. This delegation objected to regulations dictating that school milk could not be provided free to children on economic grounds and maintained that at low levels of income children must be assumed to be inadequately nourished (44).

Whilst the Committee recognised the shortcomings of the subjective clinical method of nutritional assessment, members could not agree on an alternative. Thus, they accepted the findings of an official study

which condemned the use of height and weight indices as a basis for assessing nutritional status (45) for reasons that are explained in Section 4.2.4. Although it was agreed that anthropometry provided a useful mass indicator, the Committee asserted that this was not helpful in the assessment of individuals (46). The study of anthropometry was largely neglected by nutrition scientists during the inter-war period, and the investigation of anthropometric indices was confined to a small number of School Medical Officers; it was only during the 1960s that the potential of anthropometry as a method of assessing nutritional status was recognised by nutritionists (47). The Advisory Committee was unable to provide the Ministry with the yardstick it needed to settle the question of nutritional assessment, and the matter was left, pending further investigations along the lines proposed by Mellanby. These investigations did not take place until the Second World War, when funds became available for massive clinical surveys.

3.2.8. Field Studies in Nutrition

The measurement of malnutrition was not the only question which the Committee failed to resolve. It also grappled with the problem of devising a field study which would provide irrefutable evidence of the benefits of supplementary feeding and so force the government's hand on nutritional intervention (48). Mellanby pressed hard for such a study. He argued in a paper 'Nutrition and Child Bearing' that the time had arrived for the

"practical application of certain discoveries made in the course of experimental work" (49),

and proposed a large scale field experiment in order to demonstrate

"the importance of a proper diet in pregnancy and lactation".

Various schemes for carrying out this experiment were suggested which, although they appear bizarre by modern standards, demonstrate the problems involved in the transition from laboratory to epidemiological

proof. These difficulties are now familiar to nutrition scientists. However, in 1934 epidemiology was still in its very early stages and an experimental methodology for human populations had not yet been developed. For example it was recognised that a basic difficulty in conducting a feeding trial among free living human populations was to ensure that the control and experimental groups ate what they ought to. It was suggested that this difficulty might be overcome by carrying out the trial in a small community where all the workers were engaged in the same industry and where factory owners exerted a strong paternalistic influence over the population; the Nottinghamshire shoe manufacturing towns were suggested for this purpose (50).

Mellanby's field experiment remained another piece of unfinished business. However, throughout the 1930s he continued to press the case for universal Vitamin dosing in Maternal and Child Welfare practice. The prophylactic use of cold liver oil therefore appeared among the recommendations of the League of Nation's Report on the PHYSIOLOGICAL BASIS OF NUTRITION, published in 1935, and in the Interdepartmental Advisory Committee on Nutrition's First Report, published in 1936.

3.2.9. Divided Councils

The questions of measuring malnutrition and planning a controlled field experiment illustrate practical problems in the application of nutrition science which have not yet been fully resolved. However, the Committee was also divided over more fundamental matters than these, in spite of the fact that it had had been selected with the specific purpose of producing a "positive policy" (51). In the following section, underlying philosophical differences between the older knowledge of nutrition and the new are illustrated through the debates which took place in the Committee and in the public statements of E.P. Cathcart who was one of the most outspoken critics of the Newer Knowledge of

Nutrition during the inter war period. In the deliberations of the Committee, the main points of contention were as follows:

1. How extensive were qualitative deficiencies in the national diet? Mellanby thought they were widespread, and advocated a policy of mass dosing in order to supplement perceived shortages of fat soluble vitamins (52). Cathcart, who represented the 'older knowledge of nutrition', argued that generations of healthy Scots had been raised on diets which were, according to modern standards, deficient in almost every respect. On the 'bedrock of ordinary experience' he maintained that the modern standards must therefore be excessive (53).

2. Was the publication of official dietary guidelines a useful exercise? Mellanby saw this as one of the Committee's most important functions (54). Cathcart argued that guidelines were of no practical value, since they did nothing to change actual dietary habits; moreover, he argued, "houswifely intelligence", rather than the purchase of particular foods, was the factor which determined differences in the standard of nutrition between households (55).

The first indication of these differences emerged towards the end of 1931. Fletcher had written to the Committee's Chairman, Major Greenwood, accusing the Ministry of failing to implement discoveries in nutrition science. Greenwood doubted that any discoveries had been made which would reduce inequalities in health between social classes (56). However, he suggested that a document should be prepared by the Committee in answer to the question:

"What discoveries had been made and not acted upon that might reasonably have been?"

V.H. Mottram produced a detailed memorandum in which he maintained that there was conclusive evidence that increased consumption of Vitamins A and D would banish rickets and dental disease and that increasing the

production and supply of foods containing these vitamins, together with the education of the public in their consumption represented a "pressing task of statesmanship" (57). Hopkins and Mellanby concurred with this view; Cathcart did not. However, a set of proposals were drawn up on the basis of Mottram's paper which were ultimately embodied in the Ministry of Health Circular 1290. Circular 1290 recommended prophylactic dosing of Vitamin D; the use of iron ammonium citrate in the treatment of anaemia and the use of Vitamin C supplements for artificially fed infants. It also stressed the crucial role of milk to make good qualitative deficiencies in the diet. Cathcart, who had never fully accepted the dietary explanation for the aetiology of rickets (58) registered his dissent from the recommendations concerning Vitamin D dosing, arguing that the evidence concerning the relationship between Vitamin D and dental caries and rickets was not conclusive. Furthermore, he maintained that the Ministry was not justified in recommending the the issue of Ergosterol at Infant and Child Welfare clinics on the grounds that

"we know too little about the possible ill effects of long term overdosing" (59).

The incidence of hypercalcaemia during World War II demonstrates the wisdom of Cathcart's reservation. Indeed there was already experimental evidence suggesting the possible toxicity of relatively small doses of Vitamin D and by the outbreak of the War evidence of the dangers of widespread fortification of infant foods combined with a policy of prophylactic dosing were conclusive (60).

However, the view which in 1932 called for caution was not on the ascendant and the opposing position, which called for a positive nutrition policy to eradicate the problems of rickets and dental disease, was increasingly supported by the public health movement. The necessary steps were described by Mellanby as follows:

"Every child under 5 years of age should have 1 pint of milk per day, children over that age should have at least 1/2 a pint of milk per day and ...all children should be given 1 or 2 teaspoon-fulls of cod liver oil daily and .. the diet of pregnant and lactating mothers should be controlled" (61).

The divided councils within the Committee were noted by the Ministry; Carnwath, who summarised the differences between the two camps admitted that he favoured the 'newer school' which assumed a widespread deficiency in dietary quality, at least during pregnancy and lactation (62) and it was on this assumption that war time welfare feeding policies were based. The debates of the pre war Nutrition committees therefore prepared the way for the massive programme of nutritional supplementation which was undertaken when the political leadership changed in 1940 and Winston Churchill assumed office.

Little was heard of the opposition view during the war years. However, in the course of the 1930s, Cathcart continued to express his disagreement with the teachings of the Newer Knowledge of Nutrition on public platforms as well as on panels of experts. For example, in his London School of Hygiene and Tropical Medicine Lectures delivered in 1931, he presented a lucid exposition of the case against the public health inferences which were being drawn from experimental work on the micronutrients. Thus, at a time when nutrition science was increasingly concerned with the precise definition of dietary requirements, Cathcart argued that, although it was a truism to say that

"the welfare of the race, its health, energy, resistance to disease, etc., is largely a function of its state of nutrition... Fortunately... the organism is not too delicately balanced where food is concerned.." (63).

In defiance of the view which prevailed at the MRC, and was described in Chapter 2, he asserted that

"It is a wise dispensation of nature that the foodstuffs are so compounded that they represent a mixture of the essential materials"

and anticipating the arguments that were to dominate the nutrition

debate up to the outbreak of the War he maintained that

"It is only too commonly believed that it is an easy problem to devise a system of adequate dietaries suited to all purses on which a man may live and flourish."

Cathcart criticised his fellow scientists for making premature statements about the public health applications of the Newer Knowledge of Nutrition and claimed that there had been a loss of balance following Hopkins's postulate of necessary accessory substances. He abhorred the view held by 'certain investigators' that

"the only slogan necessary is 'Take care of the vitamins and the diet will look after itself'"

and held firmly to the belief that the average everyday diet was not so deficient as to produce

"the astonishing results which one might deduce from experimental work on pure diets".

However, Cathcart did not reject all aspects of contemporary nutrition research. For example, he agreed that there was probably a considerable range of variability in individual vitamin requirements and that dietary needs changed according to the individual's physiological state. His argument was not with the experimental evidence, but with the inferences drawn from these:

"It is not that the experiments in themselves are wrong", he asserted,

"it is the deductions drawn, the stress laid upon them, that so often are doubtful" (64).

Cathcart frequently drew attention to the absence of clinical evidence of vitamin deficiency disease. This lack of evidence of dietary deficiency during the period of mass unemployment prompted Greenwood, Chairman of the Advisory Committee on Nutrition, to submit an official memorandum in which he noted that

"the apparent contradiction between the economic facts and the physiological showings in this matter of nutrition"

had caused both himself and Cathcart to query

"whether the teaching of scientific men on standards of nutrition, minimum energy requirements etc, well founded though they seem to be, may not after all be erroneous" (65).

Cathcart may have encouraged doubt and dissent in some quarters; for example shortly before his London School of Hygiene and Tropical Medicine Lectures were published, an editorial in the MEDICAL OFFICER observed that

"with all our science and experiences we ...are unable to say wherein lies the difference between diets which are sufficient and insufficient, between those which keep us health and those which render us vulnerable to pestilence" (66).

However, he remained a minority voice on the Ministry of Health's Nutrition Committee, and Mellanby was happy to accept his resignation from the Chair of the MRC's Nutrition Committee in December 1933 (67). The two men stood at opposite poles in the debate about the nature and extent of dietary deficiency.

CHAPTER 4 - PART I

THEORY INTO PRACTICE

4.1. Nutrition and the Public Health Profession, 1919-1929

The public health applications of the new science of nutrition have so far been discussed in relation to the views of laboratory scientists, the medical research establishment and the public health administration. This Chapter looks at their impact on the public health movement and in particular on the practice and opinions of Medical Officers of Health. The application of nutrition science in public health practice falls into two distinct periods during the inter war years: 1919-1929 and 1930-1939. During the 1920s there was a consensus between the public health administration and Medical Officers of Health over the inferences of the Newer Knowledge of Nutrition for public health practice. The 1930s was by contrast a period of controversy and re-appraisal, when public health practitioners came into open conflict with public health administrators.

It has been shown that from 1909 when the Board of Education's annual reports first appeared, until the outbreak of World War II, the public health administration upheld the view that nutrition was a 'complex process' influenced by a variety of social and environmental factors which included overcrowding, fresh air, exercise and sleep in addition to food. It was argued that good domestic nurture was sufficient to offset the effects of an unfavourable environment, and that the nation therefore required efficient mothers to keep children well rested, well exercised and well fed. During the 1920s the MRC published a series of studies which both reinforced this view and at the same time strengthened the case for nutrition education as a cure for many of the problems of poor physical status and general ill health in the community. The consensus over the nutrition question which characterised the 1920s broke down during the economic recession of the

early 1930s; in the period of mass unemployment, which coincided with the appearance of officially sanctioned dietary standards, many Medical Officers urged the administration to adopt new criteria for the definition and assessment of nutrition. The administration, however, held tenaciously to existing views and re-affirmed its commitment to the 'older' knowledge of nutrition, for which Paton's and Cathcart's Glasgow School of Physiology provided a theoretical basis.

4.1.1. Malnutrition: A Social and Environmental Problem

In the early years of the School Medical Service, the Chief Medical Officer, Sir George Newman, encouraged Medical Officers to undertake studies into the aetiology of malnutrition. In 1911 Newman wrote that this was

"One of the most fruitful fields for enquiry and investigation... likely to yield results of great practical value" (1).

Studies carried out in response to this appeal stressed that the nutrition problem was essentially one of bad domestic management and ignorance, rather than poverty. They appeared to confirm the view of the Committee on Physical Deterioration that a "great scheme of social education" (2) was required to cure the 'evils of physical degeneracy' by "raising the standard of domestic competence and home life" (3).

For example, a study carried out in 1910 by Dr Badger of Wolverhampton, which is typical of these School Medical Officers' reports, listed factors such as "neglect, dirt or drink", "defective feeding due to poverty or ignorance" and "overwork or insufficient sleep" as the cause of malnutrition in over half the children studied; factors associated with disease made up the rest of his list. Nevertheless, Badger concluded from his 'statistical' study that:

"malnutrition is a condition that affects all classes; that it has a close association with disease; that much of it, being associated with ignorance and defective hygiene, is due to preventable causes; that poverty operates powerfully in its production, but that in view of the number and diversity of the factors producing malnutrition, the former may not occupy per se, the prominent position that is sometimes popularly assigned to it" (4).

In these and similar studies an association between poverty, malnutrition and disease was either ignored or described as 'preventable'. Thus, greater maternal effort rather than the alleviation of poverty was prescribed as the cure (5).

Two MRC studies, POVERTY, NUTRITION AND GROWTH (6) and a REPORT ON THE NUTRITION OF MINERS AND THEIR FAMILIES (7), which were carried out during the early 1920s, seemed to corroborate this view and at the same time put it on a more rigorous scientific basis. Consequently, the debate about the cause of malnutrition, the social aetiology of which was described by Newman in 1910 as a "baffling" problem (8), appeared by the mid 1920s to be settled. POVERTY NUTRITION AND GROWTH was an extensive inquiry into factors influencing child health. It was undertaken by the MRC's Child Life Committee in Scotland (9) and reached the unequivocal conclusion that "maternal efficiency" was the chief causal factor in the aetiology of malnutrition. The report on the NUTRITION OF MINERS AND THEIR FAMILIES, carried out during the same period, seemed to confirm this view. It claimed that thrift, diligence, intelligence and efficiency were required to achieve a 'physiologically adequate' diet at low levels of income (10). At this time, both scientists and public health workers agreed that the poor were in urgent need of education in nutrition and domestic management; the notion that maternal inefficiency was a key factor in the aetiology of malnutrition was fully compatible with the ideas of the proponents of the Newer Knowledge of Nutrition.

4.1.1.1. Paton and the Concept of Maternal Inefficiency

POVERTY, NUTRITION AND GROWTH was the work of D. Noel Paton and Leonard Findlay, a Professor of Paediatrics. Paton has already been identified as a protagonist of the 'older knowledge of nutrition'. He had argued from the early years of the century that the main cause of poor nutrition was not poverty, but the carelessness and incompetence of working class housewives. For example, in a dietary survey carried out in Edinburgh in 1901 (11) he claimed that there had been a deterioration in the diet of the Scottish labouring classes, caused by the demise of porridge as a staple food and its replacement by bread and jam (12). The reason, he maintained, was that working class women were too lazy to cook the traditional dish; the importance of fuel costs and inadequate kitchen equipment in dictating this dietary change, were not considered.

In 1919 Paton and Findlay embarked on an MRC sponsored study of factors influencing the nutrition of poor children. Their aim was to set infant welfare practice on a sound footing (13); much contemporary practice was, they maintained, "mere groping in the dark" (14). The study sought to define those social and environmental factors which determined nutritional status. Their work was based on the premise that the nutritional process was a complex interplay of social and environmental factors and that the role of the physiologist was to evaluate the relative importance of factors which determined the outcome of this process (15).

In its investigation of the nutritional process, POVERTY NUTRITION AND GROWTH attempted to correlate output (assessed by the height and weight of children) with input (or environment). Environment was reduced to categories which included income, family size, food expenditure, overcrowding (air space per person) and "maternal efficiency" (16); this

last category was assessed on the basis of the cleanliness of the home, the mother's cooking skills and the general appearance of the children. Of course maternal efficiency was a notional variable whilst all the others had scalar values; simple correlations between the two were therefore statistically suspect. Efficiency was nevertheless included in the general analysis of results which involved the lengthy and arduous calculation of partial correlations. On the basis of this analysis, the study concluded that maternal efficiency was the most important factor in determining nutritional status (17) and in its final recommendations the report asserted that raising family income was not an appropriate means of dealing with the problem of poor nutrition (18). Despite these bold claims, the amount of variability explained by maternal efficiency was in fact trivial. If Paton's correlation coefficients are accepted, r^2 can be used as a predictor of variation explained by extra factors. On this basis, "inefficiency" explained none of the observed variation in growth of children over one year old, and at most accounted for 25% of the variability in growth of one group of boys aged 6-9 months (19). Nevertheless, the view that 'maternal efficiency' was the key factor in the determination of nutritional status was endorsed by Newman (20) and Paton continued to be quoted as an authority by the public health press as late as 1932 (21).

4.1.1.2. Maternal Inefficiency re calculated: The Nutrition of Miners and their Families

In 1922 the MRC's Committee on the Quantitative Problems of Human Nutrition selected coalmining communities as the subject for its first nutritional survey. The mining industry had been badly hit by the post war recession (22) and in 1921 had experienced a gruelling strike which left it with reduced rates of pay and continuing high levels of unemployment. In consequence, it was stated in the House of Commons that

"there is actual starvation existing in the mining districts of this country"

and that

"the vast mass of the mining population are in a state of semi starvation, them and their children" (23).

The Committee maintained that its aim was to collect observations on the "single issue of nutrition" and disclaimed any interest in economic aspects of the problem (24). However, its major conclusions were concerned with the question of family budgeting rather than with nutritional status.

I am aware of the theoretical objections that can be raised against 'retrospective' analysis of this kind. However, it is not my purpose to belittle the statistical achievements of the Committee with the advantages of hindsight and modern technology. Nor is it any purpose to criticise the moral and political assumptions of the Committee members. The Committee's aim was to apply modern scientific methods in an attempt to improve the well-being of an economically disadvantaged group and according to prevailing standards, they succeeded in this task. Given the extremely laborious nature of statistical computation before the advent of 'new technology', the Committee's approach to the analysis of data - for example its choice of variables for analysis - provides valuable insights into the social and moral assumptions about the kind of relationships they were likely to find. It is important that we should be aware that these were 'rational' although arbitrary choices since their analysis provided the scientific rationale which has formed a basis for much health education work both in the UK and in developing countries up to the present time. By using modern techniques it is possible to query the validity of these assumptions; it is this aspect of the process of re-analysis which is so important for the 'contemporary' historian. Statistical computation which would have involved many hours work can now be carried out in seconds. This means

that it is no longer necessary to confine statistical analysis to a narrow range of variables, selected on the assumption that they will give 'good' results (i.e. a high degree of correlation).

The Report addressed two issues; the composition and physiological value of the diet available to miners' families in different coalfields, and the physical status of the child population. Its criteria for assessing physical status were crude; the average heights and weights of children in mining communities were compared with the average for elementary school children in the same area. It was found that heights and weights fell below the average for the district in only one mining community (25) which of course implied that claims that miners' children were "starving" were inaccurate (or at least that they were no worse off).

[Using the methodology outlined in Chapter 6, it has been possible to assess the nutritional status of 14 children who appear as individuals in the report, according to current WHO standards. Although there was no evidence of acute malnutrition or 'wasting' there was, in common with other data collected in this thesis, a high level of stunting (ie height for age below the 3rd Centile of NCHS (1983)). Numbers are not large enough for meaningful statistical analysis, but the raw data are shown in Appendix I.]

Having dismissed the view that there was "actual starvation" in the coalfields, the Committee focussed on the efficiency of family budgeting. For example, it has been suggested that miners' wives who had been in domestic service might be better household managers than those who had not. Although numbers proved to be too small to undertake a statistical analysis, this hypothesis is indicative of the kind of relationships which the Committee believed could be usefully explored. Discrepancies were found in the "nutritional value for money" achieved

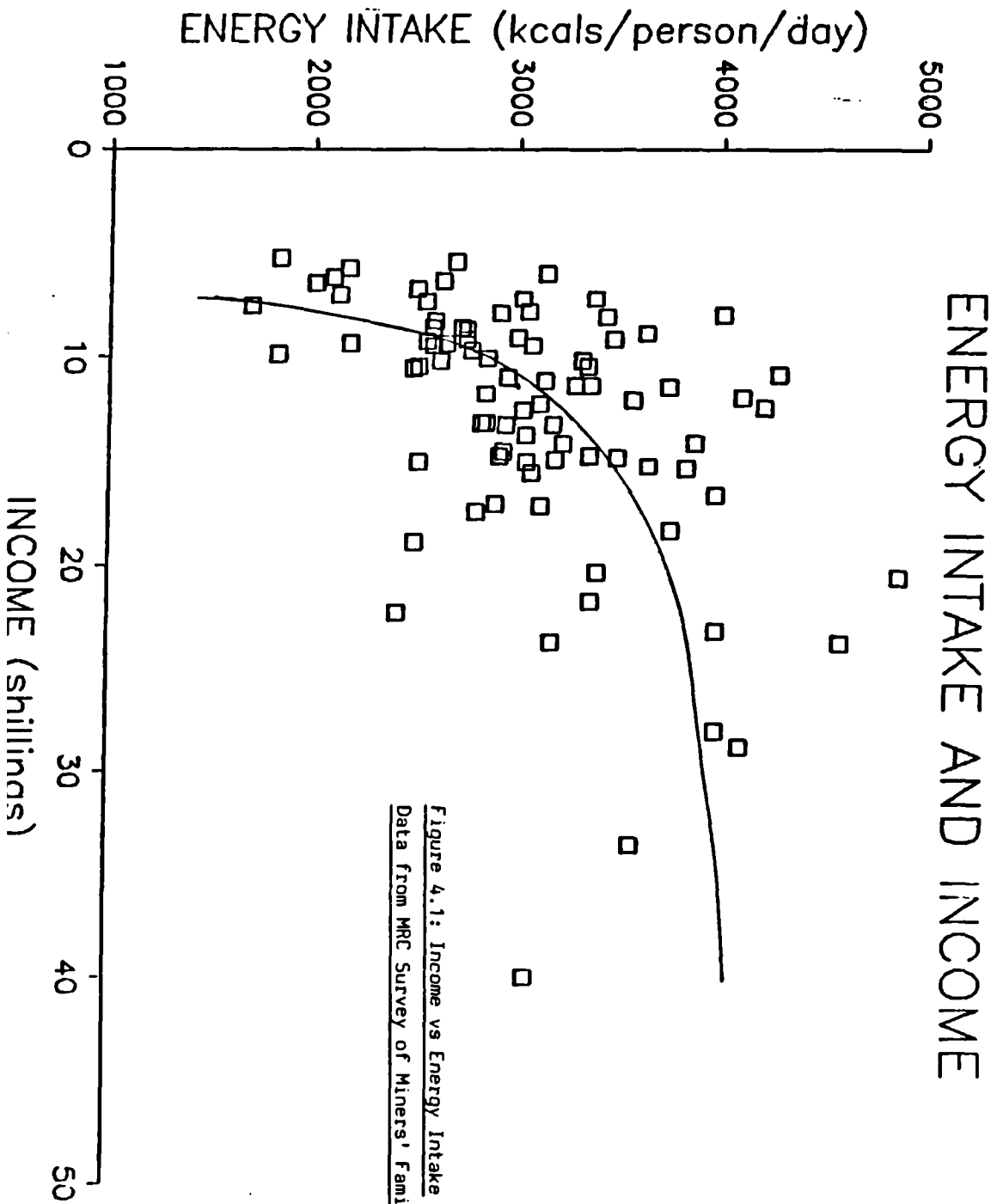
by miners' wives and an analysis of these discrepancies was undertaken (26). In order to evaluate the "adequacy of return for expenditure", linear regressions of a range of variables, which included calorie intake and calories purchased per penny on income, were made. (27). This was the same procedure used by Paton in POVERTY NUTRITION AND GROWTH and it produced correlations which appeared to support the hypothesis that more efficient marketing would increase the food intake of the poor and so the Committee concluded that "by better education and organisation more adequate physiological results can be obtained. By education we understand knowledge of the factors which ought to be taken account of in marketing" (28).

Shortly after the report was published this finding was singled out by Fletcher in a letter to the Chief Medical Officer as evidence of the need for

"a really big and campaign on this fundamental business of nutrition an the right use of cooking methods" (29).

These conclusions were in fact false, a product of the Committee's choice of linear regression analysis to evaluate the "adequacy of return for expenditure" when the actual relationships studied were curvilinear. For example, whilst linear regression assumed a constant relationship between differences in income and changes in food purchasing over the whole income range the data showed no such constancy. Thus, Figure 4.1 shows that at very low levels of income, energy intake changed rapidly with income but that above a threshold of 6s to 8s per head it changed only slowly, if at all, with changing income. Clearly, the relationship between income and calorie intake was not linear and linear regression analysis totally distorted the true relationship: the actual relationship was curvilinear. In fact, as income fell, purchasing became far more efficient. Thus, Durham families with incomes of around 16s per man value per week purchased 250 calories

Survey data recalculated by the present author



per penny, whereas families with incomes of between 8s-100s per man value purchased 400-500 calories per penny (see Figure 4.2). Similarly, the lower the income, the higher the fraction of income used for food: this is Engels Law, which had been known to economists since the 19th Century and it shows that in the poorest families almost the entire income was spent on food. Again, the relationship is best described as a curve (see Figure 4.3). Moreover, at lower incomes bread and flour came to dominate food expenditure with total income spent on bread and flour rising steeply, indicating once more that there was a critical income threshold of around 10s per man value per week (see Figure 4.4).

From its fallacious analysis of these budgets the Committee believed that the poor could be taught to make better use of their incomes (30). In fact, the data provide evidence that the poor made extremely efficient use of inadequate resources and had a better grasp of economic realities than their academic critics. However, these MRC studies appeared to put on an objective basis the attitudes concerning working class ignorance and inefficiency which had been current in the public health movement since the beginning of the Century.

Survey data recalculated by the present author

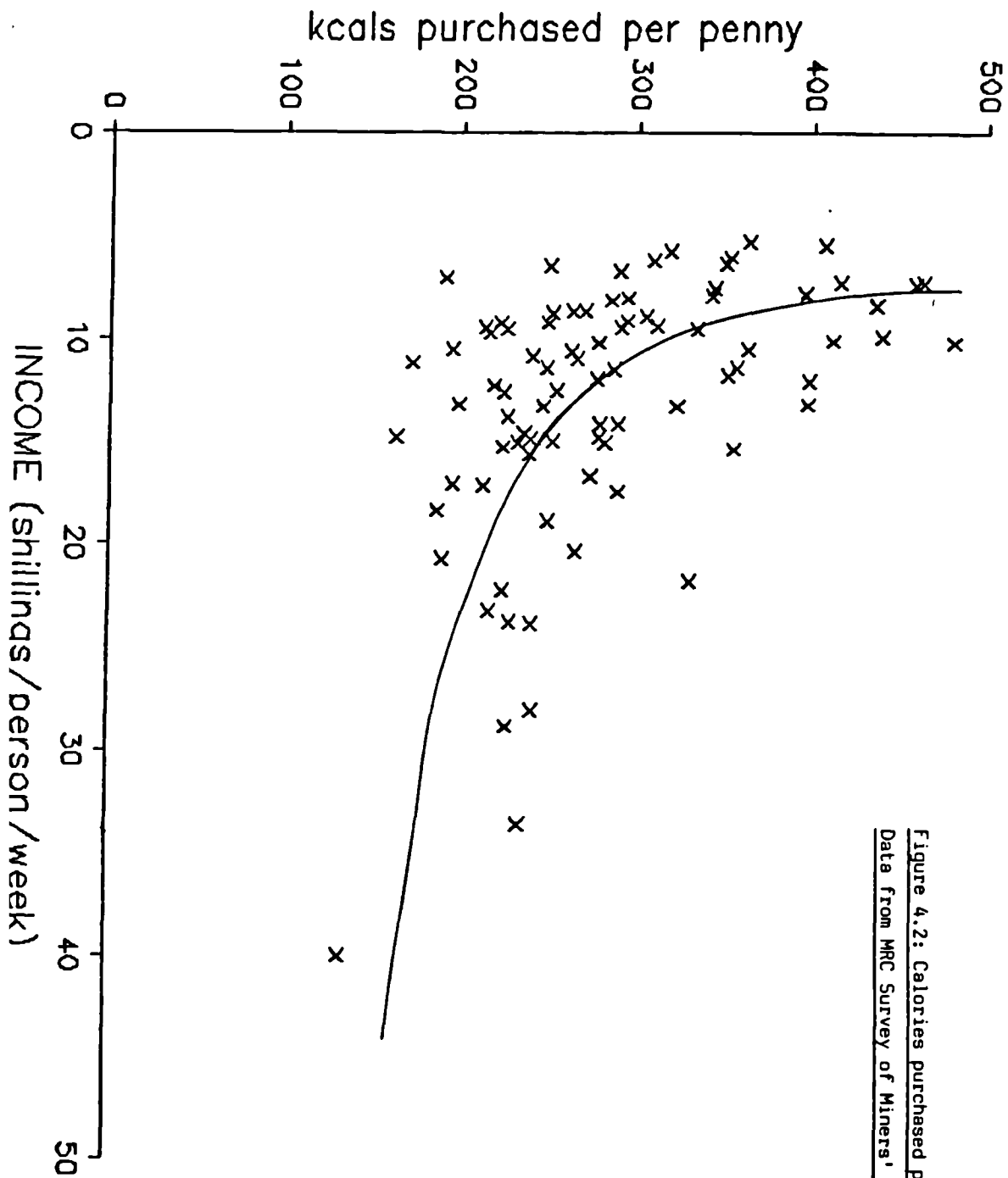
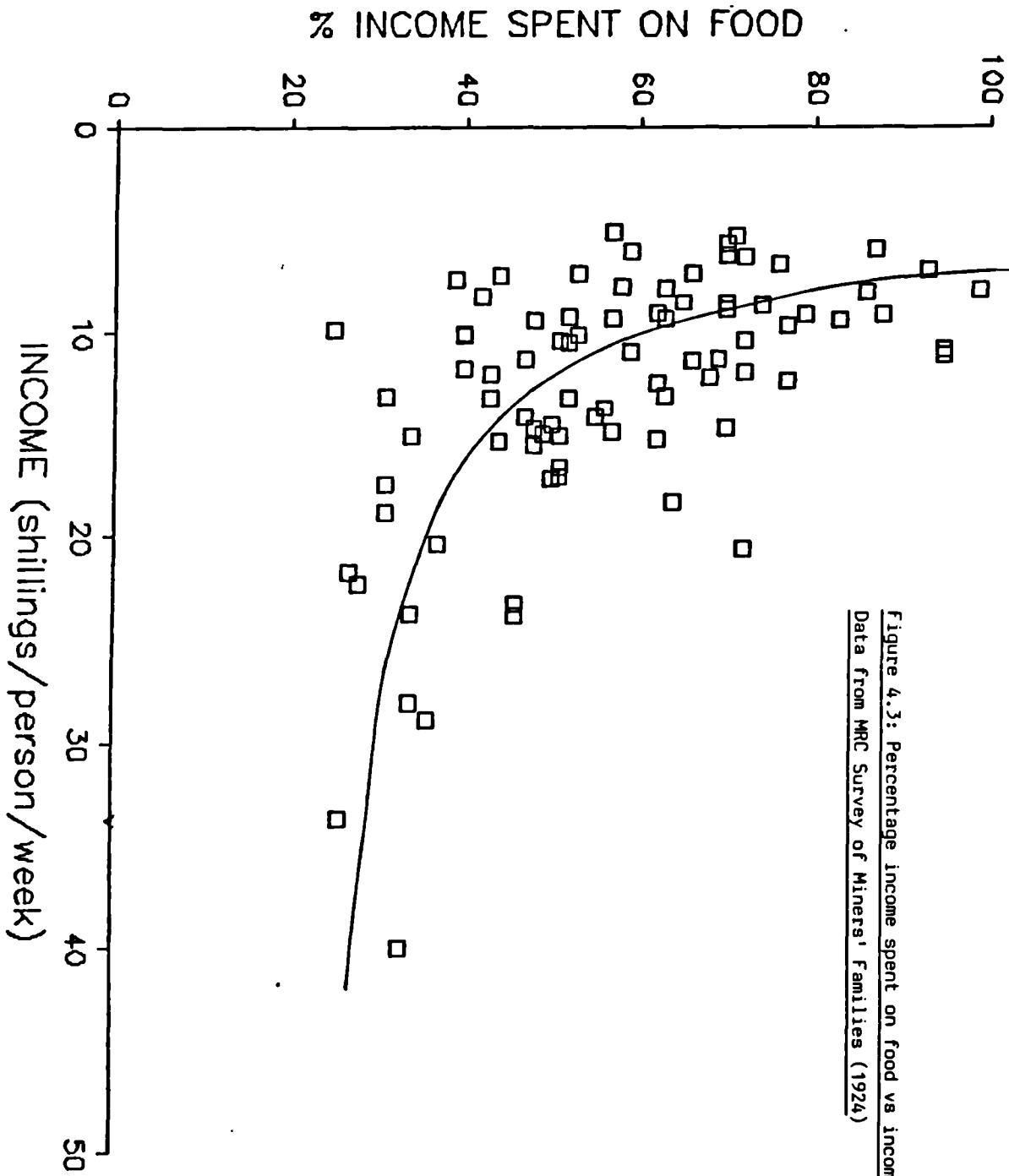


Figure 4.2: Calories purchased per penny vs income
Data from MRC Survey of Miners' Families (1924)

Survey data recalculated by the present author



Survey data recalculated by the present author

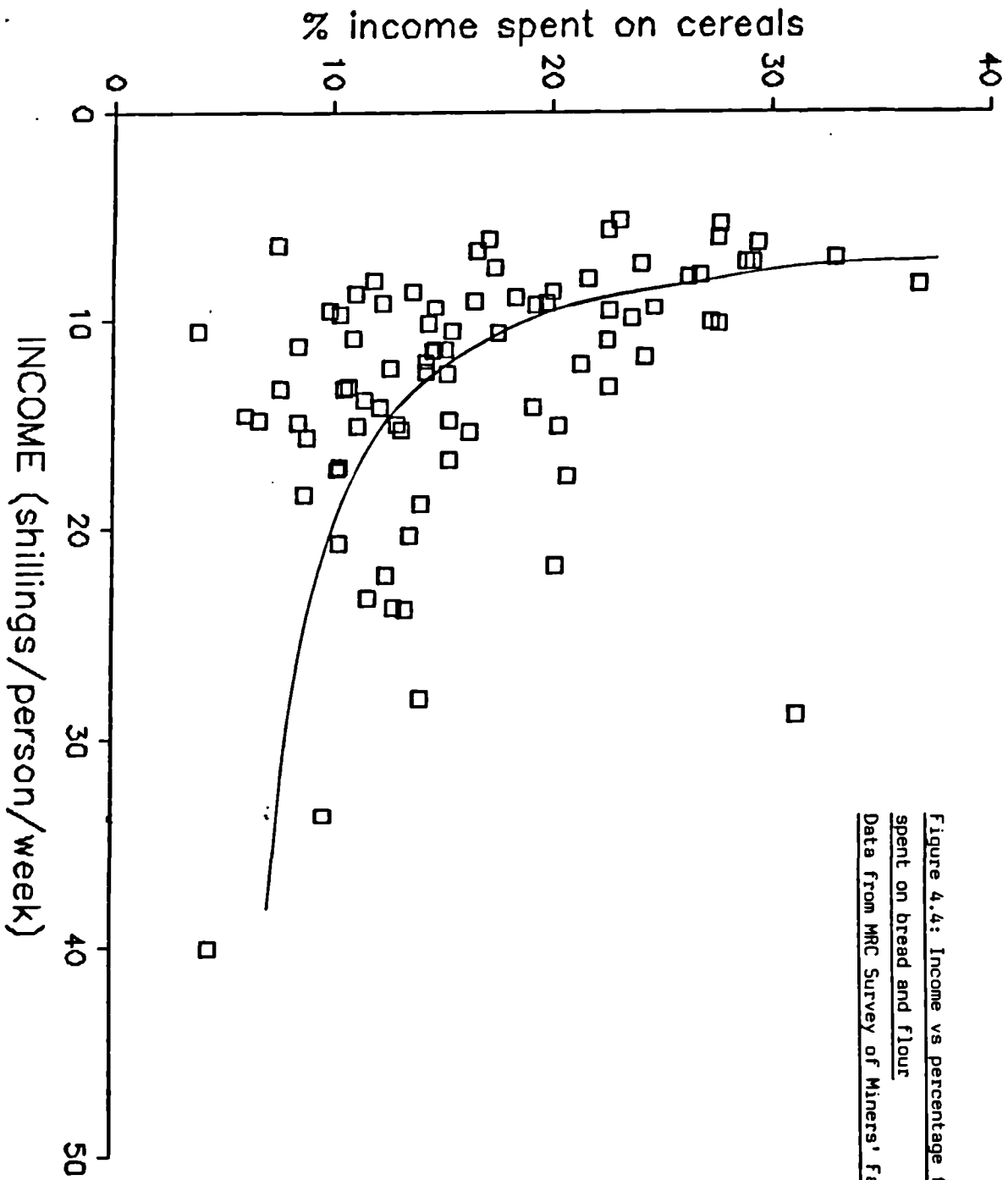


Figure 4.4: Income vs percentage food expenditure spent on bread and flour
Data from MRC Survey of Miners' Families (1924)

4.1.1.3. The Dietary Surveys of E.P. Cathcart

Further evidence which supported this view was gathered by E.P. Cathcart, who in 1922 had been appointed Chairman of the MRC's Committee on the Quantitative Problems of Human Nutrition. This appointment was made on the strength of his calorimetric studies which were undertaken with Benedict in the pre 1914 period (31). Cathcart maintained that there was an urgent need for data on the normal British diet (32) and between 1926 and 1936 he carried out a series of nutritional surveys on behalf of the Council, to determine this (33). However, these were not simply dietary surveys; they also sought to ascertain whether factors other than diet influenced the outcome of the 'nutritional process' (34). This was the same question Paton had asked in POVERTY NUTRITION AND GROWTH, and Cathcart's methodology was in fact modelled on Paton's work. Cathcart's MRC studies therefore included an evaluation of the nutritional status of children in the study families and an assessment of 'parental efficiency' (35), in addition to nutritional analyses of household diets. Maternal efficiency was assessed, as it had been by Paton, on the basis of household cleanliness, the mother's cooking skills and her general capacity as a domestic manager, whilst the criteria for assessing the father's contribution to household efficiency were his steadiness as a worker and his expenditure on alcohol. The nutritional status of children was then correlated with these social, economic and nutritional parameters; the threshold for poor nutrition was defined as it had been in the earlier MRC studies, on the basis of a comparison with the average heights and weights of the local elementary school population at any given age (36). Cathcart's findings corroborated Paton's results; his surveys consistently reported that the chief factor in determining the nutritional status of the poor was neither income nor diet, but 'parental efficiency'. For example, in his study of St Andrews (37),

Cathcart concluded that, since weights did not vary consistently with food expenditure until this reached a level of just under 15s per man value per week (38) - when results became consistently good - 'parental efficiency' was a better predictor of nutritional status than either diet or income (39). At lower levels of income, the children of 'wayward' parents tended to weigh less than their equally poor counterparts from steady homes. Although Cathcart saw this as evidence of 'inefficiency' among sections of the working classes these results could equally be interpreted as evidence that nutritional status was sensitive to poverty since it only ceased to be affected by environmental insult in the higher income groups.

Initially, Cathcart's reports were favourably reviewed by the public health press (40) but by 1937 the final report in his study of the British diet (41), was widely condemned (42). This report included the accusations that the working classes were "hidebound by tradition, full of prejudices and curious false pride", that they spent too much money on gambling and the cinema and that they frequently exhibited "stupid indifference, carelessness and laziness" (43). Cathcart believed that his MRC surveys were proof that:

"when the housewife is skilled in marketing and in cooking an excellent return per penny spent can be obtained, even with abnormally small incomes" (44).

His work is steeped in class prejudice and a nostalgic view of the past. For example, he claimed that many middle class families provided themselves with better diets than working class families with higher incomes, on account of the greater intelligence of the middle classes (45) and he wrote that

"The old race of housewives who were prepared to give the necessary time and take the necessary trouble are dying out" (46).

The idea that the art of 'economic cooking' had been lost is reminiscent of views presented to the Committee on Physical Deterioration, and it

continues to be voiced during periods of economic depression. This loss of domestic skills which the Committee on Physical Deterioration had reported in the towns had now, according to Cathcart, spread to the countryside; the traditional diet was being undermined by "travelling vans supplying tinned articles" (47).

His argument that inadequate feeding could not simply be ascribed to inadequate income, but that

"Bad buying and bad cooking accounted for a great deal" (48)

and that an improvement in nutrition was

"as much a matter of improved education as of increased wages" (49)

was compatible with the Newer Knowledge of Nutrition. However, Cathcart saw in his work not only evidence of the need for domestic education among the poor. He also interpreted it as proof in the "bedrock of experience" that the nutrient requirements put forward by advocates of the 'Newer Knowledge of Nutrition' were excessive. Here, the Glasgow physiologists parted company with the the teachings of the 'newer knowledge' and the more powerful voices of the medical research establishment.

For the public health administration, Cathcart's results were proof that, although adverse environmental circumstances including poverty and poor housing were often associated with malnutrition, the key factor in its aetiology was the ability of the mother to ameliorate their effects.

In Newman's words:

"more often it is careless mothering, ignorance of upbringing and lack of nurture than actual shortage of food which results in a malnourished child. Insufficient sleep, chronic fatigue, absence of fresh air and lack of exercise are exerting a very great influence day by day on the well being of multitudes of children" (50).

Many examples of this belief can also be found in the reports of public health practitioners written during the 1920s. Comments made by the

School Medical Officer for Hornsey in 1924 are typical of the prevailing view

"parental neglect, irregularly and badly cooked meals, late hours and over fatigue are all responsible for the ill nourished child" (51).

Since 'bad parenting' was identified as the source of nutritional problems in the community, preventive public health work sought to change patterns of behaviour in the home. In practice this work was generally confined to the distribution of pamphlets listing the 'protective foods' and the essentials of good child rearing (52) . However, in a number of areas 'nutrition clinics ' were set up during the 1920s and 1930s; the objective of these clinics was to identify in individual children the particular source of their poor physical condition and by regular contact with parents to modify 'causal' factors such as diet and hours of sleep. Children selected for attendance at such clinics were generally those who were described as 'delicate and of tubercular type'. The work of these clinics was reported in the public health press, and was endorsed by Newman in his Annual Reports (53).

4.1.2. The Role of Diet: incorrect and improper eating as the cause of malnutrition

The Committee on Physical Deterioration had identified a poor quality diet, chosen out of ignorance and fecklessness by untutored working class wives, as a major cause of physical degeneracy and throughout the 1920s Medical Officers continued to criticise the poor for their lazy and ignorant dietary habits. The Newer Knowledge of Nutrition did not change the interpretation of the underlying cause of 'bad' dietary practice among the working classes. However it did produce a new definition of a 'correct' diet and the public health movement accepted these changes, which were embodied in the concept of the 'protective

foods'. Using the public health press and the annual reports of local authority Medical Officers it is possible to describe the way in which the Newer Knowledge of Nutrition merged with the prejudices and beliefs which were so vividly expressed in the evidence gathered by the Committee on Physical Deterioration in 1904.

In 1914, Newman had written in his annual report to the President of the Board of Education that unless children were educated in 'correct eating' at school, they would:

"remain content with a sordid and ignorant standard of feeding...Another generation will grow up to eat unwholesome food at irregular hours under unsuitable conditions" (54).

He went on to argue that malnutrition was primarily a problem of bad feeding rather than of insufficient feeding and that the remedy for this problem was to 'teach the children for the benefit of the next generation'. During the 1920s the MEDICAL OFFICER and PUBLIC HEALTH, which were the main journals of the public health profession, continued to preach this doctrine. Both editorial comment and articles submitted by practicing Medical Officers stressed the importance of dietary quality, now defined by specific 'protective' foods and argued that malnutrition was due, not to lack of food but to a combination of ignorance and bad domestic management. According to this view, the role of the public health worker was essentially an educational one. For example, an editorial which appeared in the MEDICAL OFFICER in 1920 asserted that

"It is generally recognised that the cause of malnutrition in young children is more often due to ignorance in the purchase, preparation, preservation and care of food than to an insufficiency of food. Much of the poverty existing is relative in that the public does not know how to spend money to the best advantage from the standpoint of food value, especially as relates to the growth and nutrition of children" (55).

Twenty years after the Committee on Physical Deterioration had published its report, the decline in domestic cooking was still described as an

important cause of poor nutrition. Thus in 1925 the MEDICAL OFFICER quoted the SMO for Norfolk who wrote

"I am strongly of opinion that the chief factor of the well being of a child is not the amount of wage earned by the father but the common sense and industry of the mother. There is far too little cooking done in the majority of our cottages and far too great a preponderance of bread and jam in the dietary of our children" (56).

Similar echoes of the Committee's report appear in comments made by the School Medical Officer for the West Riding who claimed during the Miners Strike of 1926 that

"in those classed as ill nourished, many are not so by reason of poverty of money, only by poverty of resourcefulness of their parents"

and argued that a knowledge of food values would

"quickly lift a good proportion of cases of malnutrition out of this category altogether"

With a remarkable ignorance of the rural economy, he deplored the fact that

"Although eggs, milk, cream, butter and vegetables are produced locally, the children do not seem to get an adequate share of these" (57).

The importance of a knowledge of food values was also stressed by the Chief Medical Officer for Durham during the 1926 Coal Strike. One of his county Medical Officers suggested during this period that Medical Officers in areas affected by the strike should:

"find some means of publishing advice on the subject of food values, so that the unfortunate housewives who must provide meals for hungry families may know how to spend their money to the best advantage" (58).

The MRC's 1924 inquiry into the diets of miner's families, which had reported discrepancies in calories purchased per penny and included a study of Durham miners, argued precisely this point (59).

In 1925 the SMO for Liverpool, Dr Mussen, wrote that of the many causes to which malnutrition could be attributed, 'improper feeding' was the most important, and that

"education in up to date principles of feeding ...[was] urgently required to help the poor spend what money they may have available to the the best advantage" (60).

Investigations into the home diets of Liverpool children classed as malnourished found that they consisted largely of bread, potatoes and vegetable stews containing left overs from a family joint eaten on Sundays. Such diets were said to be marked by a

"deficiency in fats and proteids...and also vitamins
...essential for satisfactory growth and the maintenance of health" (61).

In the absence of dietary intake data it is perhaps dangerous to comment on statements of this nature. Nevertheless, it is difficult to conceive how children could become malnourished on a diet which included meat, vegetable stews, potatoes and bread unless they were also underfed. During the inter war period, however, it remained axiomatic that defective quality rather than insufficient quantity was the chief dietary cause of malnutrition.

This view was summed up in the report of the SMO for Lancashire in 1929 when he asserted that

"the great bulk of malnutrition which is not due to to disease, or bad hygienic conditions and habits is caused not so much by actual lack of food as by the improper selection and preparation of food" (62).

However a re-analysis of working class diets from this period, which appears in Chapter 6 shows that the poor were already spending their very limited resources to excellent advantage and that they demonstrated remarkable skill in meeting the most fundamental physiological need, that is to satisfy hunger.

CHAPTER 4 - PART II

4.2. Nutrition and Public Health Practice, 1929-39

In contrast to the previous decade, the period 1929-1939 was one of controversy rather than consensus. Differences of opinion between Medical Officers and the public health administration were prompted by official claims that the economic depression, which produced unprecedented levels of unemployment in the UK during the early 1930s, was not extracting a price in child health (1). Many public health practitioners disagreed with this assertion. Their criticisms were based both on their own observations (2) and on studies which showed that a 'minimum' diet, as described in both official Ministry of Health guidelines and the BMA nutrition report of 1933, was beyond the means of the unemployed and low paid (See Chapter 3). The public outcry provoked by these revelations was at least as loud as that which had been raised in the first years of the Century over the condition of Boer War recruits, and it led to a change of emphasis in the debate about malnutrition among the ranks of the public health profession. This ceased to be a discussion about maternal ignorance and inefficiency; practising Medical Officers instead addressed themselves to the problem of monitoring and measuring malnutrition. During the 1930s, preventive medicine began to shift from education to politics and two of the major public health journals, the *MEDICAL OFFICER* and *PUBLIC HEALTH*, acted as a forum in this process of professional re-evaluation and re-orientation.

4.2.1. The Campaign for Improved Nutrition

The BMA report on the minimum cost of a physiologically adequate diet which appeared in 1933, heralded a new stage in the malnutrition debate and provided the welfare movement with a formidable weapon. The belief that dietary adequacy could be assessed with scientific precision seemed to put the debate about the impact on health of unemployment and

low pay on a measurable basis. Public health campaigners claimed that it was now possible to quantify the amount of malnutrition present in the community and the specific remedial measures necessary to eradicate this. Their highly plausible welfare targets brought together a number of politically heterogeneous groups in a movement to provide an adequate diet for all. The most notable of these campaigning bodies was the Children's Minimum Committee (CMC) which was founded in 1934. The aim of the CMC was to raise public awareness of the nutrition problem and to lobby for increased supplementary feeding so that no child was, through poverty, deprived of the food it required to achieve its full mental and physical potential (3). Women's organisations, teachers' unions, church and political groups were affiliated to the campaign as well as leading nutrition scientists such as F.G. Hopkins and J.C. Drummond. The Committee Against Malnutrition (CAM) (4) was also founded in 1934, and was actively supported by a number of prominent scientists as well as young researchers and clinicians. The Committee's initial public statement, whose signatories included Hopkins, J.B.S. Haldane and F. Le Gros Clark made the assertion that there was widespread undernourishment among the families of the unemployed and low paid; that this would inevitably lead to a deterioration of the physical standard and health of the population; and that "the last thing upon which a country must economise is the nutrition of the working class" (5). To support this view, the CAM carried out research into various public health applications of the 'Newer Knowledge of Nutrition' and was particularly incisive in its criticisms of the Board of Education's feeding policy and the credibility of the official nutrition returns (6).

4.2.2. New Perceptions of the Extent of Malnutrition: Unemployment, the Slump and the Cost of an Adequate Diet

In 1929 the Wall Street crash inaugurated a period of world economic recession and mass unemployment. Between 1929 and 1932 annual rates of unemployment doubled in the United Kingdom, rising from 9% to 18% of the workforce (7), and totalling 2.7 million men and women at its peak. There were marked variations in the incidence of unemployment, both between industries and between regions (and for some industries, such as coal mining, the entire inter war period was one of high unemployment (8)); however, government and public opinion saw the early 1930s as 'crisis' years, which many commentators believed would result in a general deterioration in child health. This view was documented in a survey of the reports of Medical Officers of Health for 1932 and 1933, carried out by the Children's Minimum Campaign* [See end of Chapter 4 for starred notes].

The survey demonstrates very clearly the way in which many Medical Officers linked increased ill health with a decline in dietary quality due to economic conditions (9). For example, a fall in the consumption of the 'protective foods' was described as the cause of an increased incidence of tuberculosis in Southampton and Sheffield (MOH, Southampton, 1932 and Sheffield Tuberculosis Officer, 1932) and Birmingham's Medical Officer of Health claimed that poor quality diets had resulted in a "general failure to thrive" (MOH, Birmingham, 1932).

Similarly, a rise in maternal mortality in Smethwick from 1.95 per 1000 to 5.43 per 1000, was attributed largely to "poor nutrition among the mothers" (MOH, Smethwick, 1932) and "long term ill effects" resulting from the "inadequacy of national allowances" (set at 2s per child) were predicted by the SMO for Darlington (SMO, Darlington 1932).

In 1933, the BMA published its controversial report on the minimum cost of a physiologically adequate diet (10) which is discussed in Chapter 3. Although this report has been described by commentators such as Church and Walker (11) and Rivers (12) as reactionary and irrelevant, its impact during the 1930s was in fact to focus the debate about poverty and ill health more sharply. After its appearance, local authority Medical Officers began to refer to the specific problem of low income which was believed to prevent many working class families from purchasing an adequate diet. Thus, the 1933 report of the MOH for Sunderland (13) included details of a survey into the weekly food expenditure of applicants for welfare milk in which it was found that the average expenditure per head was 3s2d compared with the BMA minimum of around 5s7d. This sum, the report claimed, was "too little for the maintenance of a healthy life and accounts inter alia for the lack of resistance against illness and the prolongation of the period of convalescence which has been noted in children suffering from infectious diseases" (MOH, Sunderland 1933).

In the same way, the MOH for Bethnal Green referred in 1933 to

"recent enquiries concerning nutrition [which] show that the amount of assistance they receive is inadequate to maintain the families of the unemployed or low paid workers in a proper state of health" (14).

Similar arguments appeared in a major report on the effects of unemployment on Children and Young People, published by the Save the Children Fund in 1933 (15). Commenting on the nutrition problem in the context of this report, the MEDICAL OFFICER noted that the weight of scientific evidence now provided almost certain proof that children being raised on diets dictated by current levels of unemployment relief

"though it does not cause what we mis-call ill nutrition, does cause inhibition of function so that children so fed cannot be educated to make useful citizens" (16)

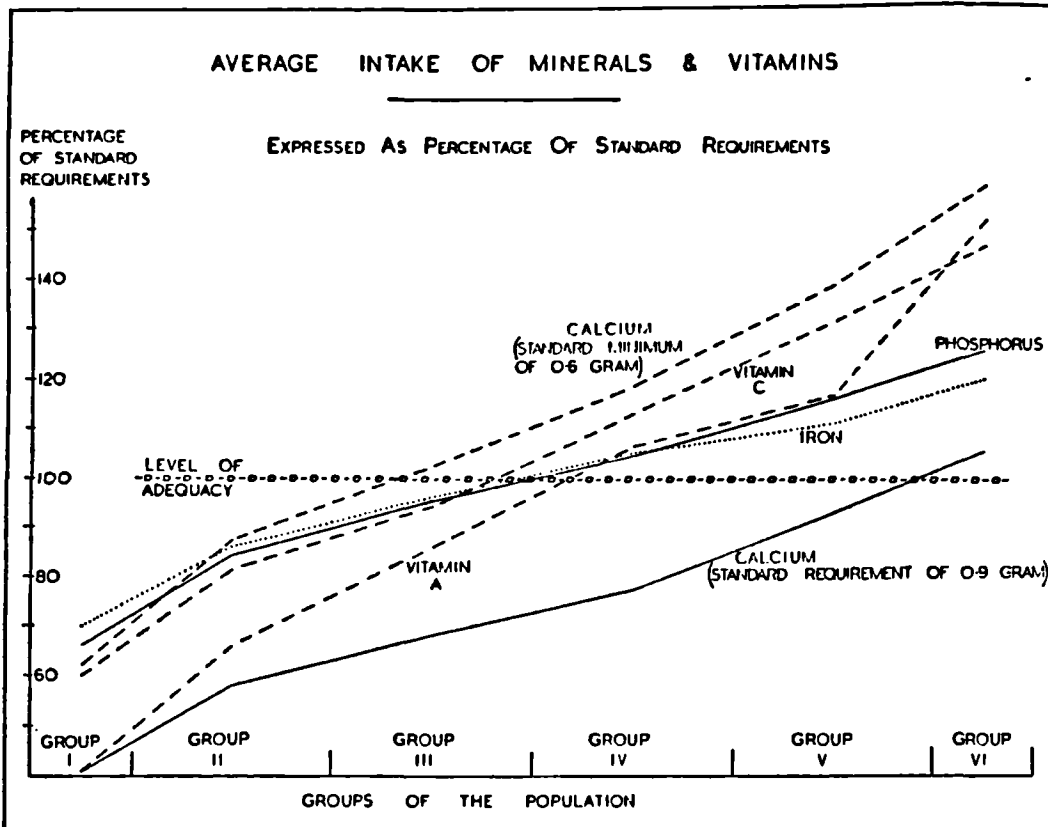
4.2.3. Measuring Malnutrition: The Economic Standard

Following the publication of the BMA study, it was widely argued that economic criteria should be used to assess the prevalence of malnutrition in the community. Various surveys carried out during the period 1933-1939 adopted this approach; of these, Boyd Orr's study, FOOD HEALTH AND INCOME was the most comprehensive and had the greatest political impact (17). FOOD HEALTH AND INCOME looked at the food consumption of families at various levels of income, and assessed the adequacy of these diets in relation to contemporary nutritional standards (see Figure 4.5). It then extrapolated for the country as a whole the amount of dietary deficiency (or 'malnutrition') which, according to these standards, might be assumed to exist.**

The study received a sensational press coverage, and produced headlines which proclaimed that half the population of the UK was malnourished; its political impact was equally dramatic and the vehemence with which the Minister of Health denied Boyd Orr's claims (19) is a measure of its success as a piece of public health propaganda.

A number of small scale investigations were also carried out after 1933 which used the BMA criteria to assess the local prevalence of malnutrition; studies undertaken by the Ipswich Committee Against Malnutrition, by the Sheffield Social Survey Committee, and by the Birmingham Social Survey Committee are typical of these inquiries which confirmed the view that low income families could not afford to buy the minimum diet which according to the BMA standards, was required for health (20).

Fig 4.5



Population is grouped according to family income available per head for food.

Group I is the poorest group

Source: J. Boyd Orr, 'Food Health and Income' (17).

4.2.3.1. Nutritional Guidelines and the Case for Higher Benefits

The economic standard was adopted by welfare campaigners to press the case for increased benefits, as Cathcart had predicted it would be (21). For example, Eleanor Rathbone used the BMA standard to calculate the shortfall of state benefits in relation to the physiological minimum required by families of various sizes and her report on this subject, which appeared in 1934 (22), was used by pressure groups including the Children's Minimum Committee (CMC) and the Family Endowment Society (FES). The CMC used it in its efforts to secure higher national benefit rates from the Unemployment Assistance Board (23) and the FES cited it in its submissions to the Unemployment Insurance Statutory Commission (24). Table 4.1 shows family income shortfalls for families of various sizes according to various standard of living criteria as calculated by Rathbone***. [See Note *** for references 25-29]

4.2.3.2. Measuring Malnutrition: The Official View in Doubt

The high prevalence of malnutrition indicated by the use of economic criteria cast doubt on traditional methods of nutritional assessment and the need to find an acceptable 'objective' test was recognised by all sections of the public health movement.

According to official statistics, the economic crisis had produced no perceptible increase in malnutrition (30) yet, according to the 'objective' BMA standard, large sections of the population were consuming a diet which was inadequate for health; they were malnourished. The inference, therefore, was that existing methods for measuring malnutrition were inadequate. Proof of the existence of 'hidden hunger' or 'marginal malnutrition' consequently became a priority for nutrition research since it was recognised that without it politicians would continue to argue that existing levels of welfare provision were adequate. This new challenge was summarised in an

Table 4.1

FAMILY INCOME SHORTFALL ACCORDING TO VARIOUS STANDARD OF LIVING CRITERIA
CALCULATED BY RATHBONE, 1934 (22)

Family size	New London 1928	Merseyside 1928-9	W'End Review 1933	BMA 1933
Man, Woman, one child	-1s8d	+3s2d	+2s1d	+3d
Man, Woman, 3 children	-6s4d	-9d	-2s7d	-5s3d
Man, Woman, 5 children	-15s2d	-7s10d	-10s5d	-14s3d

Table shows amount of money below various estimates of the poverty line families of 1, 3, or 5 children would be, if family income was equal to the 1934 Unemployment Benefit level.

editorial which appeared in the MEDICAL OFFICER shortly after the BMA report was published

"We fail to find evidence of malnutrition in the population even though we know that it must be present...We must show that families below a certain income level are suffering from an ill nutrition which those above that level are not" (31).

In the same issue, W.R. Dunstan, who was School Medical Officer for Lewes, and an authority on the use of anthropometric indices in the assessment of nutritional status, wrote:

"We must find out the clinical signs of malnutrition, for these we do not know" (32).

Previously Medical Officers and the public health administration had expressed a generalised concern about the low consumption of the 'protective foods' in the depressed areas and the importance of these foods had been stressed in the annual reports of the Chief Medical Officer since the early 1920s (33). Now, however, many public health workers believed that:

"if food [intake] falls below the standard defined as adequate..then from the point of view of the biochemist that individual is malnourished" (34).

Using this criterion, it was argued that

"We have districts where the amount spent on food is utterly inadequate..and we report the observed nutrition of the children to be 90 to 95 per cent sound. We know this is false. We must find out the clinical signs of malnutrition for these we do not know" (35).

The urgency of this problem was expressed at the Committee Against Malnutrition's first public meeting, where leading research workers such as V.H. Mottram, spoke of the need to establish deficiency syndromes for "salts, vitamins, protein with fat and total calories" and as a first step, Dunstan suggested that malnutrition should be classified according to:

"(a) those cases in which some food deficit is the primary factor and (b) all others" (36).

In the light of the Newer Knowledge of Nutrition, the only methods by which nutritional status could be reliably assessed were, according to

the CAM, to

"(a) assess exactly how much food of the necessary quality the children are consuming week by week (b) to subject each child to a series of tests as precisely as they can at present be made - blood tests, urine tests, tests for muscle reaction and so forth" (37).

Until funds were made available for the mass biochemical and clinical studies of the Second World War, the first method was adopted as the only practical 'objective' method of quantifying the extent of malnutrition in the community. Thus Harris and many other nutritionists, including Boyd Orr, argued during the 1930s that

"When the amount of money available is less than that which expert opinion considers less than the minimum necessary for the maintenance of health, then a presumption must be raised that some degree of undernutrition is present" (38).

Harris favoured an economic rather than a physiological diagnosis on the grounds that there were no agreed reference standards; such standards as existed might be debased; and that the worst and more obvious effects of undernutrition might be delayed (39).

E. Wilkins, another Medical Officer who contributed regularly to the nutrition debate in the professional journals, presented similar arguments in an article published by the MEDICAL OFFICER in 1937. Having listed the by now familiar shortcomings of the traditional clinical assessment, he went on to stress the importance of an economic enquiry in order to determine:

"not..the diet itself, but..the possibility of an adequate diet being purchasable" (40).

4.2.4. Unemployment and Health: the attack on 'official complacency'

A number of specific criticisms were levelled against the Board of Education's optimistic account of the nutritional status of the child population and these criticisms appeared to be confirmed by a series of reports produced by the Committee Against Malnutrition (CAM). For example, in 1935 it published an analysis of the malnutrition returns

submitted by 40 School Medical Officers, grouped according to social and economic conditions. When these reports were compared with each other and with more prosperous or more depressed areas, striking discrepancies were found both between towns in the same group and between groups. Thus Newport, with an unemployment rate of 22%-41%, reported 24.3% malnutrition and Radnorshire, with 39%-43% unemployment, reported 0.02% malnutrition. Discrepancies of this magnitude led the CAM to conclude that

"the conception of malnutrition cannot mean the same thing in these two areas" (41).

Many similar discrepancies were found: towns with low unemployment rates such as Canterbury produced far higher malnutrition returns than depressed areas such as Merioneth and South Shields; these differences could not be accounted for by differences in school feeding policy. A subsequent CAM report published in 1936, showed that changes in the classification of nutrition introduced in 1934 had not led to a diminution in obvious irregularities (42). The single category 'malnourished' was replaced by four categories which ranged from "A", which denoted excellent nutrition to "D" which denoted 'bad' nutrition but the inconsistencies remained. During the mid 1930s, papers were published by Betenson (43) and Hws Jones (44), which attempted to explain why the official returns were so unreliable. These concluded that Medical Officers based their assessment of nutrition on personal concepts of normal nutrition, which in turn were based on the 'average' standard of the children they examined. These studies showed that, under experimental conditions, the same children were often placed in different categories and that their assessment was strongly influenced by the average standard of the group in which they were examined. It was argued that these inconsistencies were the unavoidable result of the clinical examination itself and that an alternative objective method

for assessing nutritional status was therefore urgently required. The following reforms were widely canvassed:

a) The use of clinical tests to distinguish cases of 'primary' malnutrition (resulting from 'lack of proper or sufficient food'), from 'secondary' malnutrition (caused by other factors such as recent illness).

The importance which was attached to distinguishing between these two classes of malnutrition was mentioned in the previous section. It was symptomatic of the view which defined malnutrition according to a set of dietary standards and it produced the prolonged search for the clinical signs of marginal malnutrition, which remained a major preoccupation of nutrition science for the next two decades. This view can be traced directly to ideas concerning micronutrient deficiency and sub optimal health described in Chapter 2.

Although under MacNalty's tenure of the post of Chief Medical Officer, the public health administration supported research into biochemical tests for malnutrition (45) and by the end of the decade, in addition to haemoglobin levels Vitamin A and Vitamin C levels could be monitored using relatively simple techniques, the Board did not have the resources to undertake clinical tests on a mass scale. Moreover, it was already evident that the 'normal' covered a wide range that these tests were of little practical value for the diagnosis of malnutrition in individual children (46).

b) The use of anthropometric indices.

Anthropometric indices appeared to offer a more promising objective standard for nutritional assessment, and their use was seriously considered by the Board. However, a study carried out by R.H. Simpson in 1934 (47) concluded that the problem of defining an acceptable cut-off

for the diagnosis of malnutrition was insurmountable; an unacceptable number of children who would be classified as malnourished according to traditional assessment criteria were not so classified by low weight for height criteria. The Advisory Committee on Nutrition concurred with this view (48).

Nevertheless, the CAM published surveys which showed a relationship between poverty and low weight for height (49) and these surveys were cited as evidence that anthropometric indices should be used in the classification of nutritional status. For example, one of the surveys, carried out by the SMO for Breconshire, Dr Betenson, compared children from South Wales with children from Sussex using Dunstan's standard reference population of Sussex school children. Betenson found that among children from the depressed area 11.4% showed a deficiency in weight for height of more than 7%, whilst the figure for Lewes was 9%. In its report of the study, the MEDICAL OFFICER commented

"Here, at least, is evidence that the economic depression has exerted 'a measurable physical ill-effect on the child population'" (50).

Subsequent studies also found that low weight for height was more prevalent in depressed than in non depressed areas and indeed by 1937 the Board of Education was using aggregate heights and weights to evaluate the credibility of LEA clinical returns (51). However, the Board held fast to the view that anthropometric indices should only be used for the purpose of mass monitoring and not for assessing the nutritional status of individual children.

4.2.5 Diet and Health: the Public Health Paradox

Although high levels of unemployment and reduced family income were increasingly accepted as a priori evidence for the existence of widespread malnutrition, it proved impractical to adopt economic criteria for the assessment of nutritional status in individuals; some

poor children were clearly healthier than others. Similarly, it was found that neither anthropometric indices nor laboratory tests could produce 'objective' evidence that all poor children were to some degree malnourished. So, although Boyd Orr had adopted as his standard for perfect nutrition

"a state of well being such that no improvement can be effected by a change in the diet" (52)

and had argued that families below a given level of income were by definition malnourished, this view did not help Medical Officers in compiling their annual nutrition returns. The paradox which confronted every Medical Officer during this period had been described in 1924 by an SMO in Kent, who observed that

"it is true that where there is poverty, malnutrition is likely to be greater in degree, it is also true that one finds well-nourished and fit children, living in the most unpromising environment, in families which are below the poverty line" (53).

More than a decade later, Stott (54) found in a study of the family food expenditure of children classified in nutrition groups "A" to "D" that only 60% of the children from families who were spending 2s to 2s11d per head per week on food were identified by an 'independent observer' as malnourished, yet this food expenditure was less than half the sum defined by the BMA as the minimum requirement (55).

Although some investigators continued to argue that bad household management was the cause of the problem of poor nutrition (56) far more radical inferences were drawn from studies such as those carried out by Spence in Newcastle (57) and by Medical Officers in Cardiff (58) and Bristol (59), which emphasised the role of both poverty and slum housing as causes of malnutrition. This broader understanding of the aetiology of malnutrition was described in F. Le Gros Clerk's collection of essays NATIONAL FITNESS (60) and its appearance in 1938 bears witness to a revival of the radical analysis of the nutrition problem set out in

Arthur Greenwood's pre First World War study, THE HEALTH AND PHYSIQUE OF SCHOOL CHILDREN (61).

Greenwood, a future Labour Minister of Health, had argued in 1913 that ill health and poor physique were consistently associated with low income and environmental deprivation and that these factors, rather than maternal inefficiency or a badly chosen diet, were responsible for social class inequalities in health. In this study, Greenwood had observed that

"It seems clear...that malnutrition is in a large degree the result of poverty and its concomitants - heavier incidence of disease, unhealthy conditions, overcrowding, ignorance etc."(62)

His conclusions were based on an analysis of the causes to which malnutrition was attributed by School Medical Officers, in the investigations which Newman had requested in his annual report of 1910 . Greenwood argued that the causes listed in these enquiries were "the accompaniment of poverty" and so he concluded that malnutrition could only be eradicated by social and economic reforms which brought an end to poverty (63).

During the 1930s, the nutrition question became less partisan and the most influential voice in the campaign against poverty and inequalities in health was Boyd Orr, who chose to stand outside party political affiliations. Orr's study, FOOD, HEALTH AND INCOME was welcomed by the left, and his findings were cited as proof that existing levels of unemployment assistance were unacceptably low (64); however, it was also welcomed by non political groups such as the Federation of Women's Institutes and the People's League of Health (65) who saw it as evidence of the need for for cheap milk for the poor and more welfare feeding. This blurring of political divisions was caused by the adoption of an objective 'scientific' standard for the definition of dietary adequacy. But the scientific standard proved to be a double edged weapon, for in

the absence of clinical signs of deficiency disease, it was increasingly argued by government and the public health administration that existing levels of welfare benefits must be adequate (66).

4.2.6. The Diagnosis of Malnutrition: the development of a multifactoral approach

This official view was to some extent counterbalanced by a series of investigations which attempted to grapple with the complex problems of nutritional assessment and the aetiology of malnutrition; they were more relevant to public health practice than the increasingly sophisticated but inconclusive clinical tests for Vitamin deficiency developed during this period. The first and most influential of these studies was that carried out by Spence in Newcastle upon Tyne in 1933 (67). Spence took samples of working class and professional class children under five years of age, and included in his assessment of their nutritional status heights and weights, general physical condition, evidence of rickets and anaemia, the incidence of 'other nutritional diseases' (including night blindness, beri beri and scurvy) and dietary adequacy, which was estimated on the basis of family income and parental consultations. Housing conditions were also considered in this attempt to put the study of malnutrition on a more rigorous basis. Spence's results suggested that the main factors contributing to malnutrition were (a) overcrowding and poor housing conditions which led to mass infection among young children at susceptible ages and (b) improper and inadequate diet which prevented satisfactory recovery from illness.

This study was followed by an investigation by Newcastle's Medical Officer of Health, J.A. Charles (68), which included a study of the prevalence of mortality and morbidity due to infectious disease according to municipal wards, and a dietary survey of 15 households where income was derived from Unemployment Benefit or Public Assistance. The study compared food expenditure per head with sums recommended in

the BMA nutrition report and found that this was deficient by around 20%. It also found that mortality due to infectious disease was closely associated with poor housing, and that housing was more closely related with nutritional status than food expenditure. The report therefore concluded that

"it is probable that the range of permissible diets is much wider than is usually held to be the case".

Subsequent studies carried out by public health officers in Cardiff and in Bristol illustrate the same awareness of the need for a new approach to the investigation of malnutrition. For example, the Cardiff study of 1936 was based on a detailed comparison of the

"economic position and dietary of the families of children who are to be found at the extremes of the nutrition scale" (69).

Children selected for the study had previously been placed in nutritional category A (Excellent) and D (Bad) during routine medical examinations. Those in nutritional group A in general came from homes with higher incomes than those in group D; where there was a higher expenditure per head on food; and where food intake was 'superior in both quality and quantity'. Other factors including housing, maternal health and family size were also investigated and again it was found that children in category "A" were generally better housed and from smaller families with better maternal health; but since these factors were also associated with higher income the study concluded that low income was the most important factor in the causation of malnutrition. It did not argue that poverty, bad housing and a poor diet invariably produced malnutrition, but rather that malnutrition was produced by poverty.

The Bristol study of 1938 (70) sought to analyse the composite picture of the 'well nourished child'. Extensive clinical and home surveys were carried out on children who had been classified in nutrition group "A"

and nutrition groups "C" and "D". It was found that the well nourished children were

"taller, heavier, look more healthy, stand better, have more healthy colour, better chest expansion, more regular teeth,... are less prone to bronchitis, have less rickets..are more intelligent, stronger"

Among groups "C" and "D" there was lower income, worse housing and higher unemployment. The report therefore concluded that

"We feel we can unhesitatingly say that it is the economic state of the family [which is responsible for poor nutritional status]. Where the income is low it is difficult to provide enough food, or obtain satisfactory houses. The mother is engaged in an unequal struggle and her health often fails under the strain or she ceases to care."

Even here the notion of maternal efficiency was identified, within the broader context of poverty, as the key variable in determining whether the family was able to resist the rigours of an impoverished environment. However, her struggle was now acknowledged to be 'unequal'.

Not all public health officers were converted to the view that blamed both poverty and slum housing for ill health. For example, in 1933 Critchley published a study of factors associated with malnutrition in which he argued that it was

"not the province of a medical officer to dabble in politics over questions of wages etc"

and that one of the main causes of malnutrition was "mismanagement", which included

"unsuitable or badly prepared foods, lack of sleep, fussing etc" (71).

Similarly, an enquiry carried out by the MOH for Worcestershire which examined the home circumstances of 40 children in nutrition class "D" reported that 29 came from 'comfortable homes', only 11 were cases of 'unquestioned poverty' and in the remaining 12 there was allegedly 'definite evidence of inefficient "mothercraft"'. It therefore concluded that "poverty is a subsidiary factor in causing [malnutrition]" (72). However, opinions such as these were not on the ascendant, at least

according to the views public health practitioners expressed in their journals.

During the 1930s Medical Officers were forced by concern over the nutrition question to reconsider the range of factors associated with malnutrition. The dietary definition of malnutrition which had alerted them to the possibility that all families below a given level of income were 'malnourished' led to a far more complex understanding of the problem. At the close of the decade it was widely acknowledged that, although poverty did not always result in malnutrition, malnutrition was invariably associated with poverty. This was precisely the finding of the pre 1914 studies. However, many public health workers now recognised that the solution lay in better housing and higher wages for the poor, rather than in the reform of 'incompetent' mothers.

NOTE* The survey is particularly important since a complete set of the reports of local authority Medical Officers is not in existence - a combination of war time bombing and archival weeding has resulted in the destruction of many items in this unique source of historical and epidemiological data.

NOTE** The Ministry's major objection to Boyd Orr's inquiry was that his reference diets were unrepresentative, and contained a disproportionate number of unemployed families. However, the study can also be criticised on the grounds that these diets were based on surveys which in general included only one week's intake. These surveys did not take into account the possibility that, since dietary intake varies, a single demonstration of inadequate intake (typically lasting a week) was not necessarily evidence of general inadequacy.

NOTE*** The Hungry England and BMA publications were not the first to attempt to state the cost of a 'physiologically adequate diet'. In 1927, Mottram had carried out a similar exercise (25). However, Mottram's calculations did not receive notice outside a small circle of workers interested in domestic economy. Follow up studies included papers by M.L. Clark (26) and G.P. Crowden (27). Mottram's 1927 standard of between 7s and 7s7d per man value per week was not widely adopted. A study carried out in Wakefield (28) looked at unemployed families and found that the 7s specified by Mottram as the cost of an adequate diet, represented 60%-70% of total income before rent. Large numbers of the town's population were therefore, according to this standard, living on inadequate diets. However, the report observed that "insufficient feeding in community (by scientific standards) was not matched by the number of malnourished children in the schools". The report therefore concluded that there was no necessary link between insufficient feeding and malnutrition. An investigation carried out by the MOH for Hammersmith in 1931 (29) adopted Crowden's standard and found the incomes of unemployed and low paid families to fall short of this by over 50%.

CHAPTER 5

NUTRITION INTERVENTION AND THE NEWER KNOWLEDGE OF NUTRITION:

The Use of Milk in School Feeding, 1919-1939

5.1. Milk and Welfare Politics

Free milk for more - if not all - school children, for pregnant and lactating mothers and for infants, were objectives which united diverse organisations and pressure groups which were stirred by reports of widespread malnutrition. Between 1936 and 1938 deputations received by the Ministry of Health on this matter included the Federation of Womens Institutes; the Labour Women's Group; the Children's Minimum Campaign (CMC) and the Peoples' League of Health (1). It was widely believed that large numbers of children, including many who were apparently well nourished and from 'comfortable' backgrounds, failed to reach their full developmental potential because they did not consume enough of a growth factor present in milk. In this sense, the perceived deficiency in milk consumption was a great leveller and milk feeding provided an ideal basis for an egalitarian welfare policy.

Proposals for extending the provision of welfare milk were blocked in the 1930s by a lack of Treasury funds, and by the refusal of the milk distributors to accept the reduced profit margins which were demanded under various cheap milk proposals (2). The cost of extending existing milk feeding was analysed by an independent interdepartmental Committee in 1936 the findings of which are summarised in Table 5.1 (3). Finance for even the most moderate of these schemes was unavailable and by the late 1930s the Ministry increasingly took the view that the malnutrition question had become a political rather than a public health issue (4). However, a month after Winston Churchill assumed office in 1940 welfare feeding was radically extended. At a stroke, cheap milk was made available through the maternal and child welfare network and a cornerstone of the welfare state was laid. Free milk for all children,

Table 5.1

ESTIMATED COST OF INCREASED PROVISION OF FREE AND SUBSIDISED MILK
(according to Independent Interdepartmental Committee, 1936)

COST OF MILK FOR SCHOOL CHILDREN: 1936 £270,000

Free milk for all school children £6,000,000

Free milk for 600,000
poor children £ 660,000

2/3 pint for others
at cost of 1/2d * £1,930,000

Free milk for 1,400,000
poor children £1,320,000

2/3 pint for others at
cost of 1/2d * £1,690,000

* These estimates were based on the assumption that 50% of the children not eligible for free milk would take advantage of the scheme

which was introduced under the 1944 Education Act was symbolic of the new egalitarian post war society. Its withdrawal in 1975 by Margaret Thatcher, then Secretary of State for Education, was equally symbolic of its demise. But although milk feeding has important associations with the social welfare movement its appropriateness as a supplementary food during the 1920s and 1930s can be questioned.

5.2. Milk and the Newer Knowledge of Nutrition

Nutrition scientists and the medical research establishment argued throughout the inter-war period that the population could be raised to new levels of health and vitality by dietary reform. This was translated into a scheme of popular dietetics based on the notion of the 'protective foods'. Milk was described as 'foremost' of the protective foods since it was believed to contain more of the 'high quality' nutrients which were thought to be lacking in the diets of large sections of the population. However, evidence also accumulated during the 1920s which seemed to suggest that milk had the added benefit of stimulating growth in children (5). The dairy industry had been in a state of near bankruptcy during the 1920s, and although it is beyond the scope of this thesis to discuss agricultural and economic aspects of the subject, government had good reason for welcoming the findings of nutrition science in sharp contrast to the present situation. Thus, throughout the 1920s and 1930s, both the Ministry of Health and the dairy industry drew heavily on studies which appeared to show that milk was an especially valuable food. The most important of these studies, Corry Mann's MRC study, "Diets for Boys During the School Years" is re-examined in Section 5.5.

During the inter-war years, the Ministry of Health and Board of Education, together with the milk industry, transformed the public image of milk. This was achieved, not by improving the quality of the product

which in the absence of compulsory pasteurisation remained a considerable public health hazard, but by harnessing the results of various scientific experiments to mass advertising campaigns (6). The Ministry of Health recognised that public perceptions of the health risks associated with drinking unclean milk contributed to the relatively low consumption of milk in the UK - price was the other limiting factor (7). However, it did little to impose higher standards upon the dairy industry (8); thus, in correspondence with Lord Astor who was a leading campaigner for improvements in the milk industry, Newman argued that

"on the whole children suffer less if they drink more milk of the second class than if they drink no milk at all or if only a limited number drink the top class" (9).

A reminder from Lord Moynihan, President of the Royal College of Surgeons, that every year more than 2,000 children died of Bovine tuberculosis (10) did not change Newman's view and his public support for the 'milk and health' campaigns continued (11).

This chapter considers the impact of new ideas concerning dietary quality on local authority feeding practice, the official campaign to promote milk feeding in schools, and the experimental evidence on which the policy of milk feeding was based.

5.3. Nutritional Intervention: The School Meals Service

A state subsidised school meals service was inaugurated in 1906, partly in response to the findings of the Committee on Physical Deterioration (12) and the subsequent Committee on the Medical Inspection and Feeding of School Children (13) which were described in Chapter I. The aim of this service according to both the initial enabling legislation and later Education (Provision of Meals) Acts (14) was to supply meals to needy children who

"through lack of nourishment were unable to take advantage of the education provided".

In 1902, the London County Council had already set out guidelines for school feeding which were subsequently endorsed by the CMO; substantial meals should be provided and they should be planned to remedy both 'qualitative' and 'quantitative' defects in children's home diets. Thus, the School Medical Officer of the London County Council asserted in 1902

"To set out to relieve underfeeding by a single meal a day, it is necessary to concentrate on the proteids and fats" (15)

and in 1907, school meals in Bradford were planned

"so as to contain a good proportion of the ingredients which are lacking at home..in the case of children the more expensive forms [of proteins] are necessary because the growth of the body depends entirely on the proteids" (16).

In the pre 1914 period these nutritional objectives were adopted by the the School Medical Service, whose officers were responsible for supervising the standard of meals provided by Local Education Authorities (17). However, during the inter war years, the Newer Knowledge of Nutrition resulted in a redefinition of dietary quality and Medical Officers were directed to focus on deficiencies in the micronutrient content of the diet rather than on the macronutrients. This produced a shift in emphasis in recommended school feeding practice. Whereas school meals were initially intended as a means of providing 'wholesome' and 'well balanced' meals to an impoverished target population, during the inter war years milk feeding was promoted as a more satisfactory alternative. The appropriateness of this policy can be queried: as the dietary survey data which appears in Chapter VI indicate, the diet of this target population remained essentially unchanged. In the very poorest income groups, from which children eligible for feeding were drawn, there was still a shortfall of energy. It will be shown that the switch to milk exacerbated rather than reduced that deficit.

5.4. The Official Endorsement of the Newer Knowledge of Nutrition

The annual report of the Chief Medical Officer to the President of the Board of Education was the main line of communication between the Board and School Medical Officers, whose statutory duties included the approval of dietaries provided by Local Education Authority (LEA) school meals services. Throughout the 1920s Newman, the CMO, stressed the applied significance of discoveries in the field of vitamin biochemistry. Thus, in his report for 1920, he informed Medical Officers that, in addition to carbohydrate, protein and fat,

"the dietary must also contain a sufficiency of VITAMINS if its full value is to be gained" (18).

In 1923, Newman cited McCollum's "experiments with rat colonies" and wrote that

"An improperly constituted diet leads not only to physical inefficiency but to symptoms of nervous instability, lack of powers of endurance and of resistance to the onset of disease" (19).

In his memoranda, he appeared merely to re-affirm the view which was already well established in the pre 1914 years, that school feeding should supplement inadequacies in children's home diets. However, this inadequacy was now defined as a shortage of foods with a high vitamin content. Thus, the London practice of supplying milk or cod liver oil was praised on the grounds that these foods contained

"to a special degree the vitamins which are so often deficient in the diet of poor town children" (20).

During the period 1920-1925, Newman's Board of Education reports emphasised the dietary significance of the 'protective' foods in general. A diet which included milk, green leafy vegetables and fresh fruit was described as essential for healthy development (21). However, after 1925 official advice concentrated almost exclusively on the importance of milk. This coincided with the publication of Corry Mann's feeding experiment, "Diets for Boys during the School Years" (22) which

appeared in 1925. Corry Mann's experiment suggested that milk feeding produced better growth than other foods; it was hypothesised that a growth factor present in milk was responsible for this phenomenon. Newman described Corry Mann's results in great detail in his report for 1925, and maintained that they supported

"the claims made as to the special and supplementary value of milk in the dietary of the growing child."

He therefore welcomed as a progressive development the

"wider use of milk as a supplementary article of diet" (23)

and stressed that the findings of Corry Mann's investigations were

"of the first importance to every local authority or voluntary society engaged in the feeding of school children"(24).

Newman's 1925 report to the Board of Education also endorsed the National Milk Publicity Council's (NMPC) Milk in Schools Scheme (25) which involved the setting up of school milk clubs and provision of milk in 1/3 pint bottles at the cost of 1d.

The encouragement of milk feeding is an outstanding feature of the CMO's reports during the period 1925-1934 and the results of Corry Mann's and later Scottish feeding experiments were the main plank in this campaign. The identification of milk feeding as the answer to the national's nutritional ills reached its zenith in the late 1920s. In his report to the Minister of Health in 1927, Newman wrote

"Because of [its] protective qualities we might with confidence look to an increased consumption of milk to bring about a reduction in the prevalence of rickets, of defective teeth, of underdeveloped bodies so easily the victims of disease ... and add proportionately to the health and vigour of the nation" (26).

Official propaganda in favour of milk as the preferred form of nutrition intervention was highly effective. In 1928, Newman wrote that

"The rehabilitation of milk as the ideal food for children has proved an incentive for Authorities and their medical advisers, with the result that in a large number of areas milk has been tried as the answer to malnutrition" (27).

In the same year, the SMO for Stoke on Trent was quoted with approval

for adopting a 'cheap' and 'efficient' method of rectifying the deficiencies in the diets of poorer elementary school children. Rather than providing them with dinners, children were now given milk and cod liver oil (28). By 1930, milk was firmly established as a convenience welfare food. Whereas a decade earlier only six authorities had adopted milk feeding, now over half of all meals provided under the Education (Provision of Meals) Act took the form of milk (29).

5.5. The Use of Milk in School Feeding

Fig 5.1 shows the number of local authorities which supplied food under the Education (Provision of Meals) Acts of 1918 and 1921. Numbers increased during 1921 and 1926, which were periods of particularly severe economic stress for industrial communities (30). However, the number of feeding authorities remained stable in the period 1919-27; between 130 and 150 out of a total of 315 LEAs were feeding. The introduction of the NMPC's Milk Clubs, which were started in 1927-28, produced an undramatic upward trend. The number of authorities feeding did not in fact show a marked rise until 1933-4, when the Treasury subsidised Milk Marketing Board scheme was introduced. Cheap milk provided an inducement for authorities to become involved in nutritional supplementation. Using figures published in the CMO's annual reports, it is possible to analyse the nutritional impact of this flood of cheap milk.

Table 5.2 shows the number of meals supplied to each child selected for feeding between 1930 and 1937; this includes the two years preceding and succeeding the introduction of the Milk Marketing Board's (MMB) scheme. After 1933-4 more authorities became involved in school feeding and any child selected for feeding received more meals. However, of those meals, an increasing fraction was made up of milk. So, although the number of meals provided apparently rose, most of that rise was in milk. The

number of conventional school meals actually fell by about a third (see Table 5.3).

The nutritional effect of this change can be demonstrated by comparing the nutritional value of a conventional school meal with the nutritional value of 1/3 pint of milk (see Table 5.4). The conventional school dinner provided more than four times the calories and protein of milk. Not all non milk meals were as nutritious as the school dinner, which provided 750-900 kcals and 25 g protein (31), and there is no quantitative data on the breakfasts and teas fed. However, even if a conservative aggregate value of 600 kcal and 20 g protein per non milk meal is assumed, the introduction of the MMB's Milk in Schools initiative resulted in a 19% fall in protein and a 24% fall in the energy provided by each school meal. Because each child selected for feeding received on average more meals, this effect was nearly offset (see Table 5.5). However, the provision of conventional school meals in some of the poorest authorities, including Jarrow and Merthyr Tydfil fell dramatically when cheap milk became available (32). Complete statistics are not available, but such evidence as exists suggests that the adoption of the Treasury subsidised scheme was often followed by a fall in the availability of substantial meals (33).

Table 5.6 shows that the overall fall in the nutritional value of meals was achieved at a higher cost per unit of nutrient. The cost per Calorie of milk was nearly twice that of conventional school meals. Thus, nearly twice as many children could have been fed, or the same number fed more calories and protein, or feeding could have been undertaken for less money, if Local Education Authorities had not become involved with the MMB scheme. The desirability of such alternative strategies will be demonstrated in the following section.

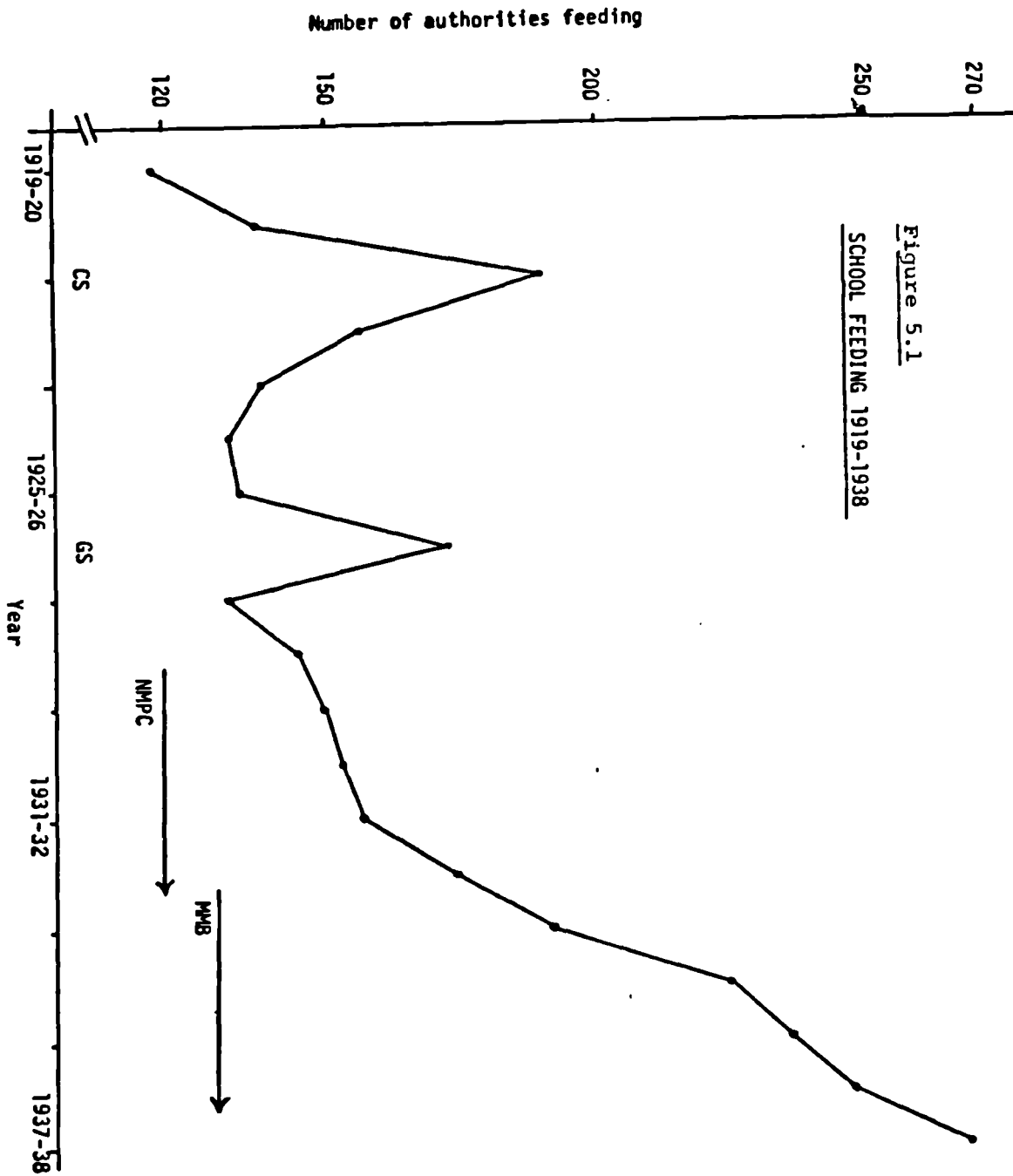


Figure 5.1
SCHOOL FEEDING 1919-1938

CS: Coal Strike (1921)

GS: General Strike (1926)

NMPC: National Milk Publicity Council scheme starts

MMB: Milk Marketing Board scheme starts

Source: Annual Reports of the Chief Medical Officer to the Board of Education 1919 to 1938, recalculated by the author.

Table 5.2

TOTAL NUMBER OF MEALS PER CHILD FED IN
SCHOOLS IN ENGLAND AND WALES, 1930-1937

Year	Total Meals Per Child
1930-31	137
1931-32	150
1932-33	156
1933-34	166
1934-35	168
1935-36	180
1936-37	187

Meals are either a conventional school meal or milk.

Source: Annual Reports of the Chief Medical Officer to the Board of Education, 1930-37.

Data recalculated by this author.

Table 5.3

MILK AS A FOOD FOR SCHOOLCHILDREN IN ENGLAND AND WALES, 1930-37

Year	% School Meals as Milk
1930-31	56.4
1931-32	56.1
1932-33	51.8
1933-34	54.4
1934-35	61.9
1935-36	73.1
1936-37	78.1

Source: Annual Reports of the Chief Medical Officer to the Board of Education, 1930-37.

Data recalculated by this author.

Table 5.4

NUTRITIONAL TARGETS IN SCHOOL FEEDING, 1919-39

	Cals	g protein
School Dinner	750	25
Aggregate Value of Non Milk Meals*	635	21
1/3 Pint Whole Milk	125	6

*Approximately 85% of the value of the conventional school dinner (see Section 5.5)

Source: Annual Reports of the Chief Medical Officer to the Board of Education, 1919-39.

Data recalculated by this author.

Table 5.5

CALCULATED NUTRITIONAL IMPACT OF SCHOOL FEEDING

	% meals as milk	per meal Cals	Protein (g)	per child fed Cals	Protein (g)
1930-33 (A)	54.8	356	12.8	52,600	1,891
1934-37 (B)	71.1	272	10.4	48,300	1,833
B % A	130	76	81	92	97

Source: Annual Reports of the Chief Medical Officer to the Board of Education, 1930-37.

Data recalculated by this author.

Table 5.6

COMPARATIVE COST OF NUTRIENTS IN CONVENTIONAL SCHOOL MEALS AND SCHOOL MILK, 1935

	Nutrients per penny	
	kcalories	protein (g)
Conventional school meal*(A)	247	8.6
School milk (B)	102	4.6
B % A	47	58

*Assumes 635 calories and 21g protein per meal.

Source: Annual Report of the Chief Medical Office to the Board of Education, 1935.

Data recalculated by this author.

5.6. An Evaluation of Milk Feeding as a Nutritional Intervention Strategy for Poor Children

Supplementary feeding was available only to children from families with exceptionally low incomes. Each local authority set its own income thresholds. Table 5.7 (34) illustrates thresholds operating in England and Wales in 1936. An estimate of the probable nutritional status of such groups can be gained from data collected in the Carnegie Survey of 1937. The Carnegie Survey is discussed fully in Chapter 6.2.3.1. It was undertaken by Boyd Orr's Rowett Research Institute and survey work was carried out by highly skilled clinicians and researchers selected personally by Boyd Orr (personal communication). Raw data from this survey was located by the author on the basement archives of the Rowett Institute.

Table 5.7

EXAMPLES OF LOCAL EDUCATION AUTHORITY (LEA) INCOME THRESHOLDS FOR THE PROVISION OF FREE MEALS, 1936-8

	Income threshold for free meals					
Number in family	2	3	4	5	6	7
Area						
Liverpool	16s	23s	29s	34s	39s	45s
Salford	18s	23s	27s	31s	35s	39s
Whitehaven	20s	27s	32s	37s	42s	45s
Radcliffe	22s6d	27s	30s7d	33s9d	37s10d	44s1d
AVERAGE ENGLAND & WALES	20s9d	25s4d	29s7d	33s7d	37s6d	41s9d

Source of data: Public Record Office, Board of Education Files (34)

The Carnegie Survey divided the population into income groups according to available food income per man value as follows:

Table 5.8

INCOME GROUPS I-IV IN THE CARNEGIE SURVEY (35)

Group	Income available for food per person per week		
I	up	to	2s 11 3/4d
II	3s	to	4s 11 3/4d
III	5s	to	6s 11 3/4d
IV	7s	to	8s 11 3/4d
V	9s	to	10s 11 3/4d
VI	over		11s

Comparison with Table 5.7 shows that the lowest two income groups were below the threshold for free feeding as described above. There are good grounds for predicting low nutritional status among these groups since the average income below which meals were provided free would have left 5/- per head for food according to contemporary calculations (36) whilst the estimated cost of BMA recommended diet in 1937 was 6s 9d (37).

Table 5.9 shows a nutritional assessment of children from among the target population for nutrition intervention. This suggests a high level of underfeeding, indicated by low weight-age and it might therefore be inferred that any policy which reduced the number of Calories fed was inappropriate. Although numbers presented here are limited, further anthropometric evidence appears in Chapter 6 which corroborates the view that an inadequate food intake was the main nutritional problem facing the poor in the UK in the inter war period. However, Medical Officers insisted throughout the 1920s and 1930s that an absolute shortage of food was no longer a problem and that the poor growth of working class children could be attributed to qualitative defects in their diets. The publication of Corry Mann's study "Diets for Boys During the School Years" appeared to provide unequivocal evidence in support of this view.

Table 5.9

ANTHROPOMETRIC EVIDENCE OF UNDERNUTRITION AMONG ELEMENTARY SCHOOL CHILDREN, CARNEGIE SURVEY; 1937

	AREA A	AREA B
No. studied	285	61
Percentage (number) below 3rd centile		
Anthropometric index		
Height for Age	22.8 (65)	38.6 (17)
Weight for Age	5.3 (15)	22.9 (14)
Weight for Height	2.1 (6)	4.9 (3)

Area A : Dundee, Tarves and Hopeman; Scottish urban and rural. Lowest income groups

Area B : Newport and Rhymney , S. Wales Coalfield: no income data
Subsistence Production society: agricultural community for long term unemployed

See Chapter 6 also for a full anthropometric analysis of Carnegie data and details of age and sex distribution in the survey population.

Source: Unpublished data held by the Rowett Research Institute. Anthropometric indices calculated by the author according to the criteria set out in Chapter 6, Section 6.2.2.

5.7. Diets for Boys During the School Age

It has been shown that, throughout the inter war period, the chief objective of school feeding was to supplement assumed qualitative defects in the home diets of elementary school children and that from the mid 1920s, milk feeding was consistently advocated by the CMO as the 'ideal supplementary ration'. The use of milk in school feeding received an immense boost from Corry Mann's feeding experiment (38) which was carried out, under the auspices of the Medical Research Council, at Dr Barnardo's Woodford Home between 1921 and 1925. This experiment was proposed by the Ministry of Health, which wanted evidence to encourage the use of milk both in schools and public institutions (39). As noted earlier, milk was mistrusted by many consumers at this time and had a reputation for being dirty, dangerous and disease ridden (see 5.1). Corry Mann's experiment was designed to show that growth in children was inhibited by NOT consuming a nutrient present in milk - the presence of such a 'growth factor' was first hypothesised by Hopkins in 1912, in an experiment described in Chapter 2.

5.7.1. Corry Mann and the MRC

Corry Mann's feeding trial was cited by Fletcher in 1932 as proof of the practical applications of the Newer Knowledge of Nutrition and in an article on the 'Urgency of Nutritional Studies' he suggested that this experiment represented one of the Council's most important contributions to public health (40). Yet, although final results were endorsed by the MRC, Corry Mann's experiment was threatened with premature curtailment in 1923 and again in 1924 (41), when serious doubts were raised by both the physiologist, D. Noel Paton (42) and by Fletcher himself (43) over the scientific value of the work. Fletcher was particularly concerned that the experiment did not merit its extremely high cost, which amounted to £2000 per annum, or approximately 3% of the MRC's annual research budget. However, it was backed by prominent members of

Figure 5.2. Milk Industry Propaganda based on Corry Mann's feeding trial, 'Diets for Boys During the School Age'



To illustrate the Medical Research Council's Report on the Diets for Boys during School Age. These figures represent groups of boys who were given an ordinary diet for a year. At the end of that period six groups were given the extras as shown. The average annual gain in weight and height of boys given a pint of milk daily was 6.68 lbs. and 2.63 ins. respectively, whilst the boys given no extras gained only 3.85 lbs. and increased in height only 1.84 ins.

Source: Medical Research Council

the MRC's Nutrition committees including Hopkins and J.C. Drummond. Hopkins presented the case for continued funding in 1923 and his faith in the usefulness of the work was such that he apparently neglected to study either interim results or familiarise himself with details such as the calorie value of the basic institutional diet before addressing the Council of the MRC on the value of the work (44). Hopkins argued that the experiment was of great national importance since he believed it would show how the physical standard of the British people could be improved. His own work had already convinced him that milk feeding would produce this objective (45). In 1924, Drummond argued the case for the experiment's continued funding on the grounds that it provided a unique opportunity to test laboratory results on human populations (46).

The trial was allowed to run its full course, and final results appeared to show that children grew better when they were fed milk than when they were fed either sugar, butter or vegetable margarine - described in the summary of results as having "equal calorie value" to the milk ration - or extra watercress or casein. These results were welcomed by Fletcher as conclusive evidence of

"what the results of laboratory experiments are continually teaching namely the unwisdom of giving a policy of better housing, desirable as that may be in itself, the priority over better nutrition" (47).

5.7.2. Corry Mann's Experiment: A Reassessment

Dr Barnardo's Home offered ideal conditions for a controlled feeding experiment. During the experimental period batches of 30 boys, living in separate houses, were fed various supplementary foods in addition to the institution's basal diet. A control group, fed the basal ration without supplements was also studied. Only healthy children were included in the experiment and an attempt was made to ensure that the groups were balanced in age and physical status. Children of 'non English type' were excluded. Maximum and minimum samples of actual servings were taken

throughout the year to ascertain the Caloric value of the basal ration (48) and in addition to Corry Mann two additional staff were employed to supervise these aspects of the feeding experiment. Corry Mann's results are reported in great detail and include records of the age, diet, initial height and weight and final height and weight of each subject. It has therefore been possible to check to validity of his work. It is clear from this that his work was flawed from the outset because his method of allocating boys to treatment groups failed to achieve the balance he had hoped for. In fact, the more poorly nourished boys tended to be placed in the groups where Mann might have expected the greatest increments in growth.

1. Height Increments

When height increments were reviewed, and boys re-grouped according to initial height for age status, it was found that non stunted milk fed boys did not grow significantly better than children fed other food supplements; indeed, they did not even grow better than children fed the basal, unsupplemented diet (see Table 5.10). However, milk feeding did produce significantly better growth than other food supplements among stunted children that is, children whose initial height for age was below the third centile (see Table 5.10). Since such children were over represented in the milk group. Corry Mann was therefore recording catch up growth in a group of chronically undernourished children, not 'super growth' in a group of already adequately nourished children, as was claimed.

According to current research milk does appear to be a good food for stimulating height growth in malnourished children. The reason for this is not yet clear: possibly calcium assists bone growth or reduces bowing of the legs; possibly protein or zinc is involved (49). However, Corry Mann's experiment was supposed to be concerned with the utilization of

Table 5.10

GAIN IN HEIGHT (cms) OBSERVED IN CORRY MANN'S EXPERIMENT

Diet	All subjects	Non-stunted subjects (a)	Stunted subjects
Milk	6.6 + 1.1 (41) ⁻	(a) 6.2 + 0.9 (19) ⁻	(b) 7.0 + 1.2 (22) ⁻
Butter	5.4 + 1.2 (26) ⁻	5.8 + 1.2 (13) ⁻	5.0 + 1.2 (13) ⁻
Sugar	5.0 + 1.3 (20) ⁻	4.6 + 1.3 (12) ⁻	5.7 + 0.7 (8) ⁻
Vegetable Margarine	4.6 + 1.2 (16) ⁻	4.5 + 1.9 (10) ⁻	4.8 + 0.2 (6) ⁻
Basic diet	4.7 + 0.9 (61) ⁻	4.65 + 1.0 (35) ⁻	4.7 + 0.9 (26) ⁻
Casein	4.45 + 0.8 (30) ⁻	4.2 + 0.6 (15) ⁻	4.7 + 0.8 (15) ⁻
Watercress	4.2 + 0.7 (26) ⁻	4.0 + 0.6 (15) ⁻	4.4 + 0.8 (11) ⁻

Source of data: H.C. Corry Mann 'Diets for Boys during the School Age' (5).

Calculations performed by the author.

- (a) There were no significant differences between the diet groups
- (b) Milk increments significantly greater than basic diet group

calories among adequately fed children, not with catch up growth or re feeding. Fletcher, in his article on the 'Urgency of Nutrition Studies' (50) argued that the children studied were already receiving a diet 'medically adjudged to be sufficient for healthy development' and living under 'ideal conditions of housing, exercise and fresh air.' He maintained that despite these favourable circumstances, children only attained their 'full potentiality' in terms of physical and mental growth when they received an extra ration of milk.

2. Weight Increments

Weight increments recorded in Table 5.11 appear to confirm the view that a growth promoting nutritional factor did exist in milk. Milk and butter supplements produced more weight gain than sugar and vegetable margarine supplements which were described as having an equal calorie value. However, it is clear from the methods section of the text (but not from the results or summaries) that the various food supplements were not of isocaloric value. Table 5.11 shows weight increments plotted against actual energy intakes, and there is a linear correlation coefficient of 0.68. Thus, weight gain varied with calories eaten, not because of some unknown factor present in milk.

Relative to calorie intake, recorded weight increments were, however, low throughout. The milk and butter fed groups, which received approximately 400 extra calories day⁻¹ gained an average of only 4 gm day⁻¹. This is about 20 times less than values found in modern nutrition rehabilitation (51). Similarly, boys did not grow well on the basal ration.

A re-evaluation of weight increments among the study population suggests that environmental conditions were far from ideal and that the caloric demands of the boys' "healthy outdoor life" explains their poor growth, rather than the lack of a nutritional factor present in milk. On the

Table 5.11

WEIGHT INCREMENTS AND ENERGY INTAKE

Diet	kcalories per day	weight gain in one year kg
Basic	1916	1.75
Watercress	1920	2.46
Casein	1990	1.82
Sugar	2264	2.24
Vegetable margarine	2295	2.36
Butter	2303	2.86
Milk	2304	3.17

Calories vs. weight gain, $r = 0.6767$

Source of data: H.C. Corry Mann 'Diets for Boys During the School Years' (5)

Calculations by this author

basal ration of 1900 calories per day, weight increased at a rate of about 4g day⁻¹, whereas normal modern growth in children of the same age as Corry Mann's subjects is about 8g day⁻¹. Using standard prediction equations (FAO/WHO 1985), the maintenance requirement of children with the average weight recorded in Corry Mann's experiment can be estimated to be 1400-1600 kcals d⁻¹. On the basal ration, this left 300-500 kcals for all activities, growth and thermogenesis. Although this figure is not unreasonable for modern children, it was clearly insufficient for the children studied. This re-analysis therefore provides an important historical insight into the actual conditions of life experienced by institutional children (and possibly many poor children) during the period.

It is not unreasonable to conclude that physical activities including farm work, and cold stress due to inadequate heating and inadequate clothing raised the boys' calorie requirements. For example, we know that they spent a great deal of their time out of doors for ten months of the year, that they never wore head gear "except for a few hours on Sunday", that their "knees [were] uncovered" and that during the summer their "forearms and neck" were "exposed to the sunshine and weather" (52). Of the extra 400 calories fed to the experimental groups, we can infer that a large proportion was spent on physical work and maintaining body heat, as well as on play and increased activity, as is normal in re feeding (53). This would account for the poor weight gain in response to supplementary feeding.

Corry Mann's results also refute the scientific hypothesis that growth in the study population was limited by a non caloric factor. The inference drawn from the experiment was that the partial efficiency for growth was improved by the presence in milk of a specific nutritional factor and therefore that by feeding children milk, extra growth per

calorie fed would be achieved. In fact milk and other calorie supplements gave the same partial efficiency for growth, that is about one gram for every 100 calories fed.

CHAPTER 6

AN EVALUATION OF NUTRITIONAL STATUS IN THE UK, 1900-1939

6.1. Clinical Evidence of Micronutrient Deficiencies

It has been shown in earlier Chapters that throughout the period 1900-1939 the nutritional problem facing the UK was perceived as one of qualitative dietary deficiency. This view pre dates the discovery of the vitamins and was put forward with great certainty by witnesses giving evidence to the Committee on Physical Deterioration in 1904 (1).

However, of the conditions which were believed to be associated with nutritional deficiency, (which included "adenoid overgrowth, mental retardation and increased susceptibility to zymotic disease" (2)) only rickets and anaemia can be accepted as unequivocal evidence of dietary inadequacy.

In framing his vitamin hypothesis Funk (3) clarified the definition of qualitative nutritional deficiency disease, and postulated a single deficit leading to a single clinical state. This hypothesis was analogous to Koch's postulates in microbiology, as Rivers has pointed out (4). Table 6.1 (5) shows the dates from which specific nutrient deficiencies were generally recognised as such by clinicians (even if their aetiology was not understood) and after such recognition was established, prevalence data can be used fairly reliably to establish the nature of the nutritional problems which existed during the period 1900-1939.

Table 6.1

CHRONOLOGY OF THE RECOGNITION OF SPECIFIC NUTRIENT DEFICIENCIES

DISEASE	DESCRIBED	NUTRITIONAL REMEDY	RECOGNISED AS DEFICIENCY DISEASE
Scurvy	antiquity & from 15th Century	from 16th Century	ca. 1918
Beri- Beri	antiquity and from 17th Century	from 1880s	ca. 1914
Night blindness and Xerosis Conjunctiva	antiquity from mid 19th Century	from antiquity mid 19th Century	ca. 1918 ca. 1918
Rickets	5th Century and from 1650s	from 19th Century	ca. 1918
Pellagra	ca 1730	from 1920s	1937
Iron deficiency anaemia	from 17th Century	iron treatment from 17th Century	1830s
Pernicious anaemia	mid 19th Century	from 1920s	1948

6.1.1. Vitamin A Deficiency

Writing of the period which preceded the Report of the Committee on Physical Deterioration, Drummond wrote

"Vitamin A deficiency must have been very prevalent in England in the nineteenth Century, particularly in the towns. The children were the worst sufferers...We know that the growth of these generations was stunted but the evidence that they showed signs of Vitamin A deficiency is remarkably slight. One can only conclude that night blindness, opacities of the cornea and other defects of the eye attributed to lack of Vitamin A were common but still unrecognised by the medical profession" (6).

The partiality of Drummond's conclusion, which assumed that a problem of Vitamin A deficiency must somehow have existed even if there was no evidence of it, reflects the professional attitude which prevailed throughout the inter war period. Yet, despite the belief that there was a widespread problem of Vitamin A deficiency (7) valid clinical evidence of this condition has always been hard to find.

It is convenient to divide clinical descriptions of Vitamin A deficiency into those which rely on symptoms of deficiency (notably nyctalopia or night blindness) and those which report clinical signs (such as xerophthalmia, xerosis conjunctiva, Bitot's spots and keratomalacia). These clinical signs are later signs of deficiency than night blindness, and are less prone to errors of interpretation. They therefore provide more reliable evidence of Vitamin A deficiency. Xerosis conjunctiva was first reported in 1803 (8). Xerophthalmia was identified by von Grafe in 1860 (9). Bitot's spots were described in 1862 (10) and a progression from xerosis conjunctiva to keratomalacia was recognised by European physicians by the late 19th century (11). Some clinicians associated these conditions with poor diet, but this view was not universal (12); the disease was, however, associated with poverty. Despite growing interest in the problem of poor nutrition, these clinical signs were seldom reported in the UK during the 19th and early 20th Centuries and the descriptions which do exist are of mild forms of the disease: in

1881 Snell described xerosis conjunctiva associated with nyctalopia and in 1898 Stephenson described its cure with cod liver oil (13).

Guggenheim (14) notes that, when a series of 1511 cases with keratomalacia were described in Japan in 1904, this extremely high prevalence aroused much interest.

McCullum claims in his History of Nutrition that in 1917 he and Simmonds

"expressed the belief that xerophthalmia or keratomalacia in animals was the analogue of the similar condition in humans, and that the disease was caused by a lack of Vitamin A" (15).

Certainly by 1919 this view was well established, and was put forward (without any reference to McCullum) in the MRC Vitamin Committee's report on the "Present State of Knowledge Concerning Accessory Food Factors", which appeared that year (16).

The idea that there was a high 'background' level of the disease during the inter-war period probably owes a great deal to Bloch's description of a major epidemic among infants in wartime Denmark (17), when an annual incidence of 50-80 cases of xerophthalmia per year was noted; this represented a 2-4 fold increase over pre 1914 values. Although Bloch's observations coincided with the rise of the Newer Knowledge of Nutrition, which produced considerable enthusiasm for the disease, unequivocal descriptions of the signs of Vitamin A deficiency in the UK remained rare. However, during the inter war period the criteria for diagnosing deficiency were liberal and this led to a belief that Vitamin A deficiency was widespread. For example, the existence of skin lesions in Vitamin A deficiency resulted in the use of skin signs as a single criterion for diagnosis and in the Carnegie Survey of 1937 (18) the reported prevalence rate of deficiency associated with these signs was as high as 20%. However, the skin signs used were relatively non specific: ichthyosis, eczema, psoriasis and even acne vulgaris were regarded as deficiency signs during this period (19). The failure to

apply rigid differential diagnoses to skin signs makes this data unusable as evidence for or against Vitamin A deficiency.

The softening of diagnostic criteria may also have produced exaggerated prevalences of xerosis conjunctiva. For example the use of slit lamp microscopy was adopted by Wiehl and Kruse in 1941 to permit the detection of marginal cases of xerosis conjunctiva and with its aid they reported 90% of school children examined in New York were, or had been, Vitamin A deficient (20). Although this study lies outside the period and country covered in this thesis, it provides a cautionary tale and demonstrates a trend which was well established during the 1930s, when both nutritionists and public health workers felt there was an urgent need to establish the presence of clinical signs for the deficiency diseases which they believed must necessarily be present in the community.

When Yudkin and Kodiceck used the same split lamp microscopy technique in the UK (21) they were more cautious in their interpretation of results and felt that normal variation could not be excluded as a cause of all apparently positive results. A subsequent study by Robertson and Morgan found no response to Vitamin A supplementation over two years in subjects diagnosed as Vitamin A deficient using the same technique, but found that 'symptoms' were related to outdoor sports activities (22).

In the light of the enthusiasm for Vitamin A deficiency, Spence's study, carried out at the Royal Victoria Hospital in Newcastle Upon Tyne in 1931 is particularly revealing (23). Spence studied the total annual ophthalmological list, which numbered 4,100 patients. In such a group the prevalence of ocular signs of Vitamin A deficiency might be expected to be highest-particularly in an area of high unemployment in one of the worst years of the economic depression. However, Spence found no frank disease and diagnosed xerosis conjunctiva in only four cases.

Thus, it seems reasonable to conclude that Vitamin A deficiency, as evidenced by ocular signs of the disease, was a clinical rarity in the UK during the period 1900-1939 and that it merited neither the extensive literature nor the preventive measures adopted. However, the belief that Vitamin A intakes were inadequate produced a series of investigations into sub clinical deficiency and work carried out on changes in function rather than observable lesions, appeared to justify the interest and concern which was sustained throughout the inter war years. Some of the functional changes investigated were ill defined. The most popular - susceptibility to infection - although real enough, is not sufficiently specific for diagnosis of deficiency. Impaired vision is the most unequivocal functional change associated with Vitamin A deficiency, particularly defective adaptation to vision in the dark (nyctalopia or hemeralopia). This symptom has been recognised for centuries and traditional Greek medicine even described liver as a cure (24). Yet, although Bitot described it in subjects presenting with frank ocular lesions, it seems that it was rarely reported by patients to physicians (25). Nevertheless it was the use of measures of dark adaptation which during the late 1930s gave rise to high reported prevalences of subclinical vitamin A deficiency.

In 1937, Jehgers (26) found 35% of American college students had abnormal dark adaptation and in the same year Maitra and Harris (27) tested for poor dark adaptation in the UK with the Birch Hirschfield photometer. Their technique involved the subject staring at a brightly illuminated (150 Watt) screen for 5 minutes, then immediately, and after 10 minutes in a darkened room, judging the minimal intensity of light which was visible through apertures in the photometer. With this technique Harris and his colleagues obtained extremely high prevalences of poor dark adaptation, which are summarised in Table 6.2. If these

Table 6.2

PREVALENCE OF POOR DARK ADAPTATION AMONG ELEMENTARY SCHOOL BOYS AND PUBLIC SCHOOL BOYS IN CAMBRIDGE, ACCORDING TO MAITRA AND HARRIS, 1937 (27)

	Elementary School Boys (n=100)	Public School Boys (n=30)
% definitely sub normal	23	0
% slightly below normal	34	10

result are accepted they clearly support the view of extensive social class related subnormal vitamin A status. However, Harris's results immediately arouse suspicion, because, in contrast to most tests of dysfunction, the incidence over the level of reported symptoms was increased by the photometer, rather than reduced. Complaints of nyctalopia were rare, with prevalences below 1% (28) yet photometry suggested a prevalence of disease which was up to 50 times as great.

Harris's results met with considerable contemporary criticism (29) which he and his collaborators attempted to refute (30), by publishing details of their validation. The dark adaptation of 22 boys who had "definitely subnormal" dark adaptation (according to Harris's criteria) and a further 32 classed as "slightly below normal" was measured initially and again after either two weeks' daily dosage of 1700 iu Vitamin A (510mcg retinol equivalent), or no treatment. The results, which are summarised in Table 6.3, show a statistically significant improvement in children treated with Vitamin A. However, these results must be treated with caution. Although Harris and Abbasey emphasise their use of a control group, their study was not a double blind, and, as their control data illustrate, the dark adaptation response was not constant, but improved with time. Other authors could not replicate Harris's results; Steinger and Roberts could not establish a relationship between dark adaptation and Vitamin A status (31) and Josephs, Barber and Conn (32) found impaired dark adaptation only in patients with the lowest vitamin A status. Workers from the Rowett Research Institute (33) also failed to show a high prevalence of impaired dark adaptation using the photometer in their population surveys, although they omitted the initial retinol bleaching step which involved staring at a bright light. Finally, Harris's own data published in 1952 (34) show the difficulty in detecting changes in short term dark adaptation with Vitamin A status: his 30 minute dark adaptation curves for subjects deprived of Vitamin A

Table 6.3

Effect of Vitamin A Treatment on Dark Adaptation
according to Harris and Abbasey, 1937 and 1939 (29)

(1) Control Tests on 'Definitely Subnormal' boys

Treated with Vitamin A			Not treated with Vitamin A		
Case	Reading at start of expt.	Reading after 2 weeks' treatment with 17,000 IU of vit. A daily	Case	Reading at start of expt.	Reading at end of 2 weeks
1	14, 6	6, < 2	12	14, 8	12, 8
2	14, 8	8, 2	13	14, 8	12, 6
3	14, 8	6, 2	14	14, 8	12, 6
4	14, 8	8, 2	15	16, 8	16, 8
5	14, 8	8, 2	16	14, 10	12, 6
6	14, 6	8, 4	17	14, 6	10, 6
7	14, 6	8, 2	18	14, 8	10, 6
8	14, 8	8, 4	19	14, 8	12, 6
9	14, 8	12, 4	20	14, 8	14, 6
10	16, 8	10, 4	21	14, 6	12, 6
11	14, 6	10, 4	22	14, 8	14, 8

Table originally published by Harris and Abbasey in Lancet, vol.2, 1939 (29)

for 1, 14, 21, 25 and 35 days show that although after 30 minutes in the dark there was a clear correlation between dark vision and days of deprivation, at 10 minutes in the dark no such gradation was visible. (Dark vision was measured as "the logarithm of minimal degree of illumination just visible".)

The results of Harris's pre-war work remain an unexplained anomaly. If Harris was correct, most children and a considerable fraction of the adult population suffered from marginal Vitamin A deficiency, which resulted in the impairment of ocular function. While this is possible, it is unlikely, since it is difficult to conceive of a population which was on the brink of nutritional deficiency yet produced so few cases of frank disease. A more likely explanation, given the disparity between Harris's results and others, is that deficiency did not exist.

The possibility of poor Vitamin A status cannot, however, be completely dismissed. In Moore's study of the liver vitamin A levels of 40 subjects who died in motor accidents during the period 1931-1935 (35) he found a median value of 220 iu Vitamin A/g liver (66mcg retinol equivalent (RE)/g) with a mean of 290 iu/g (87 mcg RE/g). Both his mean and median values are very low. When he repeated his observations on 71 subjects diagnosed as accidental deaths in 1941-44 (36), Moore obtained a mean of 455 iu/g (137 mcg RE/g) and a median of 324 iu/g (97 mcg RE/g). In 1978, Huque and Trusswell (37) found a mean of 270 mcg RE/g (900 iu/g) in livers taken at autopsy of accidental death cases in London. This rise may owe something to the population groups studied (as Moore suggested might explain the differences in his two surveys) and changes in technique between Moore's work and that of Huque and Trusswell may also be relevant. Nevertheless, it is likely that a real rise has occurred in the post war period and that this indicates an improved Vitamin A balance. But since Vitamin A stores rise with excess intake this does

not show that a deficiency existed in the period 1931-1935. The difference in median and mean values reported by Moore suggests that some subjects had very high vitamin A levels and Moore's histogram of individual results confirms this. Conventionally, a level of 40 mcg RE equivalent per gram of liver (133iu/g) or below is taken as a low liver concentration. From Moore's histogram (38) it appears that 12 (30%) of his subjects, were at or below this level and were therefore only just consuming sufficient Vitamin A to meet requirements. This may indicate a low intake, but it is by no means exceptional: 20-30% of subjects in Canada and the USA are today placed in this category, while the figure for Bangladesh (where Vitamin A deficiency is endemic) is 78% (39). These adult data therefore contradict, rather than confirm the notion that there was a widespread problem of vitamin A deficiency in pre-war Britain.

6.1.2. Vitamin C Deficiency

Although increasing emphasis was placed on the importance of Vitamin C sources in the diet during the period 1900-1939, scurvy was an even rarer phenomenon in the UK than Vitamin A deficiency. As Carpenter has shown (40) scurvy was frequently reported during the 19th Century, but it was confined almost exclusively to populations living on dry rations: outbreaks were therefore associated with armies in the field (as in the Crimean and American Civil wars), prison populations, and populations under seige (for example Paris in 1870). The only significant outbreak in the free living population was in Ireland in 1847, when the potato crop failed. Otherwise, the importance of the potato as a British staple food protected the population as a whole from the disease, and when it did occur, it was met with considerable medical interest (41).

However, towards the end of the 19th Century, scurvy was widely reported among infants in higher socio-economic groups and infantile scurvy

became separately diagnosed. The disease was known as Barlow's disease after its discoverer, and as Carpenter has noted in his comprehensive review, it was a disease of affluence, occurring among middle class infants fed cows' milk which had been sterilized (42). This process of course destroyed the Vitamin C content of the milk. Thus, although the disease was relatively common, it was not indicative of or associated with deficiencies in the general diet of the population. By 1918 it was generally accepted that infantile scurvy was caused by the overheating of cows milk, and the use of known anti scorbutics was recommended for artificially fed infants (43): this led to the gradual disappearance of the disease.

As with Vitamin A deficiency, it was claimed during the inter-war period that sub clinical deficiency existed in the general population and, despite the absence of frank deficiency disease, inadequate Vitamin C intake was cited as the cause of a number of problems, most notably a predisposition to infectious disease. The objective assessment of deficiency was pioneered by Harris and his colleagues, who developed a titration method for measuring ascorbic acid in urine (44). This assay made use of two facts associated with vitamin C deficiency: firstly, that a reduction in the Urinary ascorbic acid excretion occurs, and secondly, that a test dose of Vitamin C fails to increase urinary excretion if body pools are not saturated with Vitamin C. Using this test, Harris, Abbasey and Yudkin (45) reported that sub optimal intakes of Vitamin C were a "common occurrence". However, these studies set the optimum intake at very high levels. For example, modern experiments show that excess Vitamin C is only passed into urine when the blood concentration is greater than 1.4 mg/dl: on a daily dose an intake of at least 100 mg/day would be needed to sustain this blood level. Thus, the measurements of Harris and his collaborators merely record the number of subjects consuming less than three times the modern UK RDA of ascorbate

and for this reason, modern commentators have concluded that

"estimations of the urinary output of ascorbic acid are of no value in assessing the risk of scurvy" (46).

Although a technique for diagnosing sub clinical deficiency based on a reduced capillary resistance was developed in Sweden by Gotlin (47), it was not used in the UK during the period studied. There is therefore no clinical evidence of frank Vitamin C deficiency in the normal population and no evidence to warrant the hypothesis of subclinical disease.

6.1.3. Nutritional Anaemia

Although anaemia was often inferred from clinical examination or case history, valid survey data, based on haematological examination, was relatively rare before the 1930s. However, during the pre war decade a number of useful studies were carried out.

In 1931-2, Davidson, Fullerton, Howie, Croll and Orr (48) conducted a survey of poor persons attending the Aberdeen Dispensary. Their data, shown in Table 6.4, are revealing. Firstly, they show that, in the sample of 100 children studied, there was no severe anaemia and only two cases of mild anaemia. This was not statistically different from the prevalence of 1% mild anaemia found by Sutherland on the basis of a clinical examination of the same community (49). Adult males had significantly more more anaemia than children, although the prevalence was still only 11% among this group, including both mild and severe cases. A far higher prevalence was found among adult women: 14% were suffering from severe anaemia and 33% were from mild anaemia. However, it must be admitted that, if anaemia is regarded as a haemoglobin level below the normal range, then Davidson et al underestimate the true prevalence of anaemia in adults. Their Group I (normal) includes subjects with a haemoglobin of >117g/l. The lower end of the 95% range for adult males in modern European populations is 130g /l, and for women

Table 6.4

PREVALENCE OF ANAEMIA AMONG THE ABERDEEN POOR REPORTED BY DAVIDSON
ET.AL. 1933 (48)

		% with Mild Anaemia	% with Severe Anaemia
Children	Total number examined 100	2.0%	0
Adult males	45	6.7%	4.4%
Adult females	455	33.0%	13.8%

Mild anaemia was defined as a haemoglobin of 84-70% of Haldane's standard.

Severe anaemia was defined as a haemoglobin of <70% of Haldane's standard.

118 g/l (50). For children the 5th centile is also lower and varies with age. However, the definition of anaemia as below normal haemoglobin is an overly generous one. While the normal haemoglobin range reflects intakes and losses, the lower end is far above levels at which functional impairment can be demonstrated. Moreover, it should be noted that of the 5 adult males Davidson et al diagnosed as anaemic, 3 had diseases which might be expected to reduce haemoglobin levels. Their study therefore suggests that anaemia was only marginally important in children and adult males, but widespread in women.

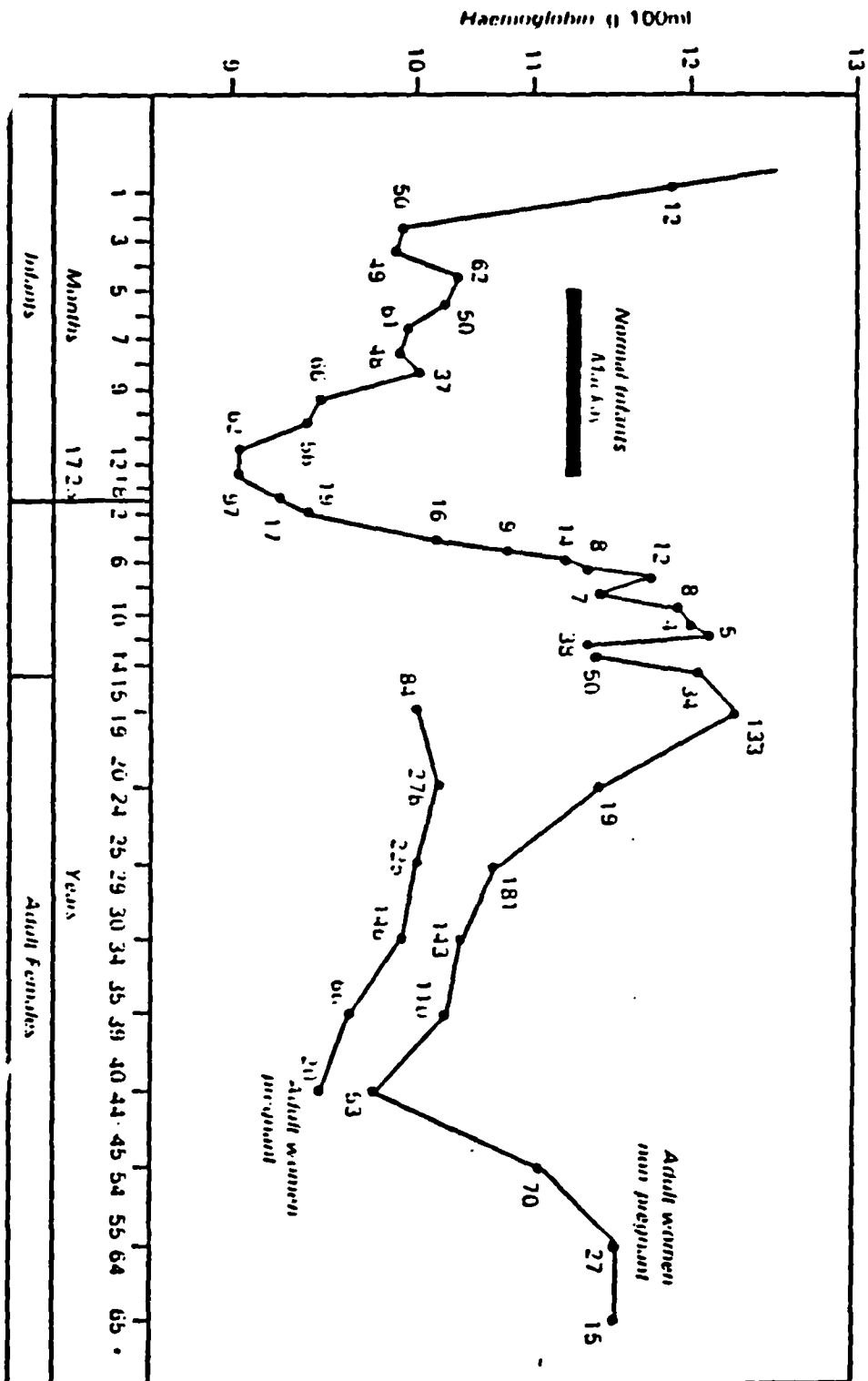
The 1937 Carnegie Survey (51) also investigated the incidence of anaemia in children and reported haemoglobin levels by age, sex and social class in 2,965 subjects aged 0-15 years. Unfortunately, the extent of breakdown in analysis means that the group size is often as low as 2 subjects and in Table 6.5 results have been partly re-aggregated to give numbers which are large enough for meaningful analysis. This survey confirms the impression of Davidson et.al., in showing that mean haemoglobin levels for children were within the normal range. However, since these data do not provide an indication of the distribution of values within each group, they do not provide direct evidence of the extent of anaemia.

Thus, the weight of clinical evidence suggests that anaemia was only marginally important in children and adult males, but widespread among women aged 15-45 years and infants: using the same diagnostic criteria as Davidson, Mackay also found one third of women bringing children to the Queen's Hospital for Children, London, were severely anaemic (52).

Figure 6.1 (53) shows mean haemoglobin values found by Mackay among poor children in London and by Davidson et.al. among adult females in their Aberdeen study of 1931. This indicates low haemoglobin values in infants and progressively higher values with increasing age until adolescence.

Figure 6.1

Mean haemoglobin levels recorded by Mackay (1935) and Davidson et al. (1933)



Source: Passmore and Eastwood (57)

Table 6.5

Mean Haemoglobin Levels Recorded in Carnegie Survey 1937

(Figures in brackets show number of subjects in group)

Age in Years	Income Group		
	I and II	III	IV, V and VI
BOYS			
0-2	80.0 (140)	78.7 (72)	80.7 (25)
3-4	86.1 (107)	81.8 (47)	88.8 (20)
5-6	87.9 (143)	88.1 (68)	90.0 (32)
7-8	91.4 (126)	89.4 (75)	90.8 (47)
9-10	92.0 (110)	93.6 (73)	92.7 (45)
11-12	93.6 (97)	95.4 (42)	96.7 (48)
13-14	94.4 (63)	96.3 (30)	97.0 (29)
Mean	89.3	89.0	90.0
GIRLS			
0-2	80.3 (154)	78.7 (50)	84.8 (38)
3-4	87 (125)	87 (54)	85 (17)
5-6	90 (155)	90 (57)	89 (44)
7-8	92 (135)	93 (61)	93 (45)
9-10	96 (136)	95 (53)	94.5 (51)
11-12	96 (104)	97 (63)	97.3 (44)
13-14	97 (52)	97 (38)	100.8 (50)
Mean	91.2	91.1	92.1

Source of data: 'Diet and Health in Pre-War Britain' (The Carnegie Survey) (51)

Data recalculated by this author.

After what may be assumed to be menarche, haemoglobin levels decline progressively until 40-44 years (presumably menopause) when they begin to rise again. Although these data are cross sectional, and compiled from different surveys, they nevertheless support the view that poor diet was not the only factor which produced a high incidence of anaemia in adult women but that female reproductive functioning also played an important part in its aetiology. The lower values for pregnant women confirm this view.

Davidson et.al. (54) found all cases of anaemia were microcytic and normochromic, characteristics which indicate that simple iron deficiency was the primary cause. They confirmed that this was the case in a sample of 20 severely anaemic women, who were treated with "30 grains of iron ammonium citrate 3 times a day" for between 14 and 97 days. In 19 of these women, both haemoglobin and erythrocyte count were increased by the treatment. Davidson et al found the best predictor of anaemia in women was menorrhagia, pregnancy or recent childbirth, with only a weak association with income.

Bradford Hill showed in an analysis of MacKay's MRC study (55) that post partum haemoglobin levels in women could be statistically significantly increased by iron supplementation in pregnancy. In controls it rose by 5.7% post partum, in iron supplemented women it rose by 10.5%. However, iron supplementation had no significant effect on mean haemoglobin in pregnant women, which rose by only 2.3%. Low folate levels possibly contributed to this-as still occurs. However, this explanation is difficult to reconcile with Davidson's assertion that anaemia in these women was microcytic.

MacKay's studies of anaemia in infants demonstrate that anaemia was a problem in the first 12 months of life. Statistical analysis of her results by Bradford Hill shows that haemoglobin level was positively

correlated with birth weight and negatively correlated with rate of growth (56). Birth weight and rate of growth were, however, correlated and when this was allowed for, there was no relationship between growth rate and anaemia, indicating that anaemia was a consequence of low birth weight (LBW). This probably reflects the fact that haemoglobin levels in the infant are dependent on status at birth. The LBW children did not deposit sufficient iron stores during gestation and as growth proceeded, they became anaemic. Although, as Mackay showed, this anaemia could be treated by feeding milk fortified with iron ammonium citrate, it was self correcting if the mother's nutritional status allowed for normal foetal growth and birth weight.

6.1.4. B Vitamin Deficiencies

Two of the earliest deficiency diseases to be described were beri beri and pellagra. However, neither of these conditions was reported in the UK during the period 1900-1939 - although the Wernicke-Korsakoff syndrome (cerebral beri beri) was observed in some hospitalised alcoholics (57). Since both pellagra and beri beri were recognised as clinical entities during the 19th Century, it is difficult to believe that, had they existed, they would not have been described.

Folate and Vitamin B12 deficiencies were not fully discriminated until the 1940s (58), but a syndrome of pernicious anaemia had been identified by Wills in India, which she reported in 1931 (59). True pernicious anaemia is a physiological B12 deficiency due to malabsorption of the vitamin, caused by the lack of an intrinsic factor. Dietary deficiency is not therefore the cause. However, folate and B12 deficiencies may have been confused with true pernicious anaemia, both presenting as megaloblastic anaemia. It is certain that pernicious anaemia was sometimes treated as a dietary deficiency, and the term was sometimes over extended to include cases of severe anaemia. However, pernicious

anaemia, or megaloblastic or macrocytic anaemia were not widespread clinical problems. In their population survey, Davidson et al noted that all the anaemia they described was normochromic or microcytic and therefore a result of iron deficiency rather than a lack of B vitamins (60).

6.1.5. Rickets

There are wide variations in the reported prevalence of rickets throughout the period of study, which reflect an absence of agreed criteria for its diagnosis. This problem is illustrated both in evidence presented to the Committee on Physical Deterioration in 1904 and in the debates over the prevalence of malnutrition of the 1930s.

For example, in 1904 Sir Charles Cameron, Medical Officer of Health for Dublin, maintained in his evidence to the Committee on Physical Deterioration (61) that he "very rarely" saw any rickets (CPD Q. 11086) whereas another Dublin physician stated that 20 cases out of 258 patients admitted to the Children's Hospital showed signs which were "generally (called).... rickets" (CPD Q. 12716).

In his survey of rickets in Leeds, Dr Hall reported a prevalence of 50% among poor non Jewish children, only 7% among poor Jewish children and 8% in a "good" school. However, an experienced teacher maintained that the prevalence of 'visible deforming rickets' in Leeds had decreased in the past 28 years from over 18% to less than 2% (CPD Q.552).

Dr Eichholtz, who had carried out a clinical survey of rickets in some of the main industrial towns, maintained that these wide discrepancies could be explained by differences in the expertise and experience of the observer. In his evidence to the Committee, he emphasised the distinction between

"disabling and deforming rickets and rickets which, though present, may be non disabling and practically of no effect as regards deformity, which may in fact be non apparent to any but the expert eye." (CPD Q. 451)

Eichholtz cited Dr Hall's figure of 50% rickets in a poor Leeds school as 'correct', but maintained that only 2%-3% of these cases would be detectable to the 'non expert eye' (CPD Q. 451). Similarly, Dr Ashby reported that in a survey of 750 children examined in Manchester on account of their "inability to learn in class", only 2.4% were suffering from marked rachitic deformities (CPD Q. 8774) - these were described as "bona fide dwarves" - but that "traces of rickets" were discernable in "a large number" of the other children examined.

Despite disagreements over its prevalence, there was a general consensus among witnesses that the severity of rickets had declined in the period which preceded the convening of the Committee. Dr Scott reported that the prevalence of rickets in Glasgow in 1884 had been 1.16% in a 'higher' working class school (CPD Q. 1681), 22.57% among poorer Glasgow children and only 6% among rural children (CPD Q. 1712) but that by 1904 the problem was "far less severe". Dr Young spoke of a decline in the prevalence of severe rickets in Liverpool and claimed that

"the malformed rachitic head and the enlarged joints were less marked than they had been in the past" (CPD Q. 2142),

and finally, Mrs Greenwood described the number of deformed (rachitic) people in Sheffield as "something terrible" (CPD Q. 8188). Whilst 'only' 114 deaths had been attributed to rickets in the past 10 years, Mrs Greenwood observed that rachitic children died from "other things-from bronchitis and convulsions" (CPD Q. 8257).

During the inter war period discrepancies in the reported prevalence of rickets persisted. For example, a survey carried out by the MOH for Durham in 1920 indicated a prevalence of over 80% (62) whereas Tully's

anthropometric survey of Glasgow school children of 1922-23 (63) reported a prevalence of only 4.6%; both these areas, however, had a reputation for "widespread rickets".

In 1927, the London County Council (LCC) undertook a survey of 1,638 unselected 5 year old children as part of a Board of Education investigation into the cause of adenoids and tonsillar enlargement which indicated that 66.1% of all children examined had more than one sign of rickets. (These signs included knock knees, bow legs, parietal bossing and 'deformity of the chest wall') and that only 12.5% had none of these signs (64). The most prevalent defect, occurring in 63% of children, was knock knee, which would not now be accepted as a sign of rachitic deformity. This was followed by parietal bossing, found in 58% of children, which would be accepted as evidence of rickets. Table 6.6 shows the prevalence of rickets reported in routine medical inspections carried out during the same year as the LCC survey.

Table 6.6

PREVALENCE OF RICKETS IN 15 INDUSTRIAL AND NON INDUSTRIAL AREAS RECORDED BY SCHOOL MEDICAL OFFICERS, 1927 (65)

County Council Borough:	% of children examined given subsequent treatment	% of children examined in which rickets noted but not treated	Total Prevalence %
Swindon	3.2	0.4	3.6
Sheffield	1.9	1.2	3.1
Sunderland	2.6	14.0	16.6
Jarrow	0.9	0.9	1.8
Bradford	8.9	5.8	14.7
Essex C C	0.08	0.7	0.8
Leeds	8.3	8.3	16.6
Hartlepool	2.5	5.1	7.6
N. Riding	2.7	0.9	3.6
E Riding	0.4	0	0.4
Tynemouth	1.5	0.6	2.1
Bath	1.2	3.3	4.5
Birmingham	6.0	5.1	11.1
Gateshead	9.4	1.8	11.2
London CC	0.5	0.19	0.7

Source: M'Gonigle and Kirby 'Poverty and Public Health' (65)
Recalculated by the present author

In even the most heavily industrialised urban manufacturing towns, the prevalence of rickets reported in the annual returns of School Medical Officers (SMOs) did not approach the rate reported in the Board of Education's investigation. This could be interpreted either as proof that ordinary SMOs lacked the "expert eye" of the special inquiry team or that members of this team were over enthusiastic in their recording of rachitic stigmata.

The returns shown in Table 6.6 also demonstrate wide discrepancies between similar industrial towns in the reported prevalence of rickets which reflects the continuing confusion over its diagnosis. This became an important issue in the course of the malnutrition debate of the 1930s: for example, in 1937 it gave rise to a heated exchange between Dr Herd, SMO for Manchester, and Dr Wilkins, SMO for Birmingham. Herd maintained that in Manchester the prevalence of rickets had declined from 7.3% in 1919 to 1.07% in 1937 (66) whereas Wilkins, an 'enthusiast' reported prevalences of "rachitic deformities" of over 89%. In 1937 he published the following data for Birmingham children: (67)

RACHITIC DEFORMITY	PREVALENCE
Defomity of the chest wall	89.4%
Knock Knee	76.7%
Bossing of parietal bone	74.2%
Bowleg	0.7%

Although high prevalences of rickets were reported throughout the period 1900-1939, the severity of the disease appears to have declined during the inter war years and the high prevalences reported during the 1930s presumably reflect a softening of the clinical criteria for its diagnosis. This change in the nature of the disease was almost certainly due to a decline in the dietary requirement for Vitamin D resulting from clean air legislation and the subsequent reduction in atmospheric pollution.

The comments of Dr Chalmers, whose career as Glasgow's MOH spanned the pre 1914 and inter war years are extremely useful in this context. Chalmers wrote in 1932 (68) that he had observed a dramatic change in the nature of rachitic deformities since the beginning of his career and argued that these changes could only be explained by the improvements which had taken place in environmental conditions. In contrast to the prevailing MRC view described in Chapter 3, he maintained that

"the prevention of rickets.. is not a question of dispensing a few tablets or doses but of maintaining hygiene in every direction".

Rickets was undoubtedly a problem in the industrial cities of the UK throughout the first half of the 20th Century. High levels of atmospheric pollution led to an elevated dietary requirement for Vitamin D which, at the turn of the Century, the normal British diet could not supply: it seems that among the urban poor only the Jewish community was able to avoid the disease, through its use of oily fish and animal fats (69). The problem of rickets became progressively less severe (and probably less prevalent) during the course of the century - not, however, as a result of nutritional intervention, but rather due to changes in environmental conditions. Nevertheless, on the outbreak of World War II the view that rickets remained a major problem which required blanket intervention was axiomatic among the government's nutritional advisors and it led to a policy of mass fortification of common foodstuffs combined with free cod liver oil supplements to women and children. This over-enthusiasm for Vitamin D deficiency had fatal consequences and resulted in a number of avoidable deaths from hypercalcaemia (70).

6.2. Growth and Nutritional Status

6.2.1. Interpretation of Anthropometric Data in Historical Studies

Growth data for the period covered by this thesis are relatively abundant and records of height and weight have been located in military,

charitable and local authority archives. Interest in the monitoring of growth was stimulated in the last decades of the 19th Century by the anthropometric survey carried out by the British Association for the Advancement of Science in 1881 (71) which was undertaken to discover the average stature of the UK population. The quality of data collected during this period can be accepted as reasonably accurate; not only was there an adequate technology for the measurement of both height and weight by the end of the 19th Century, but physical anthropology was a prestigious and precise science.

Growth data exist for isolated population groups from the 18th Century and these have been used by Floud and his collaborators in a series of studies which use mean heights of children and adults as a proxy indicator of socio economic disadvantage (72). Whatever their use as a standard of living indicator, it is difficult to use such data as a measure of the nutritional status of individuals. Mean values for attained growth within the population are of course affected by the values for all individuals; however small deficits in growth in a large number of individuals or large deficits in a small number could result in the same changes in the mean. These situations have radically different health inferences: according to contemporary observations, the increased risks of mortality associated with small deficits in growth are negligible but these rise steeply below a threshold which is roughly equivalent to the 3rd centile (73), indicating that while a small growth deficit spread across the whole population would have a minimal effect on health status, a large deficit in a small sub group would have a major impact. It is not proposed to explore this effect thoroughly in this thesis, but merely to illustrate its importance by considering the problem for a hypothetical population of 5 year old boys.

Table 6.7

TO ILLUSTRATE DIFFICULTIES IN ASSESSING RATES OF STUNTING FROM MEAN HEIGHT DATA

EFFECT OF MEAN HEIGHTS ON PERCENTAGE FALLING BELOW THE THIRD CENTILE OF HEIGHT FOR AGE, IF VARIANCE IN HEIGHT IS UNCHANGED

5 year old boys

Mean height (cms)	109.9	108	106	104	102
% below 3rd Centile	3	7.3	15.9	30	44

EFFECT OF A 1% FALL IN MEAN HEIGHT ON PERCENTAGE FALLING AT OR BELOW THE THIRD CENTILE OF HEIGHT FOR AGE : 5 year old boys.

1% fall from (cms)	109.9	108	106	104	102
% below 3rd Centile	5	11	21	36	56
increase in % below 3rd Centile (A)	2	4	5	6	12

A SELECTIVE INCREASE AT THIRD CENTILE NEEDED TO PRODUCE 1% FALL IN MEAN HEIGHT

Mean height (cm)	109.9	108	106	104	102
% increase at 3rd centile required	8	16	23	39	80
Ratio B/A	4	4	~4.6	6.5	6.5

Data are from NCHS 1983 (75).
Calculations by this author.

If the height for age of these boys were normally distributed according to NCHS (1983) standards, with a median value of 109.9 cms and standard deviation of 4.6 cms, a general reduction in height for age of 1% in all subjects would increase the fraction below the 3rd centile by 2%, from 3% to 5% of the population. But equally, a population composed of two subgroups, one comprising 11% of the total with height for age at the 3rd centile and the other 89% of the total with height for age distributed according to NCHS standards for 5 years olds, would also have a mean height 1% below 109.9 cms. It is therefore impossible to interpret the fact that a mean is 1% below standard, since in this example 5% to 11% of the population could be malnourished to a degree that significantly increases their risk of death. Moreover, the lower the population mean, the larger are such effects: if the mean height of the 5 year olds were 106 cms and the standard deviation still 4.6 cm, 16% of the population would be below 101.3 cm in height (ie the 3rd Centile). A general 1% fall would increase the fraction below this threshold from 16% to 21%. However, a selective rise in the fraction at the threshold from 16% to 39% would also be reflected in a fall in the mean of only 1% (see Table 6.7). Thus, while population means should correlate broadly with the prevalence of malnutrition, there is so much noise in the system that changes in mean height cannot be interpreted unequivocally in terms of prevalences of stunting. Although little use has been made of changes in mean weight for age, the same conclusions apply, despite the fact that the standard deviation of weight for age is larger than that of height for age; indeed, interpretation of mean weight for age data is made more problematic by the fact that the distribution tends to be positively skewed.

For the purposes of this thesis it was therefore decided to concentrate upon the analysis of disaggregated data, rather than on mean trends, for the study of nutritional status. This is particularly important, as

both evidence presented to the Committee on Physical Deterioration in 1904, and in the inter-war reports of School Medical Officers, suggest a low mean level of attained growth in children, with mean heights at around the 20th centile of NCHS standards or below (74).

6.2.2. Methodology of the Assessment of Nutritional Status by Anthropometric Examination

Anthropometric examination of nutritional status, like clinical assessment measures the state of the organism. But whereas in clinical assessment there is an accepted cut-off between health and ill health (where deficient nutrient intake impairs structure or function) in anthropometric assessment the degree of possible impairment is continuous and the degree of impairment which is regarded as abnormal is a matter of scientific dispute. However, it is now recognised that anthropometric data are crucial in attempts to estimate the nutritional status of the population. They are seen to have an objectivity and precision often lacking in clinical studies and provide the best measure of the extent of underfeeding (quantitative malnutrition) as opposed to specific nutrient deficits.

The techniques employed for the analysis of anthropometric results were those recommended by WHO in 1983 (75) with some modifications which were demanded by the nature of the data. The standards used for attained growth were those of the National Centre for Health Statistics (NCHS) as published by WHO (76). These reference values (called hereafter the NCHS standards) are based on measurements of healthy children in the USA. Attained growth can be measured by two indices commonly known as height for age (H/A) and weight for age (W/A). In each of these indices the attained value for the parameter is compared with the distribution of the NCHS reference population at that age.

When these indices are used to diagnose malnutrition, it is necessary to define a cut off, below which a value of H/A or W/A is to be regarded as outside the normal range. In this study, the third centile of weight or height for age is used as the cut off. This corresponds to a Z-score of -1.88; a Weight for Age value of 78%-82% of the median (depending on age); and a height for age value of 88%-92% of the median. By comparison, in the Gomez classification children with weight for age values of less than 80% of the median are called "moderately malnourished" (77) and in Waterlow's classification children with height for age values below 90% of the median are called stunted (78). The term "stunting" is used to here to describe height for age below the third centile but the term low weight-age, proposed in 1934 (79) has been used to describe children below the third centile of weight for age.

Use is also made of weight for height, an index which is regarded as age independent. According to a view put forward by Waterlow (80) and widely endorsed (81) the rate of weight gain and height gain in chronic malnutrition are impaired in proportion, with the result that the child's weight for height is unchanged. However, in acute (ie severe and short term) malnutrition, weight is lost and no height is gained, with the result that the child's weight for height declines. Thus, it is argued that the prevalence of low weight for height can be used to differentiate acute from chronic malnutrition.

6.2.3. Anthropometric Measures of Nutritional Status 1900-1914

6.2.3.2. Description of Data 1900-1914

The first data set is a survey of the entire elementary school population of Glasgow, undertaken by Dr Ethel Elderton in 1906 (82). Elderton's data were published as tabulated frequency distributions of height and of weight at each age. The cell size used in tabulating the distribution was sufficiently small - 1 inch (1.252 cm) and 2 lbs

(0.91kg) - that by treating the mean for a cell as the actual measurement of children in that cell, only small errors were introduced. Children in this study were measured in ordinary indoor clothing, but without boots, and to take account of this, children in the cell above the third centile of weight for age were also included for the purpose of interpretation of the data. Since on average a cell at this level of weight for age contained only 2-3 children, it was evident that clothing made only a very small difference in results, and it was therefore possible to compare the data directly with NCHS standards.

Elderton classified her results according to a measure of social class, so her data can also be used to study the relationship between poverty and growth failure, at least in Glasgow. The survey schools were classified "A" to "D" according to the estimated poverty of the district in which they were located. This was a valid procedure since children always attended local schools and economically, districts were relatively homogeneous. Class 'A' represented the poorest class and class 'D' the most prosperous, which would have included the children of skilled artisans.

The registers of the Poor Law Children's Homes of various London Boroughs provide a second source of anthropometric data. This material is unpublished but was located by the author in the record office of the former Greater London Council and results for a random sample of 80 children aged between 6-9 years old, who were admitted to the Homes between 1908 and 1912, have been analysed. The children represent the poorest section of London's population. Although there is no indication in the registers whether they were weighed clothed or unclothed, it is likely that the measurements were taken during an initial medical examination and that the children were therefore unclothed. In order to

preclude the possibility of overestimating growth failure, this assumption has been made in analysing the data.

Disaggregated anthropometric data have also been located for the last years of the period covered in this study. These consist of measurements gathered by Boyd Orr's Rowett Research Institute in 1937 as part of a nation wide survey into relationships between food intake and health, funded by the Carnegie Foundation. The study was eventually published in 1952 under the title DIET AND HEALTH IN PRE WAR BRITAIN (83), and the initial research plan was to conduct dietary surveys of 2,000 families at different social and economic levels. Children and adolescents from these families were subjected to at least one clinical examination, which included the recording of various anthropometric measurements. Although only mean heights and weights are shown in the published data, the archive material which was found by the present author in the Rowett Institute provides individual results for 574 children aged under 10 years.

6.2.3.2. Results

Elderton's results for height for age and weight for age are given in Tables 6.8 and 6.9. These results demonstrate that stunting and low weight-age were widespread among both boys and girls, although stunting was much more prevalent than low weight-age. For all classes, and overall, stunting was significantly more prevalent in boys than in girls ($p < 0.05$). The same overall tendency was observed for low weight-age but individual class rates sometimes failed to be statistically significant. Overall, stunting was 2.70 times more prevalent in boys and 2.78 times more prevalent in girls. These ratios are not significantly different. However, the ratio of the prevalence of stunting to low weight-age varied significantly with class and age. As Table 6.10 shows, the ratio fell with increasing age and with the progression from Class "D" to

Table 6.8

PREVALENCE OF STUNTING IN GLASGOW 1906,

		SCHOOL CLASS				
		ALL	A	B	C	D
BOYS						
5-6 years	Examined	3,322	1,244	1,161	500	417
	Stunted	1,971	870	652	281	168
	Prevalence of Stunting	59.3	69.9	56.1	56.2	40.2
8-9 years	Examined	4,009	1,402	1,449	630	528
	Stunted	2,559	1,028	938	368	225
	Prevalence of Stunting	63.8	73.3	64.7	58.4	42.6
GIRLS						
5-6 years	Examined	3,104	1,143	1,141	431	389
	Stunted	1,572	702	543	204	123
	Prevalence of Stunting	50.6	61.4	47.5	47.3	31.6
8-9 years	Examined	3,817	1,314	1,360	639	504
	Stunted	2,178	889	753	338	198
	Prevalence of Stunting	57.0	67.6	55.3	52.8	39.2

Classes defined in Section 6.2.3.1.

Source: Elderton, 1914 (82)

Data calculated by this author using NCHS standards.

Table 6.9

PREVALENCE OF LOW WEIGHT AGE IN GLASGOW 1906.

	SCHOOL CLASS				
	ALL	A	B	C	D
BOYS					
5-6 years:					
Examined	3,322	1,244	1,161	500	417
Low weight age	598	286	202	70	39
Prevalence of low weight age	17.9	22.9	17.3	14	9.3
8-9 years:					
Examined	4,009	1,402	1,449	630	528
Low weight age	1,080	478	391	142	69
Prevalence of low weight age	26.9	34	26.9	22.5	13
GIRLS					
5-6 years:					
Examined	3,104	1,143	1,141	431	389
Low weight age	416	199	151	41	25
Prevalence of low weight age	13.4	17.4	13.2	9.5	6.4
8-9 years:					
Examined	3,817	1,314	1,360	639	504
Low weight age	934	398	353	123	60
Prevalence of low weight age	24.4	30.4	25.9	19.2	11.9

Classes defined in Section 6.2.3.1.

Source: Elderton, 1914 (82)

Data calculated by this author using NCHS standards.

Table 6.10

GLASGOW 1906
RATIO OF THE PREVALENCE OF STUNTING
RELATIVE TO THE PREVALENCE OF LOW WEIGHT AGE

	All children	A	SCHOOL CLASS		D
			B	C	
BOYS					
5-6 years	3.31	3.05	3.24	4.01	4.32
8-9 years	2.37	2.16	2.41	2.60	3.28
GIRLS					
5-6 years	3.78	3.53	3.60	5.0	4.94
8-9 years	2.34	2.24	2.14	2.75	3.29

Classes defined in Section 6.2.3.1.

Source: Elderton, 1914 (82)

Data calculated by this author using NCHS standards.

Class "A"; however, for any class and age the ratios for boys and girls were virtually identical.

The apparent differences in this ratio conceal an underlying homogeneity which is illustrated in Figure 6.2. This shows a strong linear correlation existed between the prevalence of low weight-age and stunting ($r = 0.884$, $p < 0.01$). The intercept of 29.97 is the extrapolated prevalence of stunting in groups with no low weight-age. The slope of the line, 1.31, shows that the two prevalences are good predictors of each other, with only a 30% excess in the rate of increase in stunting relative to low weight-age. These figures suggest a background level of stunting of about 30% which was independent of low weight-age, the remainder being associated with declining nutritional status, as measured by the prevalence of low weight age.

The same pattern of growth failure was found in the London data which are presented in Table 6.11. The boys were significantly worse than the girls according to both indices and stunting was more prevalent than low weight-age (the ratio of the prevalence of stunting to low weight age was 2.26 for boys and 4.07 for girls). These ratios lie close to the regression line already established, as can be seen in Figure 6.1. Overall, the prevalences of both stunting and low weight-age were essentially the same in both Elderton and the LCC data sets. These results are compared below.

Numbers of Stunted and Low Weight-Age Children in LCC Sample Compared with Numbers Predicted from Elderton's Data

	Observed in 80 LCC children	Predicted from Prevalences in Elderton's data	p_2 (by χ^2)
LOW WEIGHT AGE			
Boys	15	11.6	>0.05
Girls	5	8.9	>0.05
STUNTED			
Boys	34	27.5	>0.05
Girls	21	21	>0.05

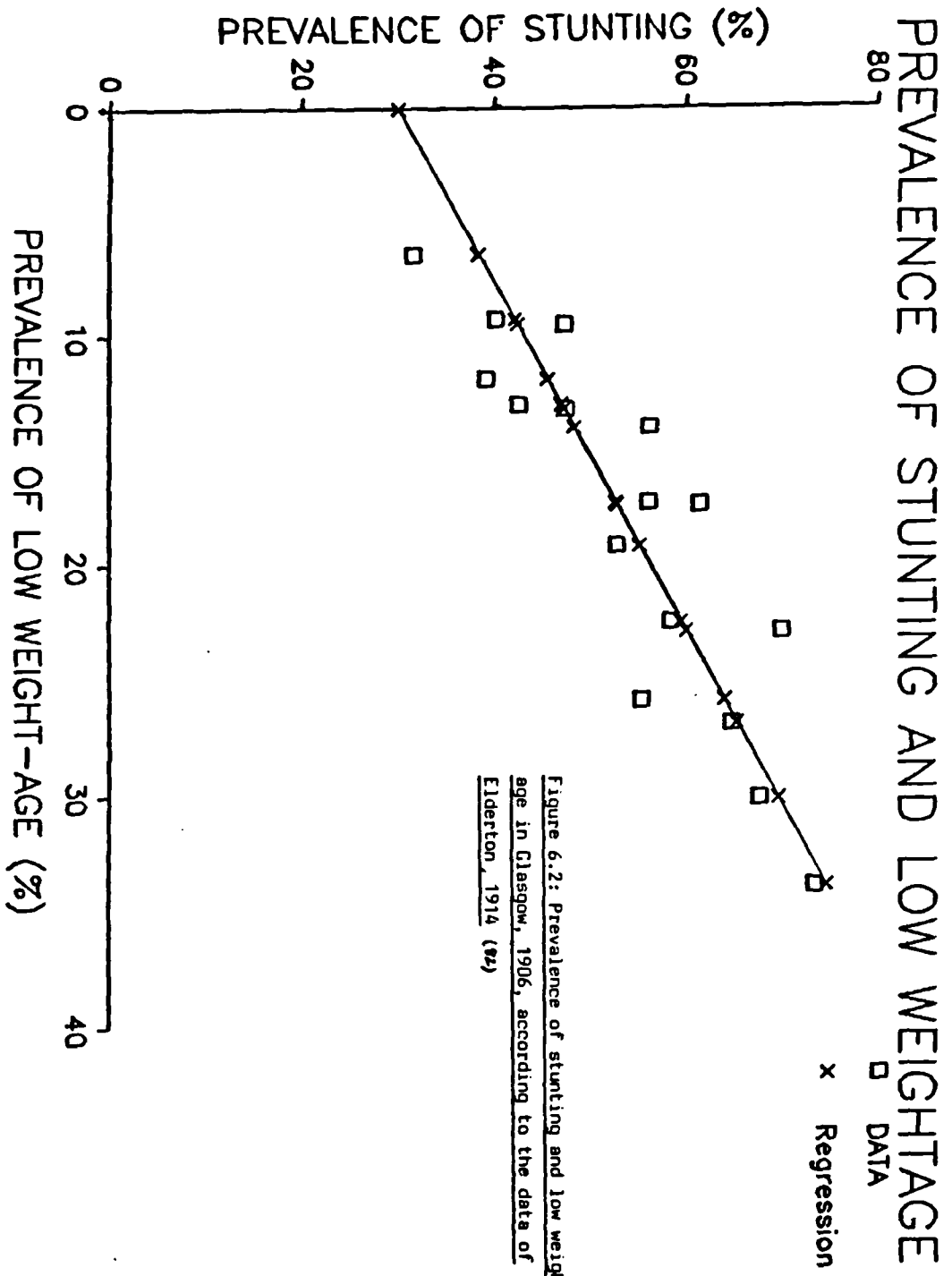


Figure 6.2: Prevalence of stunting and low weight age in Glasgow, 1906, according to the data of Elderton, 1914 (82)

Calculations by the present author

Table 6.11

ANTHROPOMETRIC ASSESSMENT OF NUTRITIONAL STATUS
AMONG ENTRANTS TO LCC POOR LAW HOMES 1908-1912

	BOYS	GIRLS	BOTH SEXES
Number examined	43	37	80
Prevalence of low weight age (%)	35	14	25
Prevalence of stunting (%)	79	57	69
Ratio Stunting: Low weight age	2.26	4.2	2.75

Source: Elderton, 1914 (82)
Data calculated by this author using NCHS standards.

For both Glasgow and LCC data sets, the prevalence of low weight-for-height (wasting) was calculated using NCHS standards. For the LCC data, only 2 children were below the third centile of weight for height although both of these were severely wasted with weight for height of less than 65% of the NCHS median. In the Glasgow data, 83 children were identified as wasted. This corresponds with an overall prevalence of wasting of 0.6%. For completeness, the distribution by class and age is shown in Table 6.12 although the numbers are too small to permit statistical analysis. Overall the levels of wasting are very small compared with the other two indicators of nutritional status used.

The conventional modern interpretation of weight for height data holds that low weight for height occurs as a result of weight loss due to severe *undernutrition* and that it therefore permits the discrimination of acute malnutrition from chronic malnutrition or normal nutrition. This interpretation rests on the assumption that there is a relationship between height gain and weight gain in all except acute restriction and that if weight or height gain is affected by nutrition, both are limited by the same factor in a linked manner. However, data presented in this thesis suggest stunting exists for reasons that are unrelated to energy balance, since weight gain was not impeded. This observation is in contradiction with modern observations and suggests that gain in weight and height may not be as tightly linked as is usually assumed. The implications of this extend far beyond nutritional history and must be explored elsewhere. It makes weight for height data difficult to interpret. The fact that the prevalence of weight for height below the 3rd centile shown in Table 6.12 is extremely low could be artefactual if a restriction in height gain distorted the normal relationship of weight and height ratio, thus giving an overestimate of "true" weight for height. In this case, the prevalence of "true" wasting could be much higher.

Table 6.12

PREVALENCE OF WASTING IN GLASGOW 1906

Results show prevalence as a percentage.
Number in brackets is the number of children who were wasted.

	ALL SCHOOLS	A	SCHOOL CLASS		D
			B	C	
BOYS					
5.5-6.5 years	0.3 (10)	0.2 (3)	0.3 (4)	0.2 (1)	0.5 (2)
8.5-9.5 years	1.15 (46)	1.36 (19)	0.48 (7)	2.86 (18)	0.38 (2)
GIRLS					
5.5-6.5 years	0.35 (11)	0.52 (6)	0.35 (4)	0 (0)	0.26 (1)
8.5-9.5 years	0.42 (16)	0.53 (7)	0.51 (7)	0.31 (2)	0 (0)

The numbers of children examined was given in Table 6.8.

Source: Elderton, 1914 (82)

Prevalence of wasting calculated by this author using NCHS standards.

Prevalences of high weight for height suggest that such a distortion does exist. The fraction of the population above the 90th NCHS centile of weight for height was calculated for Elderton's survey, where stunting was most prevalent. The cell size of this data were 1 inch (2.54 cms) and 2 lbs (0.91 kgs). By assuming all cell members had the greatest possible height for the cell and the lowest possible weight, a minimal estimate of the fraction of the population above the 90th centile of weight for height was obtained. The overall prevalence of high weight for height was nearly 10% and Table 6.13 shows that among boys it exceeded 10% of the population for all classes, with the exception of class D, the wealthiest class, and that the overall prevalence of high weight-for-height was nearly 10%. Since this prevalence could be increased 2-3 fold, according to age, by assuming all cell members were of mean size for the cell, it is clear that the population was heavy for height and hence that weight for height was probably exaggerated and wasting underestimated. The biology of these phenomena is interesting, but (perhaps fortunately) outside the bounds of this thesis. For the present it must simply be noted that no wasting was observed, but that this cannot be reliably interpreted as evidence of an absence of acute malnutrition, as is generally assumed.

6.2.4. Anthropometric Data from The Carnegie Survey, 1937

6.2.4.1. Description of Data

Original raw data which has survived from the Carnegie Survey of 1937 was collected at seven different sites. The results give unclothed weight to the nearest 28g, height to 0.1 cms and age to the nearest month. The number of results obtained at each sample site varied greatly and was in most cases too small for meaningful analysis by site. In this Chapter, the data are aggregated into two survey areas. Group A consists of children from Fulham, Barrow and the South Wales coal mining towns, Rhymney and Newport. Group B consists of children

Table 6.13

PREVALENCE AS % OF OVERWEIGHT, DEFINED AS HEIGHT FOR AGE OVER THE 90TH CENTILE OF NCHS STANDARDS IN GLASGOW, 1906

	All Schools	Schools in District type:			
		A	B	C	D
----- Prevalence % -----					
Boys					
5.5-6.5 years	12.4	14.1	11.2	14.8	9.4
8.5-8.9 years	10.3	12.0	11.9	11.3	5.9
Girls					
5.5-6.5 years	9.3	13.7	6.5	12.3	4.9
8.5-9.5 years	7.3	9.0	7.0	7.7	4.5

Number of subjects studied was shown in Table 6.8.

Source: Elderton, 1914 (82)

Data calculated by the present author

from Dundee and from two small rural communities, Tarves, an agricultural parish in Aberdeenshire, and Hopeman, a fishing village also in Aberdeenshire. Data for the Subsistence Production Society, a community of long term unemployed in South Wales, is also included in Tables 6.16 and 6.17 for completeness, but the numbers are too small for interpretation.

Family income data was not shown for Group A; however, the survey families were described in the Report as being 'economically mixed', that is containing families whose available food income ranged from 2s per head per week to 11s or more. Group B is an economically homogeneous group; all children in this group were from families with less than 5s per head per week available for food. This was 1s less than the minimum requirement as defined by the BMA in 1933 (84) and re-calculated by George in 1937 (85). However, it would be unwise to identify the two groups by economic criteria alone, firstly because group B is exclusively Scottish and secondly because this group includes the only rural communities. Height and weight data were collected by Boyd Orr on children aged 3 years to 16 years. However, in accordance with WHO recommendations (86) only data for the under 10s have been analysed for Nutritional Status by anthropometry. This avoids the complicating effect of the adolescent growth spurt on the interpretation of data.

Results

Before these data can be analysed, it is necessary to consider whether age and sex differences between subjects studied in areas A and B are statistically significant. Table 6.14 therefore shows the proportion of stunting and low weight for age by sex, in Group A, Group B and overall. In general there is a tendency for boys to be less frequently stunted or underweight than girls, but this difference is not statistically significant. Similarly, Table 6.15 shows that there was no significant

Table 6.14

ANTHROPOMETRIC EVIDENCE FOR UNDERNUTRITION IN THE CARNEGIE SURVEY, 1937

		Boys		Sex ratio as males per 100 females		Significance by X ²
		Boys numbers	Girls numbers	(a)	(b)	
Population surveyed	Area A	134	118	114	100	
	Area B	121	164	74	100	
	Total	255	272	94	100	
Stunted	Area A	28	38	74	65	<0.05
	Area B	30	35	86	75	NS
	Total	58	74	78	68	NS
Low weight age	Area A	19	20	95	83	NS
	Area B	5	10	50	44	NS
	Total	24	30	80	70	NS

(a) Sex ratios are those observed

(b) Sex ratios are corrected for a population surveyed with a sex ratio of 100

Area A is Barrow, Fulham and Newport & Rhymney

Area B is Dundee, Hopeman and Tarves

See Section 6.2.2. for cut-off criteria.

Source: Unpublished data held by Rowett Research Institute.
Data recalculated by this author.

Table 6.15

ANTHROPOMETRIC EVIDENCE FOR UNDERNUTRITION IN
THE CARNEGIE SURVEY 1937: AGE RATIOS

	Observed			Predicted (a)			Sign. by χ^2
	Age in years			Age in years			
	3-4.9	5-6.9	7-9.9	3-4.9	5-6.9	7-9.9	
Population Surveyed:							
Area A	66	83	103				
Area B	78	82	125				
Total	144	165	228				
Stunted							
Area A	14	22	30	17.2	21.7	26.9	NS
Area B	20	19	26	17.8	18.7	28.5	NS
Total	34	41	56	35.0	40.4	55.4	NS
Low Weight Age:							
Area A	7	13	19	10.2	12.8	15.9	NS
Area B	4	6	5	4.1	4.3	6.6	NS
Total	11	19	24	14.3	17.1	22.5	NS

Significance was tested for by χ^2 with 2 degrees of freedom

Area A and Area B are defined in Section 6.2.4.1.

Predicted results are those predicted if the distribution of malnutrition is independent of age.

Source: Unpublished data held by Rowett Research Institute.
 Data recalculated by this author.

age trend in the prevalence of malnutrition. Thus, in the subsequent analysis both age and sex have been ignored and the data shown in Tables 6.16 and 6.17 represent pooled results (Since wasting was so rare (see Tables 6.16 and 6.17) it was not meaningful to attempt to analyse its frequency by age or sex.)

The prevalence of wasting was not significantly different from the background of 3% which, according to NCHS standards, could be expected in a normal well nourished community. Conventionally (87), this would be interpreted as showing that there was no evidence of acute deterioration in nutritional status among the study population. However,

Table 6.16

ANTHROPOMETRIC EVIDENCE FOR UNDERNUTRITION IN THE CARNEGIE SURVEY, 1937.

Prevalence of stunting and low weight age

	Area A	Area B	Total A & B	Subsistence Society	Significance by X ²		
					AvsB	AvsSS	BvsSS
No. studied	252	285	537	37			
Percentage (number)							
Stunted	26.2 (66)	22.8 (65)	24.4 (131)	35.1 (13)	NS	NS	NS
Low Weight age	15.5 (39)	5.3 (15)	10.1 (54)	27.0 (10)	>0.01	NS	NS
Wasted	1.6 (4)	2.1 (6)	1.9 (10)	2.7 (1)	NS	NS	NS

Area A is Barrow, Fulham and Newport & Rhymney

Area B is Dundee, Hopeman and Tarves

See Section 6.2.2. for cut-off criteria.

Source: Unpublished data held by Rowett Research Institute.

Data recalculated by this author.

Table 6.17

ANTHROPOMETRIC EVIDENCE FOR OVERNUTRITION IN THE CARNEGIE SURVEY, 1937

Prevalence of Height for Age and Weight for Age above the 75th Centile:
all subjects

	Area A	Area B	Subsistence Society	Significance by χ^2		
				AvsB	AvsSS	BvsSS
No of subjects studied	252	285	37			
Percentage (number) above 75th centile of						
Height for Age	6.3 (16)	4.9 (14)	2.7 (1)	NS	NS	NS
Weight for Age	4.0 (10)	8.4 (24)	0.0 (0)	0.01	NS	NS
Weight for Height	17.5 (44)	18.9 (54)	8.1 (3)	NS	NS	NS

Area A is Barrow, Fulham and Newport & Rhymney
Area B is Dundee, Hopeman and Tarves

See Section 6.2.2. for cut-off criteria.

Source: Unpublished data held by Rowett Research Institute.
Data recalculated by this author.

for reasons described above, this assumption may not be valid. Both stunting and low weight-age were observed at levels much above the level of 3% that would be a background rate; moreover, the disparity between the prevalences for stunting and low weight-age, particularly in Group B, was marked, a phenomenon also noted in the context of Elderton's 1906 data.

An average of 10% of the children surveyed were underweight (ie of low weight-age) at a level at which would today lead to their being classed as malnourished. By contrast, nearly a quarter of the children were malnourished according to height for age criteria, ie stunted (Tables 6.16 and 6.17). Since the disparity between stunting and low weight-age parallels the observations reported for the surveys at the turn of the Century, this indicates the validity of the observation and demonstrates its continuing occurrence. The 1937 results once again suggest that stunting does not provide a good measure of underfeeding in the population studied - children could not be of normal weight but underfed if both were simply due to an inadequate energy intake.

6.2.5. A Comparison of 1906 and 1907

Both the pre-1914 results and the 1937 results indicate that linear growth was constrained by a factor which was not related to energy intake or expenditure. This may have been nutritional: for example, it has recently been suggested that Zinc deficiency (88) and protein deficiency (89) specifically impair linear growth. However, it is equally plausible that a non nutritional factor was responsible for this phenomenon, and no conclusions can be drawn at present.

It is possible that an alternative explanation for the disparity in the prevalence of stunting and low weight-age in the Carnegie Survey, is that an error was introduced in the "standard deductions" made for "trousers or knickers", the only items of clothing worn by the children

when they were weighed in this survey. No indication is made of the magnitude of this deduction, but it is most unlikely that this underestimated the weights of clothing by any significant amount. If trousers or knickers were to account for the lower prevalence of low weight-age, they would have to have weighed several kilograms.

To analyse the distribution of poor growth in the community further, the proportion of children above the 75th Centile of the NCHS standards was determined, and is shown in Tables 6.16 and 6.17. [The population above the 90th Centile was too low to be meaningfully studied.] Whilst fewer children were above the 75th Centile for either weight for age or height for age than would be expected from the NCHS standards and these shortfalls are significant ($p < 0.01$) the prevalence of weight for height above the 75th Centile did approach the predicted value of 25%. When these prevalences were calculated on a population which excluded those below the 3rd Centile, it was found that the fraction above the 75th rose by less than 1% and did not change these conclusions.

Although the two sets of data are not quite comparable, the low prevalence of high weight-for-height contrasts with the results found in Elderton's data and it is of interest that the smaller excess prevalence of stunting relative to low weight-age in 1937 is associated with fewer high weight for height children. This suggests that the factor disturbing the 'biologically normal' relationship between weight and height has become less important.

Evidence from both the beginning and end of the period of study indicates that for the purposes of the present discussion, low weight-age must be adopted as the preferred indicator of the extent of malnutrition since it gives the lower prevalences. Even on this basis we can conclude that underfeeding was a widespread problem among poor urban

children in the early years of the century; it can be diagnosed in almost one child in five in the 5.5 to 6.5 age group and one child in three in the 8.5 to 9.5 age group.

The cut-off adopted for underfeeding was the third centile of weight-for-age, which is the cut-off for the diagnosis of moderate Protein Energy malnutrition in modern nutrition. The values recorded by Elderton in 1906 are greater than those found in many developing countries at the present time, where the prevalence of underfeeding most closely resembles that recorded in Elderton's highest social group (90). By the outbreak of World War II, the prevalence of underfeeding remained high and low weight-age was found in 10% of elementary school children studied (see Table 6.18). This prevalence, too, matches that found in developing countries, where it would be seen as evidence of a need for nutritional intervention at the community level.

Table 6.18

COMPARISON OF THE PREVALENCE (%) OF STUNTING AND LOW WEIGHT AGE IN POPULATIONS SURVEYED IN 1906 AND 1937

	1906 (a) (number examined = 14,252)	1937 (b) (number examined = 537)
Stunting	57.65	24.4
Low weight-age	20.45	10.1

(a) Data calculated from Tables 6.8 and 6.9.

(b) Data derived from Table 6.16, Areas A and B.

Sources: Elderton, 1914 (82) and Carnegie Survey, 1937

6.2.6. Rickets and Growth

It was stated in the previous section that, according to the current understanding of growth in undernutrition, nutritionists would expect to find the prevalence of severe underweight and stunting to be broadly comparable in a given community and to find children who were stunted also underweight - but that this pattern of growth was not found in the data sets analysed in the present study.

Although no reliable rates can be established, it was widely believed that rickets was extremely prevalent in many industrial towns of the UK during the period 1900-1939 (see Section 6.1) and the possibility that rickets may have contributed to the high prevalence of stunting which has been described, deserves special consideration. This section investigates the hypothesis that the disproportion between the prevalence of stunting and low weight-age demonstrated in Section 6.2.1 was an artefact resulting from impaired longbone ossification. It also considers whether rickets was associated with an inadequate energy intake or with qualitative defects in the diet.

Owing to the unknown contribution of skin synthesis of cholecalciferol, dietary data do not provide a satisfactory guide to the estimation of Vitamin D status in the population and simple calculations of nutrient density for vitamin D can be misleading. The interaction of rickets and general undernutrition can, however, be deciphered by considering growth data rather than dietary survey data, and four investigations appear in the literature of the period which can be used to calculate differences in the height and weight of rachitic and non rachitic children of matched ages. These studies were carried out in Glasgow by Tully in 1921-22 (91), by G.F. Rose in Aberdeen over the period 1912-1920 (92) and by Mackay and Rose in London in 1930 (93).

Tully and Mackay and Rose tabulated data by age. G.F. Rose measured a single group of children on two occasions, at age 5 and age 13. (Although the age range reported by Tully was from 5-13 years, the number of rachitic subjects at any age or sex from 10-13 was less than five so data from children falling within this age range has been excluded from the analysis.) Results shown in Table 6.19 are based on Tully's and Mackay and Rose's surveys. They are unweighted means of the differences in height and weight of rachitic and non rachitic children at each age in the original data. These results show that on average, rachitic children were both shorter and lighter than non rachitic children. Since all data are presented as means, statistical analysis is not possible, but the effect is consistent both between studies and for different age and sex sub-groups within studies, which suggests that the difference is real. The smallest difference in the heights and weights of rachitic and non rachitic children was recorded by Mackay and Rose, where the controls were matched for social class. However, the hypothesis that the larger differences in height between rachitic and non rachitic children recorded in the other surveys resulted from the fact that rachitic children tended to come from the poorer families is not supported by Tully's data. When her rachitic children were compared with non rachitic controls in Class C (the poorest schools from which 79% of rachitic boys and 67% of rachitic girls came) the large differences in height and weight were only marginally reduced.

These studies can be used to address two problems. The first is the extent to which rachitic children were more poorly nourished than non rachitic children. All the data indicate that rachitic children were shorter and lighter than non rachitic children: thus, according to anthropometric criteria, they were less well nourished in energetic terms. This can be seen in Table 6.20 which compares weight for age, height for age and weight for height in all the data sets, with values

COMPARISON OF HEIGHTS AND WEIGHTS OF RACHITIC AND NON RACHITIC CHILDREN

Site	Survey date	Age range years	Prevalence of rickets in sample	BOYS		GIRLS		MIXED SEX		Source			
				Change in wt (kg)	Change in ht (cms)	n	Change in wt (kg)	Change in ht (cms)	n		Change in wt (kg)	Change in ht (cms)	
Aberdeen	1911-12	5	37.4%	1.9	4.1	1914	2.1	3.3	1828	2.0	3.7	3742	Rose 1924, 1926
Aberdeen	1919-20	13	37.0%	4.2	5.3	(a)	4.8	6.6	(a)	4.5	5.9	1000	Rose 1924, 1926
Glasgow	1921-22	5-9	4.6%	2.3	9.0	1018 (b)	2.0	9.8	1180 (b)	2.1	9.4	2198 (b)	Tully 1924
Glasgow (Tully's Class C only)	1921-22	5-9	7.9%	2.1	7.9	530	2.3	10.32	627	2.2	9.2		Tully 1924
London	1930-31	6-10	59.2%							0.8	3.2	85	Mackay & Rose 1931

- (a) Rose took 1000 cards at random from his original survey data. He does not state the sex distribution but if it was a truly random choice the values would be 511 boys and 489 girls. The mixed sex results are calculated on this assumption.
- (b) Tully's survey was on 1912 boys and 2083 girls aged 5-13 years, but because so few children aged 10-13 were rachitic we have excluded them from this analysis.
- (c) Prevalence figures must be treated with caution. Those for the data of Tully and of Rose are prevalences of clinical rickets in a population of elementary school children. Mackay & Rose, by contrast, took 45 rachitic patients and a control group, matched for age, sex and class, of 40.

Source: A.M.T. Tully (91)
 G.F. Rose (92)
 H. Mackay and S.F. Rose (93)

Data recalculated by this author

Table 6.20

COMPARISON OF WEIGHT FOR AGE, HEIGHT FOR AGE AND WEIGHT FOR HEIGHT
IN RACHITIC AND NON-RACHITIC CHILDREN

Source	(n)	Rachitic			(n)	Non Rachitic			
		WfA	HfA	WfH		WfA	HfA	WfH	
as percentage NCHS median									
Mackay and Rose:									
6 years	(10)	88	95	98	(10)	85	97	90	
7 years	(14)	92	108	108	(7)	101	96	110	
8 years	(6)	86	93	101	(12)	86	97	93	
9 years	(8)	81	91	105	(7)	90	93	108	
10 years	(7)	84	92	107	(4)	84	92	106	
Rose:									
5 years	Boys	91	93	104		101	97	108	
	Girls	93	94	102		106	97	110	
13 years	Boys	74	92	106		83	92	107	
	Girls	73	90	-		83	95	-	
Tully:									
5 years	Boys	(4)	87	87	111	(118)	92	92	108
	Girls	(9)	94	86	121	(147)	94	92	108
6 years	Boys	(10)	92	90	116	(172)	93	92	110
	Girls	(12)	95	90	114	(220)	93	91	111
7 years	Boys	(13)	89	87	116	(223)	90	91	109
	Girls	(10)	86	84	122	(239)	88	91	108
8 years	Boys	(18)	80	84	116	(219)	88	91	108
	Girls	(6)	79	83	118	(257)	100	91	109
9 years	Boys	(14)	77	84	115	(227)	86	90	111
	Girls	(6)	73	82	116	(274)	81	90	108

Sources present mean values for height and weight. The above table has been prepared comparing these means to NCHS standards.

Sources: H. MacKay and S.F. Rose (93)
G.F. Rose (92)
A.M.T. Tully (91)

expressed as a percentage of the median of the NCHS standard. In the data sets themselves (shown in Annex II) only means of heights and weights are given and they must therefore be interpreted with caution. However, it can be seen that mean weight for age was 7% lower among rachitic children than controls, mean height for age was 3% lower and mean weight for height 4% higher.

These lower values of both weight for age and height for Age are compatible with the hypothesis that rachitic children were relatively underfed compared with non rachitic children. The higher weight for height values in rachitic children could merely reflect the fact that stunting was artefactually high due to bowing of the legs. However, non rachitic groups also had mean weight for height values of over 100% of standard, which suggests that rickets was not the sole cause of this phenomenon. Data from these surveys indicate that rachitic children were of lower nutritional status than non rachitic children and this is highly suggestive of a lower food intake in rachitic groups, although it is recognised that this could be a consequence rather than a cause of rickets.

Comparisons in the growth of rachitic and non rachitic children can also be used to evaluate the impact of rickets on the high prevalence of stunting. Tully provides data on the stem height (defined as sitting height), and standing height of her rachitic and non rachitic children. Results shown in Table 6.21 indicate that the ratio of stem height to total height (S:T ratio) was higher among rachitic than among non rachitic children. This S:T ratio did not vary with sex in the age group studied. Data presented in the Carnegie Survey corroborates this observation (94). The smaller fraction of height accounted for by the legs suggests that bowing of the legs was responsible to some extent for the height differences between rachitic and non rachitic children.

Table 6.21

RATIO OF STEM HEIGHT (SH) TO TOTAL HEIGHT (TH) AMONG
RACHITIC AND NON-RACHITIC CHILDREN IN TULLY'S SURVEY, 1924

Age	SH cms	NON-RACHITIC					RACHITIC					SH: TH
		GIRLS TH cms	SH: TH	SH cms	BOYS TH cms	SH: TH	SH cms	GIRLS TH cms	SH: TH	SH cms	BOYS TH cms	
5	57.8	104.2	55.5	57.3	103.2	55.5	55.1	94.6	58.2	54.8	92.6	59.2
6	59.6	109.2	54.6	59.3	106.8	55.5	57.9	103.8	55.8	58.5	102.7	57.0
7	61.5	113.8	54.1	60.5	112.2	53.9	59.4	105.6	56.3	59.3	101.2	58.5
8	63.3	118.3	53.5	62.3	117.1	53.2	60.8	107.0	56.9	61.0	105.6	57.7
9	64.7	121.5	53.2	64.3	121.8	52.8	62.6	110.9	56.4	58.6	110.1	53.3

Stem height defined by Tully as sitting height. Rickets defined by Tully on clinical criteria.

Source: A.M.T. Tully (91)

Data recalculated by this author. Ratios calculated by this author.

However, it was found that this did not account for the total height shortfall between rachitic and non rachitic subjects.

Comparisons of stem height between rachitic and non rachitic subjects showed that the stem height of rachitic boys was 2.2 cms and rachitic girls was 2.3 cms below that of non rachitic boys and girls. Of the total height difference, which amounted to 9.0 cms in boys and 9.8 cms in girls, 6.8 cms in boys and 6.5 cms in girls cannot therefore be attributed to stem height differences and was presumably due to differences in limb length. An attempt to estimate the maximal extent to which leg bowing could account for the total height shortfall was made using S:T ratios. If the difference between the height of rachitic and non rachitic children were due to leg bowing, this would account for the higher S:T ratios of children with rickets. Thus, by using the S:T ratio of non rachitic children (0.542), the "true" height of rachitic children could be calculated from their stem height. This approach predicted differences between rachitic and non rachitic subjects of 4.1 cms (boys) and 4.2 cms (girls) which indicates that a maximum of the observed height differences [ie 4.9 cms for boys and 5.6 cms for girls] could be attributable to the effects of rickets on the long bones. The rachitic children were therefore genuinely shorter than the non rachitic children, for reasons that reflected both long bone growth and stem height.

6.2.6.1. Possible effects of rickets on the prevalence of stunting

It has been shown that approximately 60% of the height deficit between rachitic and non rachitic children, amounting to about 5cms, may have been artefactual in the sense that it could have been due to leg bowing. This is equivalent to approximately 15-20 percentile points for the age range 6-9 years, which seems a large discrepancy. However, the extent of stunting observed in Elderton's survey was such that an underestimate in

height of 5.1 cms (2 ins) in all subjects would only reduce the prevalence of stunting by about one half (Table 6.22) and the prevalence of stunting would still remain higher than that of low weight age. 100% is an unrealistic estimate of the prevalence of rickets in Glasgow in 1906, and in Table 6.23 the effect of assuming various prevalences of rickets in Class A (the poorest children) is illustrated. The heights of rachitic children have been reduced by 5.1 cms and it has been assumed that these prevalence figures apply to all heights. It can be seen that even at a prevalence of 50%, stunting is still twice as common as low weight age. Thus, it is extremely unlikely that rickets explains the disparity between stunting and low weight-age.

Table 6.22

ESTIMATED PREVALENCE (%) OF STUNTING IN GLASGOW 1906, ASSUMING
AN INCREASE IN HEIGHT OF 5CM AMONG ALL SUBJECTS

	SCHOOL CLASS			
	A	B	C	D
	Percent stunted			
Boys 5-6 years:				
Observed	61.4	47.5	47.3	31.6
Recalculated	29.0	25.0	18.3	5.4
Girls 5-6 years:				
Observed	69.9	56.1	56.2	40.2
Recalculated	37.0	23.2	25.6	10.2
Boys 8-9 years:				
Observed	67.6	55.3	52.8	39.2
Recalculated	35.6	27.1	20.7	13.7
Girls 8-9 years:				
Observed	73.3	64.7	58.4	42.6
Recalculated	43.2	33.8	28.1	14.8

Observed data and school class are derived from Elderton, 1914 (82).
Calculations performed by this author.

Table 6.23

PERCENTAGE OF CLASS A CHILDREN STUNTED ASSUMING VARIOUS
PREVALENCE RATES FOR RICKETS

	Prevalence of rickets %					% Low weight for age
	0	10	20	50	100	
	True prevalence of stunting					
Girls 5-6 years	61	58	55	45	29	17
Boys 5-6 years	70	67	63	54	37	23
Girls 8-9 years	68	65	62	52	36	30
Boys 8-9 years	73	70	67	58	43	34

Calculations were performed by this author on the assumption that rickets leads to an underestimate of height by 5cms, and therefore that as the prevalence of rickets rises more children are falsely classified as stunted.

Source: Elderton, 1914 (82)

Annex 1A

Heights (inches) of Rachitic and Non Rachitic Children
recorded by Tully, Rose, and Mackey and Rose

Study	Age	(n)	BOYS			GIRLS			
			Rachitic	(n)	Non-Rachitic	(n)	Rachitic	(n)	Non-Rachitic
Tully	5	(4)	94.6	(118)	104.2	(9)	92.6	(147)	103.2
	6	(10)	103.8	(172)	109.2	(12)	102.7	(220)	106.8
	7	(13)	105.6	(223)	113.8	(10)	101.2	(239)	112.2
	8	(18)	107.0	(219)	118.3	(6)	105.6	(257)	117.1
	9	(14)	110.9	(227)	121.5	(6)	110.1	(274)	121.8
Rose	5	*	101.3		105.4		100.3		103.6
	13	-	137.4		142.7		139.7		146.3
Mackay and Rose	6	(10)	113.0	(10)	115.6				
	7	(14)	114.9	(7)	119.4				
Rose	8	(6)	119.4	(12)	123.8				
	9	(8)	121.3	(7)	125.1				
	10	(7)	128.3	(4)	128.9				

Source: A.M.T. Tully (91)
G.F. Rose (92)
H. Mackay and S.F. Rose (93)

Annex 1B

Weights (in kgs) of Rachitic and Non Rachitic Children
recorded by Tully, Rose, and Mackay and Rose

Study	Age	(n)	BOYS			GIRLS			
			Rachitic	(n)	Non-Rachitic	(n)	Rachitic	(n)	Non-Rachitic
Tully	5	(4)	15.9	(118)	18.2	(9)	15.9	(147)	17.5
	6	(10)	18.8	(172)	20.1	(12)	18.3	(220)	19.2
	7	(13)	20.1	(223)	21.7	(10)	19.0	(239)	20.5
	8	(18)	20.5	(219)	23.4	(6)	20.0	(257)	22.5
	9	(14)	21.9	(227)	25.4	(6)	21.2	(274)	24.6
Rose	5		16.7		18.6		15.8		17.9
	13		33.1		37.3		32.9		37.7
Mackay and Rose	6	(10)	19.2	(10)	18.6				
	7	(14)	21.8	(7)	24.1				
	8	(6)	22.1	(12)	22.0				
	9	(8)	23.8	(7)	26.1				
	10	(7)	27.7	(4)	27.9				

Source: A.M.T. Tully (91)
 G.F. Rose (92)
 H. Mackay and S.F. Rose (93):

6.3. Dietary Surveys

Dietary surveys of varying quality were conducted on population groups during the period 1900-1939. These ranged from standard of living enquiries such as the Board of Trade Enquiry of 1904 (95) which asked participants to list main food purchases, to more detailed studies involving the weighing of all food purchases, the measurement of waste and record keeping of food eaten outside the home. Cathcart's MRC studies of the period 1926-32 (96) adopted this methodology. As part of the attempt to cast light on the nutritional status of the population, the most reliable of these surveys from the opening and the close of the period of study have been analysed. These are: (1) Rowntree's survey of 1902 and (2) the Carnegie survey of 1937.

In 1902, Seebohm Rowntree published his survey of the standard of living in York, *POVERTY: A STUDY IN TOWN LIFE* (97). This study included details of the budgets of families at various levels of income, amongst which the food purchases of 14 low income families were included. These records, which were kept for 1-3 weeks, provide sufficient data for a nutritional analysis of household food consumption. Rowntree himself undertook such an analysis, presenting his results as energy and protein intakes, corrected for assumed household requirements according to Atwater's 'Man-Values'. A nutritional evaluation of these diets was repeated more recently by Oddy (98), who calculated intakes of calcium, iron and phosphorous in addition to protein and energy. Rowntree's budgets have been re-analysed by the present author to provide information on the fat and vitamin content of Rowntree's diets as well as those nutrients studied by Oddy.

The most reliable and extensive study of the British diet to be carried out the close of the study period was the Carnegie Survey of 1937 (99), which has already been described. This study was undertaken partly in

response to criticisms levelled against Boyd Orr's study, FOOD HEALTH AND INCOME (100), which appeared in 1936. FOOD HEALTH AND INCOME was a compilation of various surveys, some of which were based simply on the recall of food purchases, and both government and Ministry of Health officials argued on these grounds that its unfavourable portrayal of the British diet was highly unreliable (101).

Nutrient intakes were computed from food consumption data using the "Compeat" programme, on an Opus II computer. The data base of food composition in "Compeat" is the Southgate and Paul version of the McCance and Widdowson food tables, supplemented by additional analytical data. The nutritional analysis of margarine and white flour was modified by this author to take account of fortification: margarine and white flour were assumed to be unfortified. The food equivalents used are shown in Appendix III.

6.3.1. Method of Analysis and Interpretation

The interpretation of data collected by Rowntree and the Carnegie Survey team is problematic: different requirement standards were adopted in the two investigations and, since Rowntree's study pre-dates the discovery of the Vitamins his work reflects a very different understanding of what constituted a nutrient from that of the Carnegie study. An additional difficulty arose over the differential use of per caput intakes by the Carnegie survey and intakes per Atwater Man-Value by Rowntree. In order to provide coherent comparative information, household food consumptions were recalculated as per caput consumptions (excluding infants) from the raw household budget and household composition data published in both surveys. The data gathered by Rowntree were household purchases over a week, while those collected in the Carnegie Survey were records of purchases, supplemented by household food inventories taken by trained investigators at the beginning and end of the week.

Neither survey provides sufficiently detailed information for an assessment of the adequacy of individual food intakes, given the complexities of interpretation this involves. However, it was considered a useful and legitimate exercise to compare household food consumptions per caput with a desirable level. The standards used for this purpose, which are shown in Table 6.24, represent either the value for a 9-11 year old child or a non-pregnant, non-lactating, active adult, whichever is the higher (102). The limitations of this method are fully recognised but it is intended only to provide a guide in evaluating nutrient intake; diets which achieved a desirable value would be broadly adequate for all members of the family with the exception of pregnant and lactating women and infants.

Intake relative to this standard was called "adequacy of household supply" and where the supply of a nutrient reached this level, it was assumed that the household was adequately supplied with that nutrient. It must be stressed that, although failure to meet such a level of supply does not necessarily mean that intake was inadequate, it does indicate that there was a risk an intake below RDA for some - or all - members of the household if food was evenly distributed. The limitations of this approach are freely admitted, but it provides the best available measure of the adequacy of diets given the nature of the data which is extant.

In comparing the intake of any nutrient-either with other historical data or with current Recommended Daily Allowances (RDAs) - it is important to determine how far differences reflect qualitative changes in the diet and how far they merely reflect differences in total food consumption. This can be achieved by calculating nutrient densities. A fall in nutrient density indicates that dietary changes have lowered the nutritional quality of the food consumed: if no change in nutrient

Table 6.24

DESIRABLE VALUES FOR DIETARY EVALUATION

Energy (kcal's)	2625
Protein (gms)	72
Iron (mg)	12
Calcium (mgs)	700
Thiamin (mgs)	1.0
Vitamin A (mcg RE)	750
Vitamin C (mg)	30

The "desirable value" is the RDA for a 9-11 year old child, or a non pregnant, non lactating active adult, whichever is the higher. The limitations of this definition are fully recognised but it is intended only to provide a guide in evaluating nutrient intake. Diets which achieve a "desirable value" may broadly be accepted as adequate for all members of the family with the exception of infants and pregnant and lactating women. For energy, a desirable value of 2625 kcal's was assumed: see text; 6.3.1.1.

density occurs, then differences in total nutrient intake must reflect differences in food energy intake. The relative importance of poor dietary quality and low energy intake in determining an inadequate nutrient intake can be quantified by comparing the observed nutrient density to the nutrient density needed to meet the RDA of that nutrient where energy intake meets the mean energy requirement (which is the RDA for energy). This calculation is crucial in the evaluation of the nutrition intervention policies advocated during the inter war period. In the present study, nutrient density is defined as the intake of the nutrient per unit of energy. Modern commentators including Yudkin (103) sometimes calculate nutrient density as the ratio of

$$\frac{\text{nutrient intake}}{1000\text{kcal}} \times \frac{\text{Energy requirement}}{\text{Nutrient requirement}}$$

This is not the way Nutrient Densities are used here for reasons which will become clear below.

Estimation of the Desirable Value for Energy

The desirable value for energy intake is important both in assessing the adequacy of energy intake and in calculating all nutrient densities. It is clearly unreasonable to adopt the lowest value of the energy RDA since all households consume a multiple of this. The value of 2625 kcals d-1 was adopted as an estimated mean desirable value for energy. The estimated requirement for energy has declined steadily during the post-war period (104) and it was felt that current RDAs for energy could not be used to evaluate the adequacy of either Rowntree's or the Carnegie diets, since these would seriously underestimate the expenditure of adults, and would very probably underestimate the expenditure of children. It is highly likely that both physical work and cold induced thermogenesis increased energy requirements over present levels in the populations studied. This view is corroborated by surveys of working class families from the late 19th Century, which place intakes in the

range 3,200-3,500 kcals d-1 (105) and by estimates of requirements, which are in the same range (106). These values are for normal working men, not those regarded as undertaking heavy activity. A value of 3250 is the median of these estimates. The current UK RDA for a moderately active man is only 85% of this (2900 kcals d-1). It is very probable that, without labour saving domestic appliances female requirements were increased by at least the same amount. On this basis a requirement of 2,400 kcals d-1, rather than the current value of 2150 kcal d-1 has been assumed for females (ie 75% of the male value).

Currently, children's requirements, expressed as a fraction of the adult male value, give average values for a 5 year old as 70% of the adult male requirement and for a 10 year old as 75%. By the age of 12 years the value for boys is 90% of the adult male and for girls, 100% of the adult female. Thus, if a family composition of two 5 year olds, one 10 year old and two parents is assumed, the per caput requirement is 78% of the adult male requirement. This is close to Rowntree's estimate of the 'man value' of the household which was 75% of the adult male value. 2625 kcal d-1 has been taken as a desirable value for energy intake; this is 80% of the assumed value for adult males of 3250 kcal d-1 and 90% of the modern RDA for adult males. Within the limits of accuracy of this method, this is thought to be a reasonable estimate of the likely energy needs per caput of families with children over the age of infancy.

6.3.2. Rowntree's Diets

Nutrient intakes estimated from Rowntree's household food consumption records are shown in Table 6.25. This indicates no deficits in the intake of thiamin and vitamin C, small deficits (less than 10%) for protein and vitamin A and larger deficits for energy (17%), calcium (24%) and iron (29%)

6.3.2.1. Protein

The deficit in protein intake was small, and probably less than the error of the method. According to the method adopted for assessing household supply, it was regarded as adequate. Table 6.25 shows that the value obtained in the re-calculation of results matched that reported by Rowntree almost exactly. The fraction of energy provided by protein (12.4%) is within the range found in the diets of affluent societies today (107) and overall, protein intakes must be regarded as adequate.

6.3.2.2. Calcium

The calcium intake of 533 mgs per day was below the desirable intake. The value for calcium was much higher than that obtained by Oddy (101) in his recalculation of Rowntree's results: Oddy's value was 310 mgs per day. However, the disparity of 223 mgs almost exactly matches the contribution of baking powder, calcium bicarbonate, to total calcium intake. This provides 39% calcium by weight and consumption provided 200 mgs of the total calcium intake; the disparity with Oddy's data may therefore reflect his not including baking powder in his calculations. Even Oddy's value is not exceptionally low and is only at the lower end of the range of calcium intakes at which communities have been observed to live and maintain normal calcium balance. RDAs for calcium (from which the desirable value was calculated) are set so far above any reasonable estimate of the physiological requirement that the possibility of calcium deficiency due to an inadequate supply is remote. Table 6.26 shows that the shortfall below the desirable value was mostly due to a low energy intake. The nutrient density for calcium was 92% of the desirable value and so, had energy intakes been adequate Calcium intakes would have reached 92% of the desirable value.

Table 6.25

ESTIMATED NUTRIENT INTAKES OF 'LABOURING CLASS' HOUSEHOLDS STUDIED BY ROWNTREE, 1902 (97) (Values per caput per day)

	Calculated value	% desirable value*	% value reported by Rowntree (97)	value calculated by Oddy (98)
Energy (kcal/s)	2173	83	93	2069
Protein (gms)	67.6	94	102	57
Protein (kcal/s %)	12.4	125	91	11.0
Fat (kcal/s %)	20.1		-	25.6
Calcium (mgs)	533	76	-	310
Iron (mgs)	8.5	71	-	
Vitamin A (mcg)	720	96	-	
Thiamin (mg)	1.0	100	-	
Vitamin C (mg)	31.3	104	-	

*For desirable levels see Table 6.24.

A desirable value for energy of 2625 was assumed : see text, 6.3.1.1.
Data recalculated by this author from food consumption data reported by Rowntree (97).

6.3.2.3. Vitamin A

The intake of vitamin A was 96% of the desirable value and, as with protein, little significance can therefore be attached to this small deficit. The nutrient density for vitamin A (Table 6.26) was well above the desirable value.

6.3.2.4. Thiamin

Despite the fact that bread was made with unfortified flour, thiamin intakes reached the desirable value. Two factors were responsible for this. Firstly, although thiamin values in bread were only 42% of current fortified values, the total consumption of bread was high. Secondly, most meat consumed was bacon, which contains 0.45 mg thiamin per 100g was high.

6.3.2.5. Iron

The iron intake was 8.5 mg per day, which represents 71% of the desirable value. Less than half for this shortfall was due to a low nutrient density: the nutrient density of iron was 85% of the desirable nutrient density value. Iron is a problematic nutrient. Although intakes may have been adequate for males, according to current views they would not have been adequate for pregnant and lactating females.

The role of poor iron absorption is now seen as an important factor in the aetiology of intractable iron deficiency anaemia in adult females, and as 35% of the iron intake in Rowntree's diets was from meat - almost twice the fraction of the modern British diet - this would be highly absorbable. Since bread flour was unfortified, cereal iron represented only 20% of total iron intake and as white flour predominated, the intake of phytates, which reduce iron absorption, would not have been exaggerated. However, the extent to which Rowntree's subjects were at risk from iron deficiency anaemia cannot be deduced with certainty from dietary data and there can be no doubt that the intake of iron, particularly for adult females, was undesirably low.

Table 6.26

ESTIMATED NUTRIENT DENSITIES (NDs) OF 'LABOURING CLASS' HOUSEHOLDS
STUDIED BY ROWNTREE, 1902 (97)

	Estimated ND	Desirable value*	Ratio
Protein (kcal %)	12.4	10.9	1.13
Calcium (mg/Mcal)	245	266	0.92
Iron (mg/Mcal)	3.9	4.5	0.85
Vitamin A (mcg/Mcal)	331	286	1.16
Thiamin (mcg/Mcal)	0.46	0.41	1.12
Vitamin C (mg/Mcal)	14.3	11.4	1.26

Nutrient densities calculated by this author from data in column 1 of Table 6.25. Desirable nutrient densities calculated from data in Table 6.24.

6.3.3. The Carnegie Survey, 1937

Data in Table 6.27 show mean nutrient intakes per caput for the poorest families (Group I) and most affluent families (Group VI) studied in the Carnegie survey, and a desirable value derived from current UK RDAs (111). This demonstrates a steep gradient in nutrient intakes between the economic groups and low intakes relative to current standards among Group I families, where intakes of iron, calcium and vitamins A and C and thiamin declined to levels of about 50% of the desirable value. Group I diets also indicate a lower level of thiamin, Vitamin A and calcium intakes than in Rowntree's survey. In the case of thiamin, the critical difference between the poor households surveyed in 1902 and in 1937 was the lower bacon consumption recorded in the 1937 survey. Indeed, if Group I's bacon consumption had matched that of Group VI, the social class difference would have been bridged. However, even at Group I's reduced level of consumption there was no serious risk of deficiency. The decline in calcium intakes between the 1902 and 1937 surveys can be accounted for by the exceptionally low consumption of milk among the lowest income groups studied in 1937. Finally, technological change, as well as low milk consumption, accounted for the dramatic reduction in vitamin A intake between the 1902 and 1937 surveys. Whereas the poor in 1902 ate butter, in 1937 this had been replaced by unfortified margarine.

In Table 6.28 intakes are expressed as nutrient densities which can be compared with a desirable or "target" nutrient density, derived from the adjusted RDA for energy. Table 6.28 demonstrates that for all nutrients, the lower in nutrient densities among Group I families relative to Group VI were less than the lower in nutrient intakes shown in Table 6.27. Indeed, for protein and iron nutrient density remained the same, which indicates that any shortfall in intake was due entirely to a lower total consumption in Group I families. Shortfalls of vitamin C and thiamin

Table 6.27

DAILY NUTRIENT INTAKES OF HOUSEHOLDS STUDIED IN THE CARNEGIE SURVEY, 1937 (values per caput per day)

	Group I (poor)	Group VI (affluent)	Desirable Value	Intake as % desirable value*	
				Gp I	Gp VI
Energy (kcal)	1570	3300	2625	60	126
Protein (gms)	44	93	72	61	129
Protein- (kcal %)	11.2	11.3		102	103
Fat- (kcal %)	24	37	-		
Calcium (mg)	297	1040	700	42	149
Iron (mg)	6.4	13.3	12	53	111
Vitamin A (mcg)	238	1088	750	32	145
Thiamin (mg)	0.53	1.60	1.0	53	160
Thiamin (%1000 kcal)	0.34	0.48	0.41	83	117
Vitamin C (mg)	14.8	64.5	30.0	49	215

*Desirable value given in Table 6.24.

Nutrient intakes were calculated by this author in data given in the Carnegie Survey (99).

Table 6.28

ESTIMATED NUTRIENT DENSITIES IN GROUP I AND GROUP VI HOUSEHOLDS STUDIED
IN THE CARNEGIE SURVEY, 1937

	Group I	Group VI	Desirable value
Protein (kcal %) (% D.V.)	11.2 (103%)	11.3 (104%)	10.9
Calcium (mg/Mcal) (% D.V.)	189 (71%)	315 (118%)	266
Iron (mg/Mcal) (% D.V.)	4.1 (91%)	4.0 (89%)	4.5
Vitamin A (mcg/Mcal) (% D.V.)	152 (53%)	329 (115%)	286.0
Thiamin (mg/Mcal) (% D.V.)	0.34 (83%)	0.48 (117%)	0.41
Vitamin C (mcg/Mcal) (% D.V.)	9.4 (82%)	19.5 (171%)	11.4

Figures in brackets are as percentage desirable value for nutrient densities shown in Table 6.24.

Data calculated from intakes shown in Table 6.27.

intakes relative to desirable values are reduced from 50%, when judged on an absolute basis, to approximately 20% when judged on the basis of nutrient density. Similarly, the shortfall of calcium is reduced from 50% to 30% and even for vitamin A, where the reduction is least, it falls from 70% to 50%. With the exception of vitamin A, the shortfall in dietary quality was therefore essentially secondary to the low energy intake. The low nutrient density for vitamin A demonstrates very clearly that income elasticities were greatest for those foods most needed to improve the quality of the diet; it is the higher consumption of meat and dairy products in Group VI households that explains the sharp contrasts between the inadequacy of Group I diets and the adequacy of Group VI diets. The poor were heavily dependent on cereal foods which gave a high return on energy purchased per penny and any shortfall in dietary quality was self correcting with a rise in income. These relationships are also demonstrated in the analysis of miners' diets which appears in Chapter 4.

When the nutrient densities recorded in Group I families are compared with the "target" or "desirable" nutrient density, based on the RDA for energy, it can be shown that densities for thiamin and protein were acceptable for all physiological groups. Given the variability in desirable nutrient densities between physiological groups, pregnant and lactating women fell below the desirable range for calcium, iron and possibly vitamin C and young children would also fall below the desirable range for calcium. Among other physiological groups, only the nutrient density of vitamin A was unequivocally deficient. However, this is NOT proof of qualitative inadequacy in the diet since, with the exception of energy, the desirable range used in the calculation of RDAs is exaggerated; whilst the RDA for energy is equal to the requirement, the RDA for other nutrients is considerably above mean requirements. At the most conservative estimate, this is increased to

cover individual variation, that is to a point where it meets the estimated requirement of all the population. In general a safety factor (which is usually unstated) is also built in to even the mean estimated requirement of the population. For example, Hume and Krebs have shown in the case of vitamin A that an intake of 440 mcg per day retinol equivalent (RE) was sufficient to cure deficiency in adult male volunteers (112) and concluded that the minimal protective intake was no greater than this, which is only 60% of the adult male RDA for vitamin A. If it is assumed that the estimated RDA exceeds the intake needed to prevent disease in all age groups by a comparable factor, a nutrient density as low as 210mcg RE (700 iu vitamin A) per Mcal would be adequate for all age groups, and only the value for Group I was below this.

In summary, then, it is extremely unlikely that the nutrient densities of the diets consumed by Group I families fell to a point where nutrient deficiencies would have developed if the energy intake of the diet had been adequate. Where intakes were marginal, the income elasticities for animal products were such that an increase in income was associated with a rise in their consumption without the need for educational intervention. However, throughout the inter-war period, the nutritional strategy recommended to government by its scientific advisors was a combination of nutrition education, intended to improve the quality of working class diets, and milk feeding for underfed school children.

The inaccuracies which are inevitably associated with dietary survey data are freely admitted. However, a comparison of Rowntree's labouring class diets with those of the Carnegie survey does suggest that there was a qualitative deterioration in the diet of the poorest sections of the community during the period of study, and that this was largely due to the replacement of butter by cheap, unfortified margarine (see Table 6.29). Even so, it is unlikely that inadequacies in dietary quality were

Table 6.29

A COMPARISON OF DIETS STUDIED BY ROWNTREE, 1902 AND BOYD ORR, 1937

	Nutrient Densities			Nutrient Densities as percent of desirable value		
	1902 (a)	1937 group I (b)	1937 group VI (c)	1902	1937	1937
Protein (kcal %)	12.4	11.2	11.3	125	103	104
Calcium (mg/Mcal)	245	189	315	76	71	118
Iron (mg/Mcal)	3.9	4.1	4.0	71	91	89
Vitamin A (mg/Mcal)	331	152	329	96	53	115
Thiamin (mg/Mcal)	0.46	0.34	0.48	100	83	117
Vitamin C (mg/Mcal)	14.3	9.4	19.8	104	82	171

(a) Data from Table 6.25

(b) Data from Table 6.27

such that deficiency disease would be expected among the population as a whole: the only possible exception is that of calcium intake, but since most calcium was consumed in a highly available form (not as chalk in fortified bread flour) even this is unlikely. Increased levels of income, which would have raised both energy intake and the consumption of culturally desirable foods, represented the only viable solution to the nutrition problem during this period. However, neither government nor its scientific advisors was prepared to consider the possibility that there was a problem of underfeeding, rather than working class ignorance, at the heart of the Empire.

The pattern of evidence presented in this Chapter supports the view that:

1. Clinical nutritional disease was rare throughout the period of study, despite a widespread belief during the inter-war years that there was an extensive problem of nutritional deficiency.
2. Anthropometry suggests that poor nutrition (low food intake) existed among elementary school children, although the pattern of growth in height was disturbed for reasons which may or may not have been nutritional.
3. Dietary evidence, though difficult to interpret, does not indicate qualitative deficiency, but does suggest that there was a fundamental problem of inadequate food intake among low income families.

The only evidence of an improvement in the nutritional status of the population was a decline in stunting, and the reasons for this decline remain obscure. Where changes in poor working class diets did take place, they appear to have been associated with a deterioration in nutritional status and were a consequence not of nutrition policy or health education, but of other, unrelated factors.

It seems, therefore, that the actual nutritional problems facing the UK during the period 1900-1939 did not correspond with those which were perceived by either the medical research establishment or the public health administration: the nutritional policies which were adopted to alleviate these problems were thus inappropriate and unlikely to have been of any benefit to the target population.

CHAPTER 7

7.1. Concluding Remarks

Throughout the present century, the public health establishment has been extremely receptive to dietary explanations for the inequalities in health which divide rich and poor in the UK. Nutrition scientists, for their part, have not been reticent in pressing the public health inferences of their work and have offered a series of dietary prescriptions intended to raise the nation to elevated standards of health, vitality and longevity. Edwina Currie's criticisms of dietary habits north of Watford made shortly after she was appointed Junior Health Minister in 1986 were in keeping with a tradition - especially popular among the ruling classes - which deplores the ignorance, incompetence and laziness of working class women. This view, favoured by both the Tories and many health educators in the 1980s, dates back to the Report of the Committee on Physical Deterioration of 1904 and was nurtured during the inter-war years by discoveries in the field of vitamin biochemistry which came to be known as the Newer Knowledge of Nutrition. Whilst the Committee on Physical Deterioration argued that the poor ate badly chosen diets, deficient in protein and fat, and that this was a cause of national physical degeneration, thirty years later, when Boyd Orr published *FOOD HEALTH AND INCOME*, it was generally believed that vitamin and mineral deficiencies were the major cause of ill health among the poor. The origins of this change in the approach to human nutrition can be traced to Hopkins's milk feeding experiments of the early years of the century.

The inferences for nutrition intervention which were wrongly drawn from Hopkins' work reduced the energy value of meals fed to malnourished children; this can only have impaired their nutritional status, since the nutritional problem facing the UK during this period was one of underfeeding, rather than deficiency disease. However, the medical

research establishment, and in particular the MRC Secretaries of the inter war years, displayed a driving ambition to see the results of laboratory experimentation translated into in public health practice. A more temperate view was put forward by Major Greenwood, Professor of Vital Statistics and Epidemiology at the London School of Hygiene and Tropical Medicine, who wrote to Fletcher in 1931:

"My emotion boils over quite as much as yours, but not because the powers that be don't read the latest news from the research frontIt seems to me utterly untrue that by some simple, unexpensive change in diet, any serious improvement can be effected. The matter is much bigger...it is a matter of economic organisation" (1).

.....

Work presented in this thesis is in many ways a hybrid of nutritional and historical analysis. For example, by re-working nutritional data it may be possible for historians to gain additional insights into the quality of daily life as it was experienced by earlier generations as the interpretation of data from Corry Mann's study suggests. Questions are also raised in this study concerning the social, political and economic factors which influence not only the application of science in public health policy but its very production and conduct. These questions are approached through the comparison of original and modern analyses of nutritional data. Historical nutrition may also have important implications for contemporary nutrition science. For example, the discovery of high prevalences of stunting relative to low weight-age in historical populations raises a number of challenging questions and queries many of the premises on which anthropometric monitoring of nutritional status is currently based.

Finally, the value of history for the contemporary application of nutrition science in public health work is, I hope, demonstrated in this thesis. It is crucial that both scientists and politicians should understand that neither the processes of nutrition committees nor the assumptions on which the present committee structures are based is pre-ordained. By elucidating the historical roots of these structures it is hoped that the process of creative reform may be advanced.

APPENDICES

APPENDIX I

HEIGHTS AND WEIGHTS OF MINERS' CHILDREN SURVEYED IN THE REPORT ON THE
NUTRITION OF MINERS AND THEIR FAMILIES (MRC 1924)

Age in Years	Sex	Height (cms)	Weight (kgs)
8	M	116.84	24.49
6	M	116.84	18.14
2	M	86.36	13.61
4	M	101.60	17.92
6	M	106.68	17.12
9	M	127.64	26.19
5	F	104.78	14.97
2	F	83.82	11.79
3	F	88.90	14.97
6	F	106.05	14.97
10	F	144.78	34.47
7	F	107.95	17.92
7	F	110.49	22.23
7	F	102.87	21.32

APPENDIX IIa

CHILDREN'S MINIMUM COUNCIL: Supporting Societies

"To ensure that no child shall by reason of the poverty of its parents be deprived of at least the minimum of food and other requirements necessary for full health" .

HEALTH AND CHILD WELFARE

National Society for the Prevention of Cruelty to Children; Save the Children Fund; Nursery School Association of Great Britain; National Baby Week Council; Women's Public Health Officers' Association; Family Endowment Society; Food Education Society; Maternal Mortality Committee; National Council for Maternity and Child Welfare.

EDUCATIONAL

Association of Assistant Masters; Association of Assistant Mistresses in Secondary Schools; Association of Headmasters; Association of Headmistresses; National Association of Schoolmasters; National Association of Headteachers; Workers' Educational Association; London Teachers' Association; National Council of Girls' Clubs

WOMEN'S

Women's Standing Joint Committee of Industrial Women's Organisations; Women's Cooperative Guild; National Council for Equal Citizenship; National Women's Citizen's Association; Women's Freedom League; Women's Liberal Unemployment Enquiry Group; Young Women's Christian Association; Fabian Society (Women's Group)

RELIGIOUS

Christian Social Council; Industrial Christian Fellowship; Catholic Social Guild; Society of Friends; Social Welfare Department of the Methodist Church

Appendix III

ROWNTREE and CARNEGIE SURVEYS:

Foods listed with index number	Foods described in text (if different)
11	Flour White
10	Flour Brown
17	Oatmeal Raw
19	Rice polished raw
23	Semolina Raw
30	Bread Wholemeal
33	Bread White
69	Semi Sweet Biscuits (Biscuits)
77	Fruit Cake Plain
84	Currant buns
124	Milk Cows Fresh Whole Summer (Fresh Milk)
131	Milk Cows Fresh Skimmed
132	Milk Cows Condensed Whole Sweetened
140	Butter Salted (Butter)
152	Cheddar Cheese type (Cheese)
157	Cottage Cheese (Curd Cheese)
165	Eggs Hens' Raw
184	Dripping Beef
185	Lard
193	Suet Block
213	Bacon Collar Joint Raw Lean and Fat (Bacon)
216	Gammon Joint Raw Lean and Fat (Bacon Shank)
242	Brisket Raw Lean and Fat (Meat Pieces:Meat)
259	Stewing Steak Raw Lean and Fat
261	Topside Raw Lean and Fat (Beef)
287	Lamb Scrag and Neck Raw Lean and Fat (Mutton)
303	Pork Chops Loin Raw
308	Pork Leg raw Lean and Fat
350	Rabbit Raw
358	Heart Lamb Raw (Offal)
373	Liver Chicken Raw
375	Liver Lamb Raw
394	Ham (Boiled Ham)
406	Polony
411	Sausages Pork Raw
417	Brawn
426	Beef Steak Pudding (Beef Pudding)
438	Cod Raw Fresh Fillets
451	Haddock Fresh Raw
485	Herring Fried (Fish)
489	Kipper Baked
491	Mackerel Raw
581	Cabbage Savoy Raw (Cabbage)
606	Lettuce Raw
613	Onions Raw
616	Onions Spring
620	Peas Fresh Raw
626	Peas Dried Raw (Dried Peas)
639	Potatoes Old Raw (Potatoes)
654	Radishes Raw
658	Spring Green Boiled
708	Currants Black Raw
727	Figs Dried Raw
750	Lemons Whole
773	Oranges Raw
809	Raisins Dried
843	Sugar White (Sugar)
844	Syrup Golden (Syrup)
849	Jam Fruit with Edible Seeds (Jam)
856	Boiled Sweets (Sweets)
868	Cocoa Powder
870	Coffee Ground Roasted
876	Tea Indian (Tea)
928	Piccalilli (Pickle)
956	Baking Powder
967	Vinegar
968	Yeast Bakers Compressed (Yeast)

NOTE: Bread, flour, cake and biscuit and margarine values were adjusted to take account of fortification.

REFERENCES

- [Abbreviations: MED. OFF. - The Medical Officer
PRO - Public Records Office
MRC - Medical Research Council
HO SC - The Health of the School Child
(Annual Reports of the Chief
Medical Officer to the Board
of Education)
OSPH - On the State of Public Health
(Annual Report of the Chief
Medical Officer to the Ministry
of Health)

INTRODUCTION

1. R.I. Rotberg and T.K. Rabb eds., HUNGER AND HISTORY (Cambridge U.P. 1985).
2. for example H.C. Trowell and D.P. Burkitt, eds., WESTERN DISEASES: their emergence and prevention (Arnold, 1981); S.Seely, D. Freed, G. Silverston and V. Rippere, DIET RELATED DISEASES: the Modern Epidemic (Croom Helm, 1985).
3. J.C. Drummond and Anne Wilbraham, THE ENGLISHMAN'S FOOD, (Jonathan Cape, revised edn., 1954).
4. D. Oddy and D. Miller, THE MAKING OF THE MODERN BRITISH DIET (Croom Helm, 1976); DIET AND HEALTH IN MODERN BRITAIN (Croom Helm, 1986).
5. John Burnett, PLENTY AND WANT (Nelson, 1966); Chris Wardle, CHANGING FOOD HABITS IN THE UK (London 1977).
6. T.C. Barker, D. Oddy and J. Yudkin, THE DIETARY SURVEYS OF EDWARD SMITH (Staples Press 1970); J. Yudkin and J.C. MacKenzie, CHANGING FOOD HABITS (MacGibbon and Kee, 1964); T.C. Barker, J.C. MacKenzie and J. Yudkin, OUR CHANGING FARE (MacGibbon and Kee, 1966).
7. C. Webster, 'Healthy or Hungry Thirties?', HISTORY WORKSHOP JNL, (1982) No.13, pp.110-129.
8. W.P. James, AMERICAN JNL. OF CLINICAL NUTRITION (1978), 31 p.1419.
9. See for example 'HOW BRITAIN WAS FED IN WAR TIME, Food Control 1939-45', (HMSO 1946); Report of the Chief Medical Officer of the Ministry of Education, 1939-45 (HMSO 1946); Lord Woolton, MEMOIRS OF SIR FREDERICK MARQUIS, EARL OF WOOLTON (Cassel 1959).
10. See for example E.V. McCollum, THE NEWER KNOWLEDGE OF NUTRITION (Macmillan, New York 2nd ed., 1922); H.C. Sherman, FOOD AND HEALTH (Macmillan, New York, 1934 and 1947).
11. Cmd 2175, 'Report of the Interdepartmental Committee on Physical Deterioration', 1904.
12. R. Floud and K.W. Wachter, 'Poverty and Physical Stature', SOCIAL SCIENCE HISTORY, VI, (1982), pp422-452; R.W. Fogel et. al. 'Secular Changes in American and British Stature' in R.I. Rotberg and T.K. Rabb HUNGER AND HISTORY (Cambridge U.P. 1985).

CHAPTER I

1. A.S. Wohl, ENDANGERED LIVES (University Paperback, 1984) p119.
2. Wohl, op.cit. p.118.
3. Wohl, op.cit.p.123.
4. Wohl, op.cit.p.123.
5. TABLE 1.1 . Source: G. Rosen, HISTORY OF PUBLIC HEALTH (M D Publications New York, 1958) p.314.
6. Sir Arthur Newsholme, THE MINISTRY OF HEALTH (G.P.Putnams, 1925) p.33.
7. H.W. Rumsey, ESSAYS ON STATE MEDICINE, Essay IV, II pp.281-2.
8. Cmd. 2175, 'Report of the Interdepartmental Committee on Physical Deterioration', 1904.
9. B.B. Gilbert THE EVOLUTION OF NATIONAL INSURANCE IN GREAT BRITAIN. THE ORIGINS OF THE WELFARE STATE (London Michael Joseph 1966)Ch II 59-101 ; see also G.R. Searle THE QUEST FOR NATIONAL EFFICIENCY (Oxford U.P., 1971) and Bernard Semmel, IMPERIALISM AND SOCIAL REFORM (Allen and Unwin, 1960).
10. C.F.G. Masterman, ed., THE HEART OF EMPIRE (London, 1901).
11. Karl Pearson, THE GRAMMAR OF SCIENCE (London, 1900) pp.26-7; letter to THE TIMES, 25 August 1905; DARWINISM, MEDICAL PROGRESS AND EUGENICS (London, 1912) p.29.
12. Semmel, IMPERIALISM AND SOCIAL REFORM, op.cit., 'Social Darwinism' pp.29-52
13. G. F. McCleary, THE MATERNITY AND CHILD WELFARE MOVEMENT (King and Son 1935) pp.4-5.
14. Cmd. 1501, 'Report of the Inspector General on Recruiting', 1903.
15. Major General Sir John Frederick Maurice, 'National Health, a Soldier's Study' CONTEMPORARY REVIEW, (1903) LXXXIII, pp.41-56.
16. Cmd. 2175 'Report of the Interdepartmental Committee on Physical Deterioration ' 1904, p.v.
17. 'Report of the Committee on Physical Deterioration', 1904.
18. Gilbert, op.cit.p.112 and pp.102-3.
19. Cmd. 2210 'Interdepartmental Report on Physical Deterioration. Minutes of Evidence ', 1904.
20. Newsholme, op.cit.p.31.
21. ibid, p.36.
22. see McCleary, op.cit.; Emelia Kanthack, THE PRESERVATION OF INFANT LIFE, A Guide for Health Visitors (H.K. Lewis 1907); Jane Lewis, THE POLITICS OF MOTHERHOOD (Croom Helm 1980).
23. Cttee Phys Det., op.cit. para 82.
24. ibid., para 216.
25. see C. Darwin ORIGIN OF SPECIES (London 6th Ed. 1873) Ch

111; Pearson op.cit.; Semmel op.cit.

26. see B.M.J. Dec 1903 and Jan 1904 for a series of articles on the question of physical deterioration among the British people.

27. A. Newsholme, 'Social Evolution and Public Health', LANCET, 1904, ii, 12 Nov pp.1331-5 and FIFTY YEARS IN PUBLIC HEALTH (London 1935).

28. Cmd. 2175 'Report of the Interdepartmental Committee on Physical Deterioration ', 1904, para 249.

29. ibid., para 216.

30. ibid., para 348.

31. ibid., para 310-314.

32. Robert Hutchison, FOOD AND THE PRINCIPALS OF DIETETICS, (Arnold, 1900).

33. D.S. Miller and P.R. Payne, 'A Theory of Protein Metabolism', JNL. THEORETICAL BIOL. (1963), 5, pp.398-408.

34. M. Pember Reeves, ROUND ABOUT A POUND A WEEK, (Virago, 1979) p.57.

35. Major General Sir John Frederick Maurice, op. cit.

36. Charles Booth, LIFE AND LABOUR OF THE PEOPLE OF LONDON I (London, 1892)

37. 'Report of the Committee on Physical Deterioration', op.cit., para 217.

38. B. Seebohm Rowntree, POVERTY, A STUDY OF TOWN LIFE, (London 1901).

39. 'Report of the Committee on Physical Deterioration', op.cit., para 313.

40. M.E. Bulkley, THE FEEDING OF SCHOOL CHILDREN (G. Bell, London, 1914) p.10 and THE SCHOOL BOARD CHRONICLE, 13 Dec. 1884, pp.628-9.

41. Cmd. 2779, 'Report of the Interdepartmental Committee on the Medical Inspection and Feeding of Children attending Public Elementary Schools' 1905.

42. Cmd. 2784, 'Interdepartmental Committee on the Medical Inspection and Feeding of Children attending Public Elementary Schools. Minutes of Evidence ' 1905.

43. 'Report of the Committee on the Medical Inspection and Feeding of School Children' 1905, op.cit., Appendix II p100.

44. ibid. para 237.

45. W.D. Atwater, Bull. No. 28, U.S. Dept of Agriculture, (Washington D.C., 1902).

46. ibid. para 237; para 306 (7).

47. ibid. para 97.

48. Board of Education Circular 576, November 1907.

49. 'Report of the Committee on Physical Deterioration' 1904 op. cit. para 303.

50. PRO Ed50/204, surveys of nutrition returns for 1935, 1936 and 1938; Committee Against Malnutrition, Vol. III, Bulletin No.16, Sept 1936.

51. World Health Organisation, MEASURING NUTRITIONAL STATUS, (Geneva, 1983).

52. E. Elderton, BIOMETRIKA, (1914), X, pp.268-339.

CHAPTER II

1. Henry C. Sherman, THE SCIENCE OF NUTRITION, (Columbia 1943) p.185.
2. 'Report on the present state of knowledge concerning Accessory Food Factors (Vitamines)', MRC Spec. Rep. Ser. No. 38, (HMSO 1919) p.3.
3. see for example Kenneth J Carpenter, THE HISTORY OF SCURVY AND VITAMIN C (Cambridge U.P. 1986); E.V. McCullom, A HISTORY OF NUTRITION (Houghton Mifflin, Boston 1957); George Rosen, A HISTORY OF PUBLIC HEALTH (MD Publications New York 1958).
4. Carpenter, op.cit., pp.43-74.
5. Rosen, op.cit., p.412.
6. G.Lunin, ZTSCHR. f. PHYSIOL. CHEM. (1881) 5, 31 and MRC op.cit.,p.6.
7. C.A. Pekelharing, NEDERLANDSCH TIJSCHR N GENEESK (1905) 2, 3 and McCullom, op.cit. pp.207-8.
8. F.G. Hopkins, ANALYST (1906) 31, 395; MRC op.cit.,p.8.
9. F.G. Hopkins, JNL of PHYSIOLOGY (1912) 44, 425.
10. C. Funk, JNL of STATE MEDICINE (1912) 20,341.
11. Rosen, op.cit., p.314.
12. F.G. Hopkins, LANCET (1921) 1 Jan ,1-7.
13. R.H. Chittenden, PHYSIOLOGICAL ECONOMY IN NUTRITION, (New York, 1904).
14. F.G. Hopkins, ANALYST (1906), 31, 395.
15. S.G. Hedin ZEITSCHR. F. PHYSIOL CHEM (1895) 22,177; W. HAUSMANN ibid (1900) 29,136; McCollum, op.cit.,p.53.
16. E.G. Wilcock and F.G. Hopkins, JNL PHYSIOL, (1906) 35, 88-102 and McCollum, op.cit., p.208.
17. McCollum, op.cit., p.208.
18. F.G. Hopkins, ANALYST (1906) 31,395.
19. F.G. Hopkins, JNL of PHYSIOL (1912) 44,425.
20. MRC op.cit., p.98.
21. For a full account of this work and further references see 'Report on the Present State of Knowledge concerning Accessory Food Factors' (MRC 1919), op.cit.
22. Hopkins (1912) op.cit.
23. J.C. Drummond, BIOCHEM JNL, (1918) 12, 25 and MRC op.cit.,pp.15-24.
24. MRC 1919, op.cit.,p.3.
25. J.P.W. Rivers and T.L.Frankel, BRITISH MEDICAL BULLETIN (1981), 37, (1), 59-64
26. D.S. Miller and S.R. Parsonage, PROCEEDING OF THE NUTRITION SOCIETY (1971), 30, 76A.
27. P. Coyer, D.J. Millward and J.P.W. Rivers, BRITISH BULLETIN OF NUTRITION, (1987) June
28. H. Chick 'Biological value of proteins contained in wheat

flours', Lancet (1942), 1, 405.

29. for example MRC: E.M. Widdowson and R.A McCance, 'Studies on the nutritive value of bread and on the Effect of variations in the extraction rate of flour on the growth of undernourished children', SRS 287 (1954)

30. E.M. Widdowson 'Extraction rates-Nutritional Implications' in A. Spicer, BREAD (Applied Science, 1975) pp.235-246.

31. Sir Robert McCarrison, NUTRITION AND NATIONAL HEALTH, pp.9-29, Royal Society of Arts, Cantor Lectures 1936 (Faber 1944).

32. J. Boyd Orr, W. Thompson and R.G.Garry 'A long term experiment with rats on a human dietary', JNL of HYGIENE (1935) XXXV, 476-497.

33. H.C. Sherman, CHEMISTRY OF FOOD AND NUTRITION, (4th Ed, Columbia, 1933) pp.532-535.

34. E. Mellanby, 'Experimental Rickets', MRC Spec. Rep. Ser. No. 61 (HMSO 1921)

35. M.R. Gribbon and M.I.H. Fergusson, 'Nutrition in Vienna', LANCET (1921) 5 March, 474-477; H. Chick 'Studies of Rickets in Vienna, 1919-1922, MRC Spec. Rep. Ser. No. 77 (HMSO 1923).

36. See for example Report of the School Medical Officer for the County of Durham, 1920; J.W MacIntosh, JNL OF STATE MEDICINE, XLIII (1935) 4, 187-198.

37. E.V. McCollum, THE NEWER KNOWLEDGE OF NUTRITION The Use of Food for the Preservation of Vitality and Health, (2nd ed, Macmillan, New York 1922) p.427.

38. MRC 1500, Fletcher to MacFadden, 20 Sept 1921.

39. McCollum, op.cit. (1st ed. Macmillan, New York, 1918).

40. McCollum, (2nd ed, 1922) p358.

41. ibid., p.369.

42. ibid.

43. ibid.

44. Report of the Medical Research Council 1926-7 (HMSO 1928) p.23.

45. McCollum, op.cit.(3rd ed, 1925) p.530.

46. McCollum, op.cit.(2nd ed, 1922) p.56.

47. PRO MH55/691 Report of an inquiry into the health of school children in South Wales Colliery Districts, 11 Jan 1928.

48. see for example: MED OFF (1929) Vol.42, 31 Aug, p.90, on Ministry of Health report, 'Investigation in the Coalfield of South Wales'.

49. McCullom, op.cit. (3rd ed, 1925) p.538.

50. E.V. McCullom and N. Simmonds THE AMERICAN HOME DIET (Detroit, 1920).

51. McCullom, op.cit.(2nd ed, 1922) p.343.

52. ibid., p.429.

53. ibid., p.354.

54. *ibid.*, p.396.
55. see MRC 1500 Fletcher to MacNalty *op cit*; MRC 2100/1 Fletcher to Lord Dawson 15 April 1931; MRC 1741 Fletcher to M'Gonigle 26 Oct 1931.
56. McCollum, *op.cit.* (2nd ed, 1922) p.384.
57. *ibid.*
58. *ibid.*, p.354.
59. *ibid.*, p.355.
60. McCollum, *op.cit.*(3rd ed, 1925) p.530.
61. Robert R. Kohler, FROM MEDICAL CHEMISTRY TO BIOCHEMISTRY, (Cambridge, 1982) p.70.
62. *ibid.*
63. E.P. Cathcart, EDINBURGH MED.JNL.,(1928) 35, 21-21.
64. L. Findlay and M. Fergusson, 'A Study of Social and Economic Factors in the Causation of Rickets', MRC Spec. Rep. Ser. No. 20 (HMSO 1918).
65. MRC 99/5, correspondence between Hopkins and Fletcher, July 1918.

CHAPTER III: Part I

1. see Annual Report of the Chief Medical Officer to the Ministry of Health, 1919-1920 (HMSO 1920), Appendix IX, p.389.
2. C. Fraser Brockington, SHORT HISTORY OF PUBLIC HEALTH, (Churchill, London 1956) pp.44-45.
3. MRC Annual Report, 1932-33 (HMSO 1933) pp.10-11.
4. MRC 2100/1, Fletcher to Lord Dawson, 15 April 1931; see J.W.P. Rivers, 'Social Constraints on Nutritional Knowledge', pp.389-395, in C. Horwitz ed., ADVANCES IN DIET AND NUTRITION, (John Libbey, 1985) for current MRC spending on nutrition research.
5. MRC PF 106, Fletcher to Hopkins 23 June 1915; Fletcher to Hopkins 31 Dec. 1917; see also Robert E. Kohler FROM MEDICAL CHEMISTRY TO BIOCHEMISTRY (Cambridge U.P.1982).
6. BRIT. JNL. NUTRITION (1956) 10, 77, Obituary of Sir Edward Mellanby.
7. for example E. Mellanby, EXPERIMENTAL RICKETS MRC Spec.Rep.Ser. 61, (HMSO 1921); H.N. Green and E. Mellanby, 'Vitamin A as an Anti Infective Agency' B.M.J. (1928) 11, 20 Oct, pp.691-696.
8. MRC Annual Report, 1920-21 (HMSO 1921) p.12.
9. MRC Annual Report, 1923-24 (HMSO 1924) pp.17-18.
10. MRC Annual Report, 1925-26 (HMSO 1926) and M. Mellanby and C.L. Patterson, BRIT. DENT. JNL. (1926) 7, 1045; MRC Annual Report 1926-27 (HMSO 1927) for public health significance of the artificial manufacture of Vitamin D.
11. MED OFF, (1926) Vol 36, 25 Sept, p.141.
12. MRC Annual Report, 1920-21 (HMSO 1921), p.12.
13. MRC 2100/1, Fletcher to Lord Dawson, 15 April 1931.
14. MRC 1500 'Memorandum upon the Proposed Relations between the Ministry Health and the Medical Research Council in relation to Scientific Work upon Food'. Fletcher, 25 November 1919. See MRC 1381/1 for further insights into Fletcher's views on research methods in medical science.
15. MRC 1500, Newman to Fletcher, 3 Dec. 1920; MRC 1381/1 Fletcher to Newman, 7 Dec 1920.
16. MRC 1500 MacFadden to Newman, 17 Dec, 1920. Memorandum suggesting subjects food research including the nutritive value of milk; H. Corry Mann, 'Diets for Boys During the School Years' MRC Spec. Rep. Ser. No. 105 (HMSO 1926); 'The Incidence of Dental Disease in Children MRC Spec Rep Ser No. 97 (HMSO 1925); M Mellanby 'Diet and the Teeth' MRC Spec. Repp. Ser No. 140 (HMSO 1929).
17. See MRC 2070/25 (1922) for Ministry's refusal to fund an MRC Infant Welfare investigation; MRC 1190, Fletcher to Newman, 16 Dec, 1924 for MRC refusal to support a similar request from the Ministry.
18. MRC 1190, Fletcher to Newman, 17 Jan, 1924.
19. Report in: MED OFF (1928), 15 Dec. p.253.
20. Sir Walter M Fletcher, NUTRITION ABSTRACTS AND REVIEWS (1932) 1, 3, 357.
21. DSPH 1930 (HMSO 1931) p.163.

22. MRC 1190, Fletcher to Newman, 22 Oct, 1931.
23. MRC PF 133 II, Fletcher-Greenwood correspondence Oct to Nov 1931.
24. MRC 1500, Fletcher to MacFadden, 20 Sept 1921; MRC 2100 Fletcher to Newman, 20 June 1924 ; MRC 2100/1 Fletcher to Dawson op cit; MRC 1930, Fletcher to Robinson, 5 Dec 1930.
25. MRC 1500, Fletcher to MacFadden, op.cit.
26. 'Report on the Nutrition of Miners and their Families', MRC Spec. Rept. Ser. No. 87 (HMSO 1924).
27. MRC 2100, Fletcher to Newman, 20 June 1924.
28. MRC 1500, Fletcher to MacFadden, op.cit.
29. MRC 2100/1 Fletcher to Dawson, op.cit.
30. MRC 1741, Fletcher to M'Gonigle 26 Oct 1931.
31. G.C.H M'Gonigle, PROC R. SOC. MED , (1933) 26, 677.
32. Fletcher to M'Gonigle, op.cit.
33. See for example MRC 2030; MRC 1290, examples of Lister Institute funding applications; MRC 2033, records of Drummond and Zilva's work on Vitamin D, Vitamin A, the Vitamin B complex and Vitamin E.
34. MRC 1200/1, Minutes of Nutrition Committee, 19 Jan 1927; MRC 2037/I-VII , papers relating to the Dunn Nutritional Laboratory, 1927-1940.
35. MRC 1444/I-II, papers relating to McCance and Widdowson's studies of the nutritional composition of foods.
36. MRC 1400, papers relating to Cathcart's dietary surveys.
37. MRC 1190, Fletcher to Robinson, 5 Dec 1930. See also PRO MH56/40.
38. PRO MH56/43, minute of meeting between Newman and Mellanby, 6 Dec., 1927.
39. E. Mellanby, 'Duties of the State in Relation to the Nation's Food Supply', B.M.J.(1927) ii, 8 Oct, p.66 .
40. H.N. Green and E Mellanby, B.M.J.(1928) ii, 20 Oct, pp.691-696.
41. MRC 1200/1, Fletcher to Dawson op cit; MRC 1190, Fletcher to Robinson, op.cit.
42. see MRC 2127, Cabinet Nutrition Policy, 1934.
43. MRC 2127, Minute of meeting between Mellanby and Hankey, 23 Feb, 1934.
44. MRC 2110/14b, Meeting of EAC Scientific Research Committee, 11 May , 1934.
45. MRC 2110/14b, op.cit., 11 May 1934.
46. MRC 2127, Hankey to the Lord President, Stanley Baldwin, March 1934.
47. MRC 2110/14b, 'Report on the Need for Improved Nutrition of the People of Great Britain', EAC (SR) 74, 30 June 1934.
48. MRC 2127, Hankey to Mellanby, 12 March 1934.

49. 'Report on the Need for the Improved Nutrition of the People of Great Britain, op.cit.
50. MRC 1854, correspondence between Mellanby and F R Cowell, Secretary of the PEP Medical research group, Oct 1934-Jan 1936; see also PEP papers held at British Library of Political and Economic Science (BLPEs), PEP WG 2/1 .
51. MRC 2110/12, Scientific Research Committee, announcement of 14 Feb 1935.
52. MRC 1190, Mellanby to McNalty, 18 Sept, 1935.
53. PRO MH56/47 Greenwood to Robinson, 1934.
54. see Annual Reprot of the Chief Medical Officer to the Minister of Health, 1919-1920, (HMSO 1920), Appendix IX, p.389.
55. C. Fraser Brockington, 'SHORT HISTORY OF PUBLIC HEALTH', op.cit., pp.44-45.
56. B.B Gilbert 'THE ORIGINS OF THE WELFARE STATE' op.cit., pp.107-111.
57. see Sir George Newman, 'Outline of Preventive Medicine' (HMSO 1919).
58. Parlane Kinloch, B.M.J. (1931) 11, Oct 1931, p.43.
59. Local health education initiatives were regularly reported in the public health journals of the period : see for example PUBLIC HEALTH (1925) Aug, p.333-334; MED.OFF. (1925) Vol.34, 14 Nov, p.217; MED. OFF. (1926) Vol.35, 20 Feb pp.75-6; MED. OFF. (1927) Vol.37, 12 March p.123; MED.OFF. (1933) Vol 49, 18 March p.109. In 1927 the Health Education Council was established in response to the proliferation of activities such as these.
60. PUBLIC HEALTH, (1923), Aug 1923, p.307.
61. MRC 1520/1: Files relating to League of Nations discussions concerning nutrition ; speech by Mr S.M. Bruce, Australian delegate, before the Second Committee, League of Nations Assembly, 19 Sept 1935.
62. PUBLIC HEALTH, (1921) 7, XXXIV, p.117.
63. HOSC 1920, (HMSO 1921), p.146.
64. HOSC 1923, (HMSO 1924), pp.97-984; OSPH 1925, (HMSO 1926), pp. 159-160; OSPH 1926 (HMSO 1927) p.184.
65. OSPH 1923, (HMSO 1924), p.132.
66. OSPH 1929, (HMSO 1930), pp.146-7.
67. OSPH 1930, (HMSO 1931), pp.162-3.
68. J.M. Hamill, DIET IN RELATION TO NORMAL NUTRITION, Ministry of Health Reports on Public Health and Medical Subjects, No.9, (HMSO 1921), introduction.
69. Annual Report to Pres. Bd. Edn. 1912 p.26 (HMSO 1913).
70. HOSC 1928, (HMSO 1929), p.67.
71. HOSC 1935, (HMSO 1936), p.13.
72. E.P. Cathcart, B.M.J. (1937) 1 , 27 Feb pp.435-438.
73. Annual Report to Pres. Bd. Edn. 1910, (HMSO 1911), p.26.
74. Annual Report to Pres. Bd. Edn. 1914, (HMSO 1915), p.220.

75. HOSC 1926, (HMSO 1916), p.16.
76. HOSC 1925, (HMSO 1926), p.31 . See also MRC Spec. Rep. Ser. No. 101 'Poverty, Nutrition and Growth' (HMSO 1926).
77. Board of Education Circular No. 582, 1908.
78. Annual Report to Pres. Bd. Edn. 1910, (HMSO 1911), p.26.
79. HOSC 1935, (HMSO 1936), p.12.
80. HOSC 1922, (HMSO 1923), p.124.
81. HOSC 1926, (HMSO 1926), p.14.
82. HOSC 1933, (HMSO 1934), p.31.
83. HOSC 1930, (HMSO 1931), p.46.
84. PRO ED50/83, reports by J.E. Underwood on the physical condition of children in the S. Wales coalfield, 1927-30; PRO ED50/77, reports on conditions in Jarrow and County Durham , 1927-8.
85. HOSC 1927, (HMSO 1928), p.107.
86. PRO MH56/48 LCC Education Committee, Nutrition of School Children, 13 Nov 1933.
87. PRO MH56/53, Robinson to Hilton Young, 18 Dec. 1933.
88. see for example LANCET, (1933) 1, 25 March, p.661 ; MED.OFF. (1933) Vol 49, 29 April p.162 ; MED.OFF.(1933) Vol 50, 18 Nov, pp.211-2 and p.208.
89. HOSC 1933, (HMSO 1934), p.16.
90. HOSC 1934, (HMSO 1935), pp.26-7; HOSC 1935, (HMSO 1936), p.12.
91. MRC 1190, Mellanby to MacNalty, 18 Sept 1935.
92. J. Boyd Orr, FOOD HEALTH AND INCOME, (MacMillan, London 1936) p.18.
93. see for example PRO MH55/627, surveys of feeding provision in Cumberland and the North East, 1936-37.
94. 'Report of Independent Interdepartmental Committee', 30 Jan 1936, in papers of E.M.H. Lloyd 4/56, British Library of Political and Economic Science.
95. Education (Provision of Meals) Act, 1906; Education (Provision of Meals) Act, 1921; HOSC 1926, (HMSO 1927), p.137; HOSC 1927, (HMSO 1928) , p.102.
96. PRO ED50/80, June 1935.
97. F.G. Hopkins, Address to the Annual Council Meeting, Children's Minimum Campaign, 6 July, 1938. E. Rathbone papers, University of Liverpool archives.

Chapter III : Part II

1. PRO ED50/77 (Report on health and physique of children in Durham coalfield, 1928); PRO ED50/83 (reports on health and physique of children in South Wales coalfield, 1928-30).
2. PRO MH56/52, Carnwarth , May 1932; MH56/40, Robinson, July 1933 : comments on the need to find cheap ways to raise the quality of the diets of the unemployed.
3. DSPH, 1930 pp.162-164 (HMSO 1931).
4. PRO MH56/43, Robinson to A. Greenwood, 22 Oct 1930.
5. MRC 2106/1 , Robinson to Fletcher, 9 Dec 1930.
6. MRC 1190, Fletcher to Robinson, 5 Dec 1930.
7. PRO MH56/43, Newman to Robinson, 22 Oct 1930.
8. PRO MH56/44, Robinson to Advisory Committee, 28 Jan 1931.
9. PRO MH56/43 Newman memorandum, Oct 1930.
10. PRO MH56/44, Advisory Committee Minutes, 8 Jan 1931.
11. PRO MH56/44, Advisory Committee Minutes, 16 April 1931.
12. CRITICISM AND IMPROVEMENT OF DIETS (HMSO 1932); DIETS FOR CHILDREN IN POOR LAW HOMES (HMSO 1932).
13. PRO MH56/40 Carnwarth to Newman, April 1933.
14. PRO MH56/51, quoted in Carnwarth to Newman, 1 Dec 1931.
15. *ibid.*
16. 'Hungry England' Inquiry, WEEK END REVIEW , (1933) 1 April, pp.357-359.
17. PRO MH56/40, Carnwarth to Newman, 6 April 1933.
18. PRO MH56/52, Robinson to Hilton Young (Minister of Health), 27 June 1933.
19. PRO MH56/40, Robinson to Food Division, 5 July 1933.
20. PRO MH56/40, Advisory Committee Minutes, 28 July 1933.
21. *ibid.*
22. *ibid.*
23. *ibid.*
24. *ibid.*
25. 'REPORT OF COMMITTEE ON NUTRITION. To determine the weekly expenditure which must be incurred by families of varying size if health and working capacity are to be maintained, and to construct specimen diets' (BMA, 1933).
26. see PRO MH56/43, 'It was obviously impossible wholly to overtake the harm done by the BMA report but something might be done to mitigate its effects', Hilton Young, 11 Dec 1933.
27. PRO MH56/55, Labour Party 'Notes for Speakers' 1 Dec 1933.
28. see for example Robert Skidelsky, 'POLITICIANS AND THE SLUMP' (Pelican 1970).
29. PRO MH56/55, Robinson to Hilton Young, 8 Dec 1933.

30. PRO MH56/55, 24 Nov 1933.
31. PRO MH56/47, 30 Nov 1933 question in House of Commons asked by Labour Member for Leigh: "Would the Minister consider the necessity of making provision for more adequate allowances for unemployed families and their dependents?"
32. PRO MH56/43, Greenwood, 30 Nov 1933 .
33. PRO MH56/55, Dec 1933, Newman memorandum.
34. *ibid.*
35. PRO MH56/47 Advisory Committee Minutes, 13 Jan 1934.
36. *ibid.*
37. *ibid.* .
38. *ibid.*
39. *ibid.*
40. PRO MH56/40 Greenwood to Robinson, 1934.
41. MRC 2106/1, Greenwood to Hudson, 21 Nov 1933.
42. PRO MH56/53, Robinson to Greenwood, 11 Oct 1933.
43. PRO MH56/47, Advisory Committee Minutes, Aug 1933.
44. PRO ED40/80, Deputation from the Society of Medical Officers of Health protesting over the provisions of Board of Education Circular 1437, June 1935.
45. R.H. Simpson, PROC R. SOC. MED, (1935), XXVIII p.725.
46. PRO MH56/46, Advisory Committee Minutes, June 1934.
47. J.W.P. Rivers, 'The Biology of Famine' in G. Harrison ed FAMINE, (O.U.P.) in press, 1987.
48. PRO MH56/46, Advisory Committee Minutes, 7 June 1934.
49. *ibid.*
50. *ibid.*
51. PRO MH56/43 Ministry morandum, Oct 1930.
52. PRO MH56/45, Mellanby, 12 Feb 1932.
53. PRO MH56/46, Carnwarth to Becket, 18 April 1932.
54. PRO MH56/40, Advisory Committee minutes, 28 July 1933.
55. PRO MH56/40, Cathcart , 24 July 1933.
56. MRC PF133, Fletcher/Greenwood correspondence, 28 Oct-2 Nov 1931; see also MRC 1190 Fletcher to Newman, 22 Oct 1931 and Newman to Fletcher, 30 Nov 1931.
57. PRO MH56/45, 5 Feb 1932.
58. See MRC 99 for papers relating to the rickets controversy.
59. PRO MH56/45, Cathcart, written communication, 8 Feb 1932.
60. J.H. Hess et al JNL A.M.A. (1930), 2 Aug, p316; L.J. Harris, B.M.J.(1933) 11, 12 Aug, pp.309-310.
61. PRO MH 56/45, Mellanby, 12 Feb 1932.

62. PRO MH56/51, Carnwarth to Newman, 1 Dec 1931; PRO MH56/46; PRO MH56/52 for further official comments on differences within the Committee.

63. E.P. Cathcart, London School Of Hygiene and Tropical Medicine Lectures, MED.OFF. (1931) Vol.45, 21 March, pp.131-136.

64. *ibid.*

65. MH56/52, Greenwood memorandum, 12 Dec 1932.

66. MED.OFF. (1931) Vol.45, 28 Feb, p.95.

67. MRC 2100/1/IV, MRC Nutrition Committee, 11 Dec 1933.

CHAPTER IV : Part I

1. Bd. of Edn. Annual Report, 1911 (HMSO 1912) p.22.
2. Cmd. 2175, 'Report of the Committee on Physical Deterioration' (1904), para 293-294, p.57.
3. *ibid.*
4. Bd. of Edn. Annual Report, 1911 (HMSO 1912) p.25.
5. Bd. of Edn. Annual Report, 1913 (HMSO 1914) p.47.
6. 'Poverty, Nutrition and Growth', MRC Spec. Rept. Ser., No. 101 (HMSO 1926).
7. 'Report on the Nutrition of Miners and their Families', MRC Spec. Rept. Ser. No.87 (HMSO 1924).
8. Bd. of Edn. Annual Report, 1910 (HMSO 1911) p.26.
9. see MRC 2070/25 I, for papers relating to the Scottish Child Life Committee.
10. 'Report on the Nutrition of Miners and their families', *op.cit.*, p.29 and p.47.
11. D. Noel Paton, J.C. Dunlop and E.M. Inglis, A STUDY OF THE DIET OF THE LABOURING CLASSES OF EDINBURGH (Edinburgh, 1901).
12. *ibid.*, pp.78-9.
13. MRC 2070/25, Paton memorandum, 30 May 1922.
14. 'Poverty, Nutrition and Growth' *op.cit.*, p.6.
15. MRC 2070/25 I 'Report on the Correlation shown in Data relating to Growth and Nutrition of Children in Glasgow an Dundee', Child Life Committee paper, 1922 and 'A Report upon the data relating to the Growth of Children in Glasgow an Dundee' Child Life Committee paper, 1922.
16. 'Poverty, Nutrition and Growth', *op cit*, Parts IV, VI and VI.
17. *ibid*, pp.228-233.
18. *ibid*, pp.304-305.
19. *ibid*, Table 178, p.230.
20. HOSC 1925, (HMSO 1926) p.31 .
21. MED.OFF.(1932) Vol 47, 4 June, p.221, editorial comment.
22. see for example H. Pelling, A HISTORY OF BRITISH TRADE UNIONISM, (Pelican, 1973) pp.160-167.
23. 'Report on the condition of Miners and their Families', *op.cit.*, p.5. and HANSARD, May 18 1922, Col 569).
24. *ibid.*, p.6.
25. *ibid.*, Section VIII, pp.33-46.
26. *ibid.*, Section VII, pp.25-33.
27. *ibid.*, p.25.
28. *ibid.*, p.29.
29. MRC 2100, Fletcher to Newman 20 June 1924.
30. 'Report in the Nutrition of Miners and their Families',

op.cit., p.47.

31. MRC 2100, Greenwood to Fletcher, 30 Oct 1921.

32. MRC 2100, Cathcart to Fletcher, 26 Nov 1926.

33. 'A Study in Nutrition', MRC Spec. Rep. Ser. No.151. (HMSO 1931); 'Studies in Nutrition' MRC Spec. Rep. Ser. No.165. (HMSO 1932); 'A Dietary Survey in terms of the Actual Foodstuffs Consumed', MRC Spec. Rep. Ser. No.218 (HMSO 1936).

34. MRC 2100/1, Cathcart memorandum in preliminary papers submitted to the Committee on the Quantitative Problems of Human Nutrition, 1921.

35. 'A Study in Nutrition', Spec. Rept. Ser. 151, op.cit., p.38.

36. *ibid.*, pp.43-51.

37. 'A Study in Nutrition', op.cit.

38. *ibid.*, p.49.

39. *ibid.*, p.51.

40. for example, MED.OFF. (1932) Vol.47, 4 June, p.221.

41. 'A Dietary Survey in terms of the Actual Foodstuffs Consumed', op.cit.

42. MRC 1400, Cathcart to Lansborough Thompson, 3 Feb 1937.

43. 'A Dietary Survey in terms of the Actual Foodstuffs consumed', op.cit., p.54.

44. MED. OFF. (1931) Vol. 45, 21 March pp.143-146, 'The Foundations of the National Diet, I'. Lecture delivered at the London School of Hygiene and Tropical Medicine, Feb 1931.

45. *ibid.*

46. *ibid.*

47. The Royal Sanitary Insititute, 42nd Congress, Glasgow, 1931. Reported in MED.OFF. (1931) Vol.46, 29 Aug, pp.90-91.

48. *ibid.*

49. *ibid.*

50. HOSC 1926 (HMSO 1926) p.16.

51. MED.OFF. (1924) Vol.32, 26 July , p.47.

52. For further references concerning nutrition in public health propaganda see PUBLIC HEALTH, Aug 1925, pp.333-334; MED.OFF. (1925), Vol. 34, 14 Nov, p.217; MED.OFF. (1926) Vol.35, 20 Feb pp.75-76; MED.OFF.(1927) Vol.37, 12 March p.123; MED. OFF.(1933) Vol.49, 18 March p.109.

53. See MED.OFF. (1920) Vol.23, 21 Feb p.80; MED.OFF. (1924) Vol. 32, 26 July, p.39; MED.OFF. (1926) Vol. 36, 30 Oct, p.203.

54. Bd.of Edn. Annual Report, 1914, (HMSO 1915), p. 218.

55. MED.OFF. (1920) Vol.23, 21 Feb, p.80.

56. MED.OFF. (1925) Vol.34, 5 Sept, p.112.

57. MED.OFF.(1926) Vol.35, 22 May , p.214.
58. MED. OFF.(1926) Vol.35, 15 May p.200.
59. 'Report on the Nutrition of Miners and their Families',
op.cit., pp.25-29.
60. MED.OFF. (1926) Vol.36, 7 August, p.65.
61. *ibid.*
62. HDSC, 1929, (HMSO 1930) p.111.

Chapter IV Part II

1. OSPH 1932 p.16, p.223 (HMSO 1933); HOSC 1932 p.127, p.134 (HMSO 1933) OSPH 1933 p.220 (HMSO 1934); HOSC 1933 p.17, p.31 (HMSO 1934).
2. see for example 'Evidence of Malnutrition', report by Children's Minimum Committee, 1934.
3. see Papers of Eleanor Rathbone, University of Liverpool Archives: Children's Minimum Campaign, Reports and Correspondence.
4. Bulletins of the Committee Against Malnutrition (CAM), 1933-1939, Library of the Ministry of Agriculture, Fisheries and Food.
5. Campaign Against Malnutrition (CAM) Bulletin, No.1, Vol 1, Jan 1934.
6. See for example CAM Bulletin Vol III, No. 20 May 1937; CAM Bulletin Vol.III, No. 16 Sept 1936.
7. Robert Skidelsky, POLITICIANS AND THE SLUMP, (Pelican, 1970) p.323.
8. B.W.E. Alford, DEPRESSION AND RECOVERY? (Macmillan 1972) p.75.
9. PRO MH55/688 Children's Minimum Committee, 'Evidence of Malnutrition', Sept 1934, see PRO MH55/688 and MRC 1853.
10. "Report of Committee on Nutrition", op.cit.(BMA 1933).
11. C.L. Walker and M. Church, 'Poverty by Administration', JNL OF HUMAN NUTRITION, (1978), 32, pp5-18.
12. J.P.W. Rivers, 'The Profession of Nutrition: an historical perspective', PROCEEDINGS OF THE NUTRITION SOCIETY (1979), 38, pp.225-231.
13. Children's Minimum Committee, 'Evidence of Malnutrition', op.cit.
14. *ibid.*
15. 'Children, Young People and Unemployment part I', Office of the Union Internationale de Secours aux Enfants, Geneva, 1933.
16. MED OFF (1933) Vol 50, 8 July, p.12.
17. J. Boyd Orr, FOOD, HEALTH AND INCOME, (Macmillan, 1936) .
18. *ibid.* p.38.
19. Report on House of Commons debate on Malnutrition of 8 July 1936, LANCET (1936) ii, 156-159; PRO MH 56/215, Minister of Health's briefing papers and personal notes for debate on Malnutrition, 8 July 1936.
20. Ipswich Committee Against Malnutrition, 'An Enquiry into Malnutrition', 1938; Sheffield Social Survey Committee, 'A Study into the Standard of Living in Sheffield, 1933; M.S. Soutar, 'Nutrition and Size of Family', Birmingham Social Survey Committee, 1939.
21. see PRO MH56/40, Carnwarth to Newman , April 1933.
22. E. Rathbone, 'Memorandum on the Scale of Needs Suitable for Adoption by the Unemployment Assistance Board in Assessing Assistance to Applicants under Section II of the Unemployment Act, 1934' (Children's Minimum Campaign Committee, 1934).

23. see papers of Violet Markham, British Library of Political and Economic Science (BLPES), submissions from CMC to Unemployment Assistance Board concerning the adequacy of proposed scales of benefits, 1934. (Markham, 7/9) .
24. Markham papers, (BLPES), submissions from Family Endowment Society to the Unemployment Insurance Statutory Committee , Jan 1938 (Markham, 9/1).
25. V.H. Mottram 'The Physiological basis of the Minimum Wage', LANCET (1927) ii, 22 Oct, pp.898-899.
26. M.L. Clark, 'Household Dieteries and Family Budgets', MOTHER AND CHILD, April 1932.
27. G.P. Crowden 'The minimum cost of physiologically adequate diets for working class families' LANCET (1932) i, 3 April, pp.899-901.
28. Annual Report of the School Medical Officer, Wakefield 1932, Nutrition report pp.10-13.
29. Somerville Hastings LANCET (1932)ii, 26 Nov, p.1185.
30. DSPH and HOSC op.cit., 1932 and 1933.
31. MED.OFF. (1933) Vol.50, 18 Nov, p.211.
32. ibid. p.208.
33. see HOSC 1920 (HMSO 1921) p.148; HOSC 1923 (HMSO 1924) p.98.
34. CAM Bulletin Vol.II, No.9, July 1935.
35. MED.OFF.(1933) Vol.50, 18 Nov, p.212.
36. MED.OFF. (1934) Vol.52, 14 July, p.15.
37. CAM Bulletin, op.cit.
38. L.J. Harris 'A programme for Nutrition Surveys'. LANCET (1936) i, 25 April 1936, pp.966-968.
39. ibid.
40. E.H. Wilkins, MED. OFF. (1937) Vol.58, 2 Oct, pp.145-8.
41. CAM Bulletin, Vol. II No. 9, July 1935.
42. CAM Bulletin, Vol. II No. 16, Sept 1936.
43. W.F.W. Betenson, Annual Report, SMO for Breckonshire, 1932 and 1933.
44. R Hws Jones, 'Physical indices and the clinical assessment of malnutrition', JNL. ROY. STAT. SOC. Vol CI Part I 1938.
45. DSPH 1936 (HMSO 1937) pp.138-9; DSPH 1937 (HMSO 1938) p.130; DSPH 1938 (HMSO 1939) pp.94-7.
46. for example R.A. McCance, E.M. Widdowson et al 'Study of English Diets by the Individual Method' JNL.OF HYGIENE (1938) XXXVIII, pp.596-623.
47. R.H.Simpson, PROC.ROY. SOC. MED., (1935), Vol XXVIII pp.724-5; HOSC 1934, (HMSO 1935) p.28; PRO MH56/53 Robinson to Hilton Young, 11 Oct 1933.
48. PRO MH56/46, Advisory Committee Minutes, June 1934.
49. CAM Bulletin, Vol., No.5, Nov 1934; V Freeman, MED.OFF. (1934) Vol. 52 18 and 25 Aug; T. Cole, Annual

Report SMO for Beckenham, 1933 and 1934; W.R. Dunstan, MED.OFF. (1934) Vol.51, 3 March .

50. MED.OFF. (1934) Vol.52, 22 Sept, p.119.

51. see PRO ED50/204 for evaluations of the accuracy of Local Authority returns, 1935-39.

52. J.Boyd Orr 'Food Health and Income. Report on a survey of adequacy of diet in relation to income. ' (MacMillan 1936) p.7.

53. MED.OFF. (1924) Vol.32, 12 July, p.12.

54. W.B. Stott 'A Nutrition Survey', MED.OFF.(1937) Vol. 57, 26 June, pp.259-261

55. 'Report of Committee on Nutrition', (BMA 1933) p.42.

56. A.M. Critchely, JNL. OF STATE MEDICINE, (1933) 41, pp.667-671

57. J.C. Spence, ' Investigation into the Health and Nutrition of certain of the Children of Newcastle Upon Tyne between the ages of one and five years', Annual Report of the Medical Officer of Health for the City and County of Newcastle Upon Tyne, 1933.

58. Annual Report of the School Medical Officer of the City of Cardiff, 1936.

59. L. Roberts et al 'An investigation into the Nutrition of Children of School Age', MED.OFF.(1938) Vol. 60, 12 Nov p.209 and 19 Nov p.219.

60. F Le Gros Clark ed NATIONAL FITNESS (Macmillan, 1938) see especially foreword by Maj. Gen. Sir Robert McCarrison.

61. Arthur Greenwood THE HEALTH AND PHYSIQUE OF SCHOOL CHILDREN (P.S. King, 1913); see also F.E. Larkins 'The Influence of Wages on the Child's Nutrition', MED OFF, (1910) 17 Dec, p.347.

62. Greenwood, op.cit., p.59.

63. ibid., p.68.

64. PRO MH56/55, Labour Party, 'Notes for Speakers', Dec 1933; PRO MH56/214, papers relating to Conference of Labour Women held in October 1936.

65. see PRO MH 55/639, resolution passed by the National Council of the Federation of Women's Institutes, 20 May 1936; PRO MH55/649 delegation from People's League of Health concerning proposed feeding experiment, July 1936.

66. see PRO MH56/215, Kingsley Wood, July 1936.

67. J.C. Spence, PROC. ROY.SOC.MED, VOL XXVIII, 1935, pp.725-728 and op.cit., 1933.

68. J.A. Charles, 'A study of the Diets of 69 Working Class Families in Newcastle Upon Tyne', City Health Dept Newcastle Upon Tyne 1934.

69. Annual report of the School Medical Officer of the City of Cardiff, 1936.

70. Roberts et.al., op.cit.

71. Critchely, op.cit.

72. HOSC 1937, (HMSO 1938), p.23.

CHAPTER V

1. see for example MH55/639, National Federation of Women's Institutes, January 1937; PRO MH55/650, National Federation of Women's Institutes, October 1938; PRO MH 56/214 Labour Women's Group; PRO MH 56/688, Children's Minimum Committee, July 1936; PRO MH55/649, People's League of Health; PRO MH 55/642 Lady Juliet Rhys Williams on behalf of National Birthday Trust, May-June 1936.
2. PRO MH55/645, Note on ministerial conference concerning the refusal of the Central Milk Distributive Committee to accept a reduction in distributive margins under a proposed cheap milk scheme for mothers and infants, 11 Nov 1938.
3. Report of Independent Inter-Departmental Committee on the cost of extending the provision of cheap milk, 30 Jan 1936. E.M.H. Lloyd papers, 4/56, B.L.P.S. archives.
4. See PRO MH56/215, briefing papers for House of Commons debate on Malnutrition, 8 July 1936; PRO MH56/214, comments on National Conference of Labour Women's recommendations for an extension in the provision of free school meals, Oct 1936.
5. H.C. Corry Mann 'Diets for Boys during the School Years', MRC Spec. Rept. Ser. No.105 (HMSO 1926); J Boyd Orr, 'Milk Consumption and the growth of school children, LANCET, (1928) i, p.202.
6. See Newman to Lord Astor, 3 Feb 1931, Papers of Waldorf Astor, 1066/1031, University of Reading archives; DAILY HERALD, 'Drink More Milk Campaign', 20-25 April 1931.
7. W.H. Jones and W.J. Cowie, WELSH JNL of AGRIC (1934) X 83-107; E.L.L. Harry, WELSH JNL of AGRIC (1935), XI 23-48.
8. See National Clean Milk Society, Papers of Waldorf Astor, 1066/1025-1034, University of Reading archives.
9. Newman to Astor, Feb 1931, Papers of Waldorf Astor, 1066/1031 University of Reading archives.
10. The TIMES, 24 March 1931; for further comments on Moynihan's statements on milk borne disease, see Papers of Waldorf Astor, 1066/1032, University of Reading archives.
11. see Astor op.cit., 1066/1031.
12. Cmd. 2175, 'Report of the Inerdepartmental Committee on Physical Deterioration' (1904).
13. Cmd. 2779, 'Report of the Interdepartmental Committee on the Medical Inspection and Feeding of Children attending Public Elementary Schools' (1905).
14. Education (Provision of Meals) Acts, 1906 and 1921.
15. Report of the Medical Officer (Education) to the sub committee on Underfed Children, London County Council, 1909.
16. Report of the Schhol Medical Officer, Bradford, 1907 and M. Bulkley, THE FEEDING OF SCHOOL CHILDREN (G. Bell & Son, 1914).
17. Article 23, Board of Education Special Services Regulations concerning meals provided under Part VII of the Education Act, 1921.
18. HOSC 1920, (HMSO 1921), p.146.
19. HOSC 1923, (HMSO 1924), p.98.

20. HOSC 1923, (HMSO 1924), p.119.
21. see HOSC 1920, p.146 (HMSO 1921); HOSC 1925 p.183 (HMSO 1926).
22. H. Corry Mann 'Diets for Boys during the School Years' Spe. Rep. Ser. No. 105 (MRC 1926).
23. HOSC 1925, (HMSO 1926), p.123.
24. HOSC 1925, (HMSO 1926), p.118.
25. see MRC 2034, use of Corry Mann's results in National Milk Publicity Council advertising campaigns and launch of Milk in Schools Scheme, 1927-8.
26. DSPH 1927, (HMSO 1928), pp.153-4
27. HOSC 1928, (HMSO 1929), p.69
28. *ibid.*
29. HOSC 1935 (HMSO 1936), Tables XIX and XX, p.150.
30. for a brief summary of industrial conditions see H.Pelling, A HISTORY OF BRITISH TRADE UNIONISM, (Pelican, 1973) pp.160-180.
31. HOSC 1935, (HMSO 1936), p.38.
32. HOSC 1936, (HMSO 1937), p.20 ; see also HOSC 1937, (HMSO 1938) p.32 for official recognition of the need for more solid meals.
33. see for example PRO ED123/264, Salford; PRO ED123/265, Sheffield; PRO ED123/290a, Monmouthshire; PRO ED123/228, Derby; PRO ED123/266, Smethwick; PRO ED123/257, Plymouth; PRO ED123/42, Jarrow.
34. PRO ED123/290a, Board of Education enquiry into LEA income thresholds for the provision of free meals, 1938.
35. DIET AND HEALTH IN PRE WAR BRITAIN, Carnegie Foundation (1952).
36. PRO ED123/290a, Board of Education enquiry into LEA income thresholds for the provision of free meals, 1938.
37. R.F. George, JNL. ROY. STATIST. SOC. (1937), Part I, pp.75-95.
38. H.C. Corry Mann, 'DIETS FOR BOYS DURING THE SCHOOL YEARS', *op.cit.*
39. MRC 1500 MacFadden to Newman, 17 Dec 1920.
40. Sir Walter Fletcher 'Urgency of Nutritional Studies' NUT ABSTRACTS & REVIEWS (1932) I, 3, p.357.
41. MRC 2034, Landsborough Thompson to Corry Mann , 20 March 1923; 25 Feb 1924.
42. MRC 2034, Paton to Fletcher, 25 June 1924.
43. MRC 2034 Fletcher to Paton, 15 May 1924; 24 June 1924; 27 June 1924.
44. MRC 2034, Paton to Fletcher, 10 July 1924.
45. MRC 2034, Hopkins to Fletcher, 7 Feb 1924; 16 Oct 1924.
46. MRC 2034, 'Comment on Interim Report of Dr Corry Mann', 16 July 1924.

47. Sir Walter Fletcher, NUT ABS & REVIEWS (1932) 1,3 p. 357.
48. Corry Mann, op.cit., pp.8-9.
49. K.M. Hambidge et.al. in PROCEEDINGS OF THE XIII INTERNATIONAL CONGRESS OF NUTRITION (John Libbey, 1966) pp 513-515; L.A. Malcolm, BRITISH JNL. NUTRITION (1970) 24, pp.297-305.
50. Fletcher (1932), op.cit.
51. FAO/WHO, ENERGY AND PROTEIN REQUIREMENTS, WHO Tech Rept. Ser. No. 522 (1973).
52. Corry Mann, op.cit., p.54.
53. FAO/WHO (1973) op.cit.

CHAPTER VI

1. Cmd. 2175, 'Report of the Interdepartmental Committee on Physical Deterioration (1904); Cmd. 2210, 'Interdepartmental Committee on Physical Deterioration, Minutes of Evidence', (1904) .
2. Collie, 'Committee on Physical Deterioration, Minutes of Evidence', Q. 9970-9971.
3. C. Funk, JOURNAL OF STATE MEDICINE (1912) 20, 341.
4. J.W.P. Rivers, BRITISH JNL. OF CLINICAL PRACTICE, (1984), 38, No.5, Symposium Supplement 31, pp.100-109.
5. Sources: K.Y. Guggenheim, NUTRITION AND NUTRITIONAL DISEASES (Collamore Press, 1981) and E.V. McCollum, HISTORY OF NUTRITION (Houghton Mifflin, 1957)
6. J.C. Drummond and A. Wilbraham, The ENGLISHMAN'S FOOD (Jonathan Cape , revised edn., 1954) pp.387-388.
7. see for example H. MacKay, 'Dietetic Deficiencies and Susceptability to Infection', LANCET, 11, 29 December 1934, pp.462-466.
8. Guggenheim, NUTRITION AND NUTRIIONAL DISEASE op. cit. p.19, pp.268-9.
9. ibid. p.24, p269.
10. ibid. p.22.
11. ibid pp.26-28, p.269.
12. ibid pp.269-270.
13. ibid p.270.
14. ibid p. 271.
15. E.V. McCullom, HISTORY OF NUTRITION op cit, p.232.
16. 'Report on the Present State of Knowledge concerning the Accessory Food Factors' MRC Spec.Rept.Ser. No. 38 (1919) pp.76-78.
17. C.E. Bloch UGESKRIFT F. LAEGER (1917) 79, p.349 cited in MRC Spec. Rept. Ser. No.38 (1919); C. E Bloch JAHRBUCH F. KINDERHEILK. (1919), 89, P405, cited in McCullom, HISTORY OF NUTRITION (Houghton Mifflin, Boston, 1957).
18. DIET AND HEALTH IN PRE WAR BRITIAIN (Carnegie Foundation 1952) p.146.
19. Thomas Moore, VITAMIN A (ELSEVIER, 1957) PP.401-411.
20. D.G Wehl and H.D. Kruse MILLBANK MEMORIAL FOUNDATION, Quarterly Bulletin, (1941) 19, p.471.
21. J. H. Kodiceck and J. Yudkin, LANCET, (1942) 11, p.753
22. E.C. Robertson and A.L. Morgan, JNL. OF NUTRITION (1946) 31, p.471.
23. J. C. Spence, ARCHIVES OF DIEESEASE IN CHILDRHOOD, (1931), 6, p.17.
24. Guggenheim op. cit. pp.265-268.
25. Moore op. cit. p.264.
26. H.J. Jehgers, JNL. AMERICAN MED. ASSOC. (1937), 109, p.756.

27. M.K. Maitra and L.J. Harris, LANCET (1937), 11, p.1009.
28. J. C Spence (1931) op. cit.
29. L.J. Harris and M.A. Abbasey, LANCET (1939) 11, 23 Dec. pp.1298-1359.
30. L.J. Harris and M. A. Abbasey, LANCET, (1939) op.cit.
31. G. Steininger and L.J. Roberts, ARCHIVES OF INTERN. MED. (1939) 64, p. 1170.
32. H.W. Josephs M. Barber and H. Conn, BULLETIN JOHNS HOPKINS HOSPITAL, (1941) 68, p.375.
33. Thompson A.M. et.al. BRITISH JNL. OPHTHALM. (1939), 23, 461 and 697.
34. L. J. Harris VITAMINS (4th Ed., C.U.P 1955) pp.206-208.
35. T. Moore, BIOCHEM. JNL. (1937) 31, p.155.
36. T Moore and A.C. Cooper, M.R.C. Spec. Rept. Ser. No. 264 (1949) p.62.
37. T. Huque and A.S. Trusswell, PROC. NUTRITION SOC. (1979), 38, 41A
38. T. Moore (1937) op. cit.
39. R.Passmore and M.A. Eastwood, HUMAN NUTRITION AND DIETETICS (Churchill Livingstone, 1986) p.135 .
40. K.J. Carpenter, HISTORY OF SCURVY AND VITAMIN C (C.U.P., 1986).
41. J. C. Drummond, THE ENGLISHMAN'S FOOD, op.cit., p.392
42. Carpenter (1986) op. cit. p163.
43. MRC 'Report on the Present state of Knowledge concerning the Accessory Food Factors', (1919) op. cit. pp.78-80.
44. L.J. Harris et.al., BIOCHEMICAL JNL. (1933), xxvii, p.2011; S.W. Johnson and S.S. Zilva, BIOCHEMICAL JNL. (1934), XXVIII, p.1393.
45. L.J. Harris M.A. Abbasey and J. Yudkin, LANCET (1936) i, p1486.
46. Passmore and Eastwood (1987) op. cit. p.148.
47. G.F. Gotlin, SKAND. ARCH. F.PHYSIOL (1931)61, p.225.
48. L.S.P. Davidson, H.W. Fullerton, J.W Howie, J.M Croll and J.B. Orr, B.M.J. (1933) i, p.685
49. ibid.
50. GEIGY, SCIENTIFIC TABLES (7th Ed., 1970).
51. DIET AND HEALTH IN PRE WAR BRITAIN (Carnegie Foundation, 1952)
52. H. Mackay , LANCET (1935), i, p.1431.
53. L.S.P. Davidson, H.W. Fullerton and R.M. Campbell, B.M.J. (1935) pp.196-198 and Passmore and Eastwood (1987) op.cit. p.461.
54. Davidson et. al. B.M.J (1933) op. cit.
55. H. Mackay, 'Nutritional Anaemia in Infancy', MRC Spec. Rept. Ser. No. 157, 1931.

56. *ibid* pp.106-119.
57. Passmore and Eastwood (1987) *op.cit* p.316.
58. *ibid.* p.161.
59. L. Wills, B.M.J. (1931) 1, 1059.
60. Davidson et.al. B.M.J. (1933) *op. cit.*
61. Cmd. 2175, 'Report of the Interdepartmental Committee on Physical Deterioration, 1904'.
62. J.W. Mackintosh, JNL. OF STATE MEDICINE (1935) XLIII, 4, pp.187-192
63. A.M.T. Tully, JNL. OF HYGIENE (1924), 23, pp 186-197.
64. C.E. Macnally, PUBLIC ILL HEALTH (Gollancz, 1935), p.100.
65. G.M.C. M'Gonigle and J Kirby, POVERTY AND PUBLIC HEALTH, (Gollancz, 1936), p.56.
66. Annual Report of the School Medical Officer, Manchester, 1937, p12.
67. E.Wilkins, PUBLIC HEALTH, (1937), June, pp.291-294.
68. see Chalmers, quoted in MED.OFF., (1932) 23 Jan. p.31.
69. Cmd. 2784, 'Interdepartmental Committee on the Medical Inspection and Feeding of School Children, Minutes of Evidence, 1904': Dr Hall, Q.5646- Q.5647.
70. R. Lightwood 'Idiopathic Hypercalcaemia with failure to thrive', PROCEEDINGS OF ROYAL SOCIETY (MED), (1952) 45, p.401 and Passmore and Eastwood (1986) *op. cit.* pp.111-112.
71. British Association, 'Final Report of the Anthropometric Committee (British Association, 1883)
72. see SOCIAL SCIENCE HISTORY, (1982) VI for a collection of articles on this question; R. Steckel EXPLORATIONS IN ECONOMIC HISTORY (1979) XVI p.636; R. Steckel, HISTORICAL METHODS (1982) XV, p.1; R.W Fogel et al in R. Rotberg and T. Rabb (eds) HUNGER AND HISTORY (C.U.P. 1985).
73. A.A. Kielmann and C. McCord, LANCET (1978), 1, pp.1247-1250; L.C. Chen, A. K.M. Chowdhury and S.L. Huffman, AMERICAN JNL. OF CLINICAL NUTRITION, (1980), 33, pp.1836-1845.
74. see E.M.B. Clements B.M.J. (1953) 11, 24 Oct. pp.897-902.
75. World Health Organisation, MEASURING CHANGE IN NUTRITIONAL STATUS, (WHO, 1983).
76. *ibid*
77. F. Gomez et.al. JNL. TROPICAL PEDIATRICS (1956) 2, p77
78. J.C. Waterlow B.M.J. (1972) 3, pp.566-569.
79. W.R. Dunstan, 'Malnutrition and Weightage' MED. OFF. (1934), 3 March pp. 85-87.
80. J.C. Waterlow, (1972), *op. cit.*
81. for example W.H.O. MEASURING CHANGE IN NUTRITIONAL STATUS (Geneva, 1983).
82. E. Elderton, BIOMETRICA (1914), X, pp.288-339.

83. DIET AND HEALTH IN PRE WAR BRITAIN (1952) op. cit.
84. 'Nutrition Report' (British Medical Association, 1933) pp.21-42.
85. R.F. George, JNL. ROYAL STATIST. SOC. (1937) part1, pp.75-95.
86. WHO (1983) op.cit.
87. ibid
88. K.M. Hambridge et.al. in PROCEEDINGS OF THE XIII INTERNATIONAL CONGRESS OF NUTRITION ed T.G. Taylor and N.K. Jenkins (John Libbey, 1986) pp.513-515.
89. L.A. Malcolm, BRITISH JNL. NUTRITION (1970), 24, pp.297-305.
90. NUTRITION ABSTRACTS AND REVEIWS (1979) 46, No.8, pp.591-608.
91. A.M.T. Tully, 'The Physique of Glasgow School Children', JNL. OF HYGIENE (1924) 23, pp.186-197.
92. G. F. Rose, 'The Incidence and Effect of Rickets', MED.OFF., (1924) 27 December, p.297, 'Factors Influencing the Growth and Nutrition of Children', MED.OFF., (1926), pp.271-2 .
93. H. MacKay and S.F. Rose 'Vitamin D Deficiency, Dental Caries and Tonsillar Enlargement', LANCET, (1931), ii, pp.1230-1235.
94. DIET AND HEALTH IN PRE WAR BRITIAN (Carnegie Foundation, 1952)
95. Cmd.2337, 'Memorandum on the Consumption and Cost of Food in Workingmen's Families (HMSO 1904).
96. For a description of method used in MRC studies conducted by Cathcart, see D.E. Lindsay and D. Noel Paton, 'Study of the Diet of the Labouring Classes in the City of Glasgow (Glasgow, 1913), Introduction.
97. B.S. Rowntree, POVERTY, A STUDY IN TOWN LIFE (1902)
98. D.Oddy, ECONOMIC HISTORY REVIEW (1970), pp.314-323.
99. DIET AND HEALTH IN PRE WAR BRITAIN, (Carnegie Foundation, 1952).
100. J. Boyd Orr FOOD, HEALTH AND INCOME, (Macmillan, 1936); see also PRO MH56/215 for contemporary criticisms of this survey.
101. PRO MH 56/215, Briefing papers prepared for Minister of Health prior to the House of Commons debate on Malnutrition, 8 July 1936.
102. 'Recommended Daily Amounts of Food Energy and Nutrients for Groups of People in the UK', DHSS 1979.
103. J. Yudkin, ENCYCLOPEDIA OF NUTRITION (Penguin, 1986) pp.258-259.
104. J.P.W. Rivers and P.R.Payne 'The Comparison of Energy Supply and Energy Requirements' pp.85-105 in ENERGY AND EFFORT, ed G.A. Harrison (Arnold 1981) 105. W.D. Atwater, USA DEPT OF AGRICULTURE, (1895) Bulletin 21; Rowntree op. cit; E.P. Cathcart, STUDIES IN NUTRITION, MRC Spec.Rept.Ser.165 (1932).
106. THE CRITICISM AND IMPROVEMENT OF DIETS, Ministry of Health (HMSO 1932); Report of Committee on Nutrition

(British Medical Association, 1933)

107. FAO 'Energy and Protein Requirements': Technical Report Series No. 52 (Geneva, 1973)

108. Oddy op.cit.

109. DHSS (1979) op. cit.

110. Passmore and Eastwood (1987) op.cit.

111. DHSS (1979) op. cit.

112. E.M. Hume and H.A. Krebs , M.R.C. Spec.Rept Ser. No.264 (1949)

CHAPTER VII

1. MRC PF 133, Greenwood to Fletcher, 31 Oct. 1931