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THE DEVELOPMENT OF THE CONCEPT OF DIETARY FIBRE

H. C. Trowell*‡ and D. P. Burkitt†‡

**Woodgreen, Fordingbridge SP6 2AZ, U.K.*
†Bussage, Stroud GL6 8AX, U.K.

1. Introduction

The Royal College of Physicians report on dietary fibre (34) concluded that increasing the proportion of dietary fibre in western diets would be nutritionally desirable. Three years later the Health Education Council (22) went further; it made long term proposals that the whole population should alter their diet in several respects, and the largest recommended change was that dietary fibre intakes should increase from 20 g to 30 g per head per day. Let us look back from this starting point to see how our modern concepts about dietary fibre have evolved.

The Royal College of Physicians report (34) summarised the rise and fall of interest in bran in the United States during the early years of the present century (27, 48). Many had taken bran either to treat or prevent constipation (20) and the effects on laxation of the main constituents of fibre, namely cellulose, hemicelluloses and lignin had been studied (53). On the other hand famous gastroenterologists, such as Alvarez (1), had condemned its use as a laxative and the American Medical Association Council on Foods (2) found little in its favour.

Fibre has been called "roughage" and has been regarded as a gastrointestinal irritant. Medical opinion therefore recommended that the dietary fibre content of flour should be as low as possible and, as the correspondence columns of British medical journals during the Second World War show, the serious shortage of imported wheat, which necessitated the milling of high-fibre "National" flour, was deprecated; the crude fibre content of this flour was carefully analysed and reported at frequent intervals for several years.

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‡H. C. Trowell formerly Consultant Physician, Uganda and D. P. Burkitt formerly Medical Research Council. Both formerly Makerere University and Mulago Hospital, Kampala, Uganda.

2. Diseases of Modern Civilisation — "Western Diseases"

The old term "diseases of modern civilisation" is unacceptable because it stigmatises Third World communities as uncivilised. It is better to refer to "Western diseases" - which are diseases characteristic of modern Western technological communities (47).

Doctors in sub-Saharan Africa during the 1930s and 1940s recognised that certain diseases commonly met in Western communities were rare in rural African peasants. This hearsay talk greeted any new doctor on arrival in Africa. Even the teaching manuals of African nurses and hospital assistants stated that diabetes, coronary heart disease, appendicitis, peptic ulcer, gallstones, haemorrhoids and constipation were rare in African blacks who "eat foods that contain many skins and fibres, such as beans and maize meal, and pass a bulky stool two or three times a day" (37). Surgeons noticed that the common acute abdominal emergencies in Western communities were virtually absent in rural African peasants (3).

Necropsy studies also showed how rare were these diseases of Western civilisation (51). In 1927 the results of the first thousand Kenyan necropsies (51) were summarised thus: "not a single case of appendicitis or coronary thrombosis, only three diabetics, one healed peptic ulcer scar, no gallstones in an adult, and no evidence of essential hypertension". In the 1920s highland Kenyans had not added salt to their food; this began slowly in the 1930s and essential hypertension was first recorded in East Africans in the 1940s (46). Only since westernisation of the diet and life-style are these diseases beginning to emerge; some 5% of a 1973 necropsy series of 400 Africans had died from these metabolic and degenerative diseases and coronary heart disease was emerging in those over 60 years but not at an earlier age (19).

Table 1 lists the common western diseases together with the books that show their rarity in African peasant communities. Cleave and Campbell (16) also reported data from parts of India and Trowell and Burkitt (47) included data from medical schools in five continents which suggests that the incidence of these diseases is increasing following westernisation and they added the following to those in the table: essential hypertension, stroke, peripheral vascular disease, dental caries, gout, thyrotoxicosis, pernicious anaemia, and cancer of the breast and lung.

It is interesting to note that the only disease later removed from this list (Table 1) is duodenal ulcer. It had been included by Donnison (18), a medical officer in Kenya who made enquiries about these diseases from eleven doctors in Africa, Asia and America. Trowell noted that, although the incidence of peptic ulcer was low in most parts of sub-Saharan Africa, it was very high in immigrant labourers from the Nile-Zaire watershed (38). This book, *Non-infective Disease in Africa* (38), was written in 1960 and is based on Trowell's own clinical experience, the necropsy records of Makerere University Hospital and on over 30(references from the medical journals of sub-Saharan Africa. It carries one of the first references to the possible protective action of fibre in diverticular disease, cancer and polyps of the large bowel and includes data from J. Higginson and A. G. Dettle, working in Johannesburg, on the low incidence of colo-rectal cancer in the Bantu. Apart from the fourteen diseases listed in Table 1, Trowell also shows some evidence for an association between Western culture and another eleven diseases (stroke, multiple sclerosis, pernicious anemia, ulcerative colitis, Crohn's disease, irritable bowel syndrome, myxoedema, Hashimoto's thyroiditis, gout, Paget's osteitis and polypoid disorders of the colon) and this is repeated in the later book with Burkitt (47).

Diabetes mellitus was still a relatively uncommon disease in Ugandans in the

Table 1. Common Western Diseases Not Seen Elsewhere

Diseases mentioned	References
Constipation	38, 16, 11, 47
Haemorrhoids	38, 16, 11, 47
Diverticular disease of the colon	38, 16, 11, 47
Appendicitis	18, 21, 38, 16, 11, 47
Colo-rectal cancer	21, 38, 11, 47
Obesity	18, 38, 16, 11, 47
Diabetes mellitus	18, 21, 38, 16, 11, 47
Coronary thrombosis	18, 21, 38, 16, 11, 47
Gallstones	18, 21, 38, 16, 11, 47
Varicose veins	18, 38, 16, 11, 47
Renal stones	18, 21, 38, 16, 11, 47
Duodenal ulcer	18, 21, 38(?), 16, 11(?), 47(0)
Hiatus hernia	38, 16, 11, 47
Deep venous thrombosis/Pulm. embolism	38, 16, 11, 47

?, thought to be doubtful.

0, thought not to be an exclusively Western disease.

1950s and this was thought to be due to their traditional diet of lightly processed "high-carbohydrate (starch) low-fat diets which possibly contribute to a lower fasting blood sugar and a flatter tolerance curve" (38). Many years later this was repeated as the formal hypothesis "that (prolonged consumption of) fibre-depleted starch foods is conducive to the development of diabetes in susceptible genotypes" (42,43) and Mann has recently offered some support for this proposed aetiology of Type II (non-insulin dependent) diabetes (26).

3. Cleave and the Saccharine Disease

Undoubtedly the surge of interest in dietary fibre in Britain stemmed from the books and other writings of the late Surgeon-Captain T. L. Cleave. He based his ideas (14) on a consideration of Darwinian evolution; animals and man, he maintained, had adapted well to their natural foods since animals, for instance, rarely develop diseases comparable to those characteristic of man's modern civilisation. In recent centuries machinery has been developed excessively to process man's carbohydrate foods, and sugar has been refined and concentrated by

removing its natural fibre (bagasse). In addition consumption has risen enormously in the past 150 years. Although the same had occurred with flour, Cleave considered that the former was more injurious because sugar was far more concentrated than flour. Concentrated refined carbohydrates led to overconsumption which caused obesity, diabetes, coronary thrombosis and gallstones; removal of fibre caused constipation, haemorrhoids, diverticular disease and varicose veins (16).

Cleave had been the medical officer to the British battleship King George V during the Second World War. He had treated constipated sailors with processed raw bran, which he called natural unprocessed bran (13). How ironical to prescribe the very bran which millers had removed to treat the constipation which its removal had caused! Cleave eventually elaborated his views in a series of books: on coronary heart disease (caused, he taught, by refined carbohydrates and not by fat); on varicose veins (caused by constipation): and on peptic ulcer (caused by the removal of protein in the refined flour). Together with D. G. Campbell, a South African physician who has done a great deal of research on South African Bantu diabetics, he wrote *Diabetes, Coronary Thrombosis and the Saccharine Disease* (16), and in a later edition they were joined by N. E. Painter (17).

Painter studied intracolonic pressure in diverticular disease and had begun to treat this disorder with bran by 1967, His monograph on diverticular disease contained much evidence that this disorder was due to the low-fibre diets of modern Western countries and it is his work which has been largely responsible for the revolution in treatment of this disorder (29). Anout this time, Cleave wrote his final edition of the saccharine disease book (15) which, while still concentrating on the ill-effects of sucrose, placed more emphasis on the protective action of fibre. Burkitt (9) and Heaton (23) have expressed their gratitude for the enormous stimulus that they received from Cleave's books. Both had minds ready for solutions to unresolved but different problems - Heaton was investigating the relationship of diet to the formation of gallstones and Burkitt had realised that African peasants rarely developed many of the diseases of civilisation.

Trowell was far more cautious. It was his experience that it had taken some 30 years to unravel the various dietary factors responsible for kwashiorkor in African children which was not merely due to protein calorie malnutrition as many had supposed (49). For one who had participated in this long debate it appeared unlikely that a single cause, the concentrating of sugar and flour, could produce so many varied diseases of civilisation. Even so, Cleave's first sentence about "based on evolution" acted like a charm to Trowell.

Another colonial doctor had already wrestled with this problem. The word "evolution" occurs in the first sentence of the preface and in the last sentence of the book *Civilisation and Disease* (18) in which, in 1937, Donnison suggested that these "new diseases" appeared to be diseases of maladaptation. Man had adapted well to the diet, exercise and life-style of the hunter-gatherers and had eaten this ancient peasant diet for thousands of years. This had been high-starch (lightly processed, retaining most of the fibre), low-fat, low-sugar and low-salt (31). Modern Western diets are very different and the Health Education Council (22) has now recommended a partial return to the diet of ancient man.

4. Dietary Fibre Group

Burkitt, a surgeon, came to Uganda in 1946 and travelled to many parts of Africa and other continents to determine the geographical distribution of Burkitt's

lymphoma which he first described. While doing so he, too, became convinced that many surgical and medical conditions commonly seen in the Western world were rare in rural peasant communities. In 1966 he returned to England to continue his work on cancer with the Medical Research Council and this brought him into contact with Sir Richard Doll who, in 1969, introduced him to Surgeon-Captain Cleave. During the next two years Burkitt sent monthly questionnaires to some 150 hospitals, mostly rural institutions, in Africa and Asia and their responses confirmed his ideas about the rarity of certain diseases.

Cleave had suggested that refined sugar caused inflammation of the appendix by altering the bacterial flora of the large bowel. Although sugar is absorbed in the upper part of the small intestine, unabsorbed fibre reaches the caecum and appendix and Rendle-Short, Professor of Surgery at the University of Bristol, had said in 1920 (35) that fibre (which he called cellulose) was a protective factor in appendicitis. This was supported by Burkitt (5) who went on to suggest that colo-rectal cancer had a similar aetiology (6).

Meanwhile Trowell had left Africa hurriedly in 1958 because of a serious illness of his wife and had become ordained and vicar of a country parish in Wiltshire. This had prevented an adequate conclusion to the book on *Non-infective Diseases in Africa* (38); but in 1970 he returned to Uganda to deliver a lecture on the history of kwashiorkor and met Burkitt again in the Makerere University Medical School where he was lecturing on fibre and colonic diseases.

In the 1970s Burkitt became the focal point of a group of English doctors who had been stimulated by Cleave's hypothesis. He collected more epidemiological data and visited and lectured frequently in all five continents. He emphasised that this group of disorders were associated in geographical distribution and often in individuals; for instance diabetics often develop coronary heart disease, and gallstones are commonly associated with diverticular disease or hiatus hernia. This suggests either a single causative factor, possibly a deficiency of dietary fibre, or a group of associated causative factors, in the Western way of life (4). He examined the effect of dietary fibre on stool weight and oro-anal transit time and reported the epidemiology of varicose veins, deep vein thrombosis and haemorrhoids (7), of large bowel diseases (8), and of hiatus hernia (10), relating all these to our lower fibre diet.

5. The New Definition of Dietary Fibre

In 1971 Burkitt and Trowell wished to write a book on refined carbohydrate foods and disease, but "fibre" had never been adequately defined and they could not find terms such as fibre, roughage, or cellulose in any British textbook of medicine, surgery, or nutrition, nor in the British food tables. Even the Medical Research Council puplications were confused. The third edition of McCance and Widdowson's *Composition of Foods* (25) reported the available carbohydrates (starch, dextrins and sugars) and the unavailable carbohydrates of fruits and vegetables, but did not define the latter term. In the same book Widdowson affirmed that the "so-called unavailable carbohydrates are made up of hemicelluloses and fibre" (p. 171) but, because fibre was not defined, many readers might have considered that it was synonymous with cellulose. Indeed many investigators were adding wood cellulose to chemically defined diets given in animal experiments. In the United States Van Soest stated that "terms such as fibre, cellulose and lignin are not often found in the indices of our food and nutrition textbooks and, when they are found, the information given is usually inaccurate" (52).

Platt (32) had reported the fibre content of tropical foodstuffs without defining

fibre! His figures merely reported *crude* fibre, the food residue left after sequential extraction with dilute acid and dilute alkali. Some 85% of the hemicelluloses and up to 50% of the cellulose are destroyed in this extraction (52). Nicholls (28) and Hipsley (24) used Platt's figures in their publications without querying their validity.

When this confusion concerning fibre came to light, Trowell, after correspondence with Professor McCance, conferred with Southgate, Eastwood and Heaton and redefined fibre, calling it dietary fibre. This retained the time-honoured noun but changed the inappropriate adjective. A study of books on botany and milling had convinced him that fibre represented a *group* of substances, largely polysaccharides, best defined as "that portion of food which is derived from the cellular walls of plants and is digested very poorly by human beings ... Fibre is composed largely of cellulose ... arranged in long chains ... mingled with other substances which are poorly digested: hemicelluloses, pentosans, pectin and lignin" (39). This definition was shortened in other communications which stressed the large difference between crude fibre and dietary fibre (40, 41). Indigestibility by human alimentary enzymes is only one of the characteristics of the polysaccharides present in dietary fibre. Early in 1976 a suggestion was made that the definition should be enlarged to include all "the residue of plant food resistant to hydrolysis by human alimentary enzymes" (44).

This enlarged redefinition of dietary fibre received the support of a meeting of the Oxford Nutrition Society (50), and the agreement of Southgate (36) who incorporated it in the Fourth Revised edition of McCance and Widdowson's *The Composition of Foods* (30) as: *"the sum of the polysaccharides and lignin which are not digested by the endogenous secretions of the human gastrointestinal tract"*.

In the United States Prosky and Harland (33), acting on behalf of the U.S. Association of Official Analytical Chemists (AOAC), circulated over a hundred scientists who accepted the revised definition of dietary fibre (50). They inaugurated the AOAC collaborative study in which some 42 collaborators in 14 countries received food samples for analysis of total dietary fibre and will soon be publishing the results.

References

1. Alvarez, W. C. (1931). Opinions of 470 physicians in regard to the advantages and disadvantages of using bran and roughage. *Minn. Med.* 14, 296-300.

2. American Medical Association Council on Foods (1936). The Nutritional significance of bran. *J. Am. Med. Ass.* 107, 874-877.

3. Burkitt, D. P. (1952). Acute abdominal emergencies in Uganda and England. *E. Afr. Ned. J.* 29, 189-193.

4. Burkitt, D. P. (1970). Relationship as a clue to causation. *Lancet* 2, 1237-1240.

5. Burkitt, D. P. (1971). The aetiology of appendicitis. *Br. J. Surg.* 58, 695-699.

6. Burkitt, D. P. (1971). Epidemiology of cancer of colon and rectum. *Cancer* 28, 3-13.

7. Burkitt, D. P. (1972). Varicose veins, deep vein thrombosis and haemorrhoids; epidemiology and suggested aetiology. *Br. Med. J.* 2, 556-561.

8. Burkitt, D. P. (1973). Epidemiology of large bowel diseases: the role of fibre. *Proc. Nutr. Soc.* 32, 145-149.

9. Burkitt, D. P. (1982). The fibre story today. *Update* 37-40.

10. Burkitt, D. P. and P. A. James (1973). Low residue diets and hiatus hernia. *Lancet* 2, 128-130.

11. Burkitt, D. P. and H. C. Trowell (1975). *Refined Carbohydrate Foods and Disease: Some Implications of Dietary Fibre*. Academic Press, London.

12. Burkitt, D. P., A. R. P. Walker and N. S. Painter (1972). Effect of dietary fibre on stools and transit times, and its role in the causation of disease. *Lancet* 2, 1408-1412.

13. Cleave, T. L. (1941). Natural bran in the treatment of constipation (letter). *Br. Med. J.* 1, 461.

14. Cleave, T. L. (1956). The neglect of natural principles in current medical practice. *J. Roy. Nav. Med. Serv.* 42, 55-83.

15. Cleave, T. L. (1974). *The Saccharine Disease*. Wright, Bristol.

16. Cleave, T. L. and B. D. Campbell (1966). *Diabetes, Coronary Thrombosis and the Saccharine Disease*. Wright, Bristol.

17. Cleave, T. L., G. D. Campbell and N. S. Painter (1969). *Diabetes, Coronary Thrombosis and the Saccharine Disease*, 2nd Edn. Wright, Bristol.

18. Donnison, C. R. (1937). *Civilisation and Disease*. Bailliere Tindall Cox, London.

19. Drury, R. A. B. (1973). The cardiac pathology of elderly Ugandan Africans. *E. Afr. Med. J.* 50, 566-573.

20. Gallant, A. E. (1912). Wheat bran. Its chemical and physical characteristics in the treatment of constipation. *N. Y. Med. J.* 96, 414-417.

21. Gelfand, M. (1944). *The Sick African*. Stewart Printing, Cape Town.

22. Health Education Council (1983). *Proposals for Nutritional Guidelines for Health Education in Britain*. HMSO, London.

23. Heaton, K. W. (1980). T. L. Cleave and the fibre story. *J. Roy. Nav. Med. Serv.* 66, 5-10.

24. Hipsley, E. H. (1953). Dietary 'fibre' and pregnancy toxaemia. *Br. Med. J.* 2, 420-422.

25. McCance, R. A. and E. M. Widdowson (1969). *The Composition of Foods*, 3rd Edn. HMSO, London.

26. Mann, J. Diabetes Mellitus. In: *Dietary Fibre, Fibre-depleted Foods and Diseases*. (H. C. Trowell and D. P. Burkitt, eds.), Chapt. 16. Academic Press, New York and London.

27. National Institute of Health (1978). Role of dietary fibre in health. *Am. J. Clin. Nutr. Suppl.* 31, S1-S291.

28. Nicholls, L. (1961). *Tropical Nutrition and Dietetics*, 4th Edn. Bailliere Tindall Cox.

29. Painter, N. S. (1975). *Diverticular Disease of the Colon: A Deficiency Disease of Western Civilisation*. Heinemann, London.

30. Paul A. A. and D. A. T. Southgate (1978). *McCance and Widdowson's The Composition of Foods*, 4th Edn. HMSO, London.

31. Perisse, J., F. Sizaret and P. Francoise (1969). The effect of income on the structure of the diet. *FAO Nutrition Newsletter* 7, 1-9.

32. Platt, B. S. (1962). Tables of representative values of foods commonly used in tropical countries. *Med. Res. Coun. Spec. Rep. Ser.* No 302. HMSO, London.

33. Prosky, L. and B. Harland (1985). Dietary fibre methodology. In: *Dietary Fibre, Fibre-Depleted Foods and Diseases* (H. C. Trowell and D. P. Burkitt, eds.), Chapt. 5. Academic Press, New York.

34. Royal College of Physicians of London (1980). *Medical Aspects of Dietary Fibre*. Pitman Medical. London.

35. Short, A. R. (1920). The causation of appendicitis. *Br. J. Surg.* 8, 171-186.

36. Southgate, D. A. T. (1977). The definition and analysis of dietary fibre. *Nutr. Rev.* 35, 31-37.

37. Trowell, H. C. (1939). *Diagnosis and Treatment of Diseases in the Tropics*. Bailliere Tindall Cox, London.

38. Trowell, H. C. (1960). *Non-Infective Disease in Africa*. Edward Arnold, London.

39. Trowell, H. (1972). Dietary fibre and coronary heart disease. *Rev. Eur. Etud. Clin. Biol.* 17, 345-349.

40. Trowell, H. (1972). Crude fibre, dietary fibre and atherosclerosis. *Atherosclerosis* 16, 138-140.

41. Trowell, H. (1972). Ischemic heart disease and dietary fiber. *Am. J. Clin. Nutr.* 25, 926-932.

42. Trowell, H. (1974). Diabetes mellitus death-rates in England and Wales 1920-70 and food supplies. *Lancet* 2, 998-1002.

43. Trowell, H. (1975). Dietary-fiber hypothesis of the aetiology of diabetes mellitus. *Diabetes* 24, 762-765.

44. Trowell, H. (1976). Definition of dietary fibre and hypotheses that it is a protective factor in certain diseases. *Am. J. Clin. Nutr.* 29, 417-427.

45. Trowell, H. (1978). The development of the concept of dietary fibre in human nutrition. *Am. J. Clin. Nutr.* 31, S3-S11.

46. Trowell, H. (1981). In: *Western Diseases: Their Emergence and Prevention* (H. C. Trowell and D. P. Burkitt, eds.), Chap. 1. Edward Arnold, London.

47. Trowell, H. C. and D. P. Burkitt (1981). *Western Diseases: Their Emergence and Prevention*. Edward Arnold, London.

48. Trowell, H. C. and D. P. Burkitt (1984). Bran yesterday ... bran tomorrow? (letter). *Br. Med. J.* 289, 436.

49. Trowell, H. C., J. N. P. Davies and R. F. A. Dean (1983). *Kwashiorkor*. Academic Press, New York.

50. Trowell, H., D. A. T. Southgate, T. M. S. Wolever, A. R. Leeds, M. A. Gassull, and D. A. Jenkins (1976). Dietary fibre redefined. *Lancet* 1, 967.

51. Vint, F. W. (1937). Postmortem findings in the natives of Kenya. *E. Afr. Med. J.* 13, 332-340.

52. Van Soest, P. J. (1978). Dietary fibers: their definition and nutritional properties. *Am. J. Clin. Nutr. Suppl.* 31, S12-S20.

53. Williams, R. D. and W. H. Olmsted (1936). The effect of cellulose, hemicellulose and lignin on the weight of the stool: contribution to the study of laxation in man. *J. Nutr.* 11, 433-449.