DIET, BOWEL MOTILITY, FAECES COMPOSITION AND COLONIC CANCER*

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SUMMARY

The commonness of colonic cancer in privileged populations compared with its rarity in those pursuing a primitive manner of life suggests that environmental factors are primarily responsible. In this study, differences in diet and their ramifications are discussed in relation to populations prone and less prone to the disease. Some possible hypotheses of causation are considered. It is concluded that in the present contexts of western populations, in so far as diet directly or indirectly is involved, there is little or no likelihood of lowering the present high prevalence of cancer of the colon.

Recent contributions by Higginson,[1] Wynder and coworkers,[2,3] Burkitt,[4] and also editorial comment[5,6] have underlined the present high mortality from cancer of the colon in western populations. The disease is now responsible for 2 - 3% of all deaths; it accounts for about 15% of all deaths from cancer, being second only to lung cancer.[1-4] Several questions arise. Firstly, what aetiological leads are suggested by the epidemiology of the disease? Secondly, how do populations with contrasting prevalences of the disease differ, not only in diet and manner of life, but more particularly, in bowel motility, faeces composition and related parameters? Thirdly, in the light of such knowledge, what are the possible causes of the disease? Fourthly, and most important, in so far as diet is implicated, is there any likelihood of preventing or of retarding the development of colonic cancer?

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PREVALENCE

During the last century, mortality has increased considerably; examination of the data available suggests that the increase is due only in part to the ageing of populations. Among technically retarded populations prevalence is low, constituting as little as 1% of all cancers.[1,4,7,8] Prevalence rises when less privileged populations migrate to prosperous regions.[2] Within countries, both western and underdeveloped, the condition is more prevalent in large towns compared with country districts.[2,9] As Burkitt[4] has emphasized, the foregoing evidence indicates that the causative or promotive factors are largely, if not wholly, environmental. In the contrasting populations cited, the salient differences in environmental factors concern diet, physical activity, smoking and stress.

DIETARY DIFFERENCES

The diet consumed by primitives or the less privileged differs markedly from that eaten by sophisticated populations. In the problem at issue perhaps the most plausible difference is the far greater frequency of exposure of the latter to chemicals involved in food production and preservation, also to industrial and other pollution. Preoccupation with this possible source of carcinogens, however, has tended to obscure other dietary changes which may well be influential, yet whose physiological ramifications are insufficiently appreciated. Broadly, the diet of underprivileged populations is lower in calories, in animal protein and often total protein, and in animal as well as total fat. It has higher concentrations of less refined carbohydrate foodstuffs, and also of crude fibre. It

has lower proportions of mineral salts such as calcium and often iron, and usually, although not invariably, there are lower concentrations of most vitamins. In short, among primitive or technically retarded populations, there are higher consumptions of staples such as cereals, legumes and tubers, but lower consumptions of meat, dairy produce and sugar. Such a diet, from its high fibre content, is high in bulk-forming capacity and in residue.

An indication of the bearing of these contrasting diets in respect of bowel motility and faeces composition may be gained by referring to the South African Bantu. In Johannesburg, Oettlé[7] showed that colonic cancer has about one-tenth of the prevalence (age specific) in Bantu compared with Caucasians. Regarding bowel motility, observations on Bantu and Caucasian subjects (schoolchildren and students thus far studied) have revealed a number of points.[10-14]

The mean time of traversal of digesta is 2 - 3 times faster in Bantu than in Caucasians.[13,14] In terms of exposure of bowel mucosa to digesta, carmine studies have shown that the mean time of the slowest third of Bantu may be quicker than the fastest third of Caucasians. Even in Bantu and Caucasian subjects *with the same frequency of defaecation* observations have shown that traces of carmine colour continue to be seen far longer in Caucasians than in Bantu. In a few Caucasians the period was as long as 5 - 7 days. As already shown,[14] the inter-racial differences described will be more satisfactorily elucidated on using Hinton's[15] pellet technique.

There is a far greater frequency of motions in Bantu than in Caucasians.[10,13,14,16] In rural Bantu, 30% were found to have 3 motions daily compared with 1 -2% in the case of Caucasians; the latter proportion is similar to data reported for subjects in England.[17] There is an enormous difference in the ability to produce a stool within, say, 10 minutes of request; 80 - 98% of groups of rural Bantu children have this capacity, 70 - 80% of partially sophisticated urban Bantu, but less than 10% of Caucasian children.[13,14] Previously, it was noted that when young Caucasian adults consumed a Bantu type of diet for several days, they still could not produce a stool on request in the same facile manner as Bantu.[11]

Unformed stools are frequently encountered among rural Bantu; they are noted less often in urban Bantu, and seldom with Caucasians. Some studies have suggested that there is a greater occlusion of gas in Bantu stools.[10] The mean weight of dry faeces voided daily is double or more than that of Caucasians.[10] There are higher faecal excretions of total nitrogen, bacterial nitrogen, fat and cellulose components in the Bantu.[10] In these people, moreover, there are greater excretions *per diem* of bile acids and sterols;[18] i.e. there is less intestinal degradation of these substances in Bantu than in Caucasians.

There are very likely to be big differences in the bacterial flora in the faeces of the two populations; Aries *et al.*[19] reported a far lower proportion of 'bacteroides' in stools of Ugandan compared with English subjects, populations with very contrasting prevalences of colonic cancer. It is in recognition of the various differences described that it has been suggested that 'in the faeces, so little studied, may lie the answer' to the causation of colonic cancer.[5]

OTHER DIFFERENCES

While differences in diet are believed to be virtually wholly responsible for the contrasts enumerated above, it must be kept in mind that there are marked differences between Bantu and Caucasians in respect of habitual physical activity, cigarette smoking, and stressful situations.[20] Of these factors, at least physical activity has a significant bearing on bowel motility.

For cancer to develop, apart from the necessary presence of carcinogenic stimuli, almost certainly there are other requirements. These may well include transit time of digesta which regulates the contact time of carcinogens present, intestinal pH, also particular rates of absorption, reabsorption, secretion, and excretion of nutrients and metabolites.

HYPOTHESES

The first possibility, understandably, is that the diet of more prone populations contains higher concentrations of adventitious or even natural carcinogens. These may operate independently of intestinal conditions; on the other hand, such carcinogenesis as is feasible may be determined by special conditions, such as one or more of those listed. In most western countries for many years food additives have been subjected to intensive toxicological tests. In practice, however, dietary carcinogens, active or potential, are very difficult to detect in everyday diets.[21] It is noteworthy that Denmark[22] has one of the highest mortality rates from colonic cancer in spite of the existence of extremely stringent regulations on food additives. Furthermore, in India, Malhotra[23] has shown that widely contrasting regional incidences of colonic cancer occur in populations which are relatively little exposed to sophisticated food additives. Lack of positive proof that they have a significant role in human carcinogenesis, indicates that other sources of carcinogens merit examination.

In the diets of populations with differing degrees of proneness a particular food component may be involved. One dietary feature that has attracted attention is a high fat intake, which Wynder and co-workers[2,3] regard with suspicion. However, account must be taken of the fact that high consumers such as the South-Western Indians[24] in the USA, and also African Masai and Rendille,[25] have not been reported to be characterized by a high prevalence of colonic cancer.[1,7] This does not mean, of course, that a high fat intake and its metabolic ramifications are without influence. Workers in Czechoslovakia[22] have shown that there is a positive correlation between mortality from colonic cancer and animal protein intake. No doubt a like correlation prevails with level of consumption of refined carbohydrate foods, and, in a negative respect, with intake of crude fibre. It seems unlikely, however, that a single *staple* foodstuff or nutrient consumed in excess is specifically protective or noxious.

A third and overlapping possibility is that the diets of more prone populations may lead to or be associated with the production, or higher concentration, of carcinogenic metabolites. But their noxiousness, indeed their very presence, again may depend largely, if not wholly, on one or more of the intestinal conditions enumerated. It will be extraordinarily difficult, of course, to elucidate the nature of the intestinal cytotoxic metabolites, the bacterial flora, or the other biological components, which are directly

or indirectly involved.

Burkitt[4] believes that it is the pattern of a diet, particularly in relation to its bulk-forming capacity, which primarily determines the presence or virtual absence of a series of diseases, including appendicitis, diverticulitis and colonic cancer.

FURTHER PROBLEMS AND OUTLOOK FOR THE FUTURE

The critical problem, of course, is to demonstrate an aetiological link between what is consumed, the bowel situation ensuing, and the development of colonic cancer. But to do this, formidable difficulties must be overcome. The characterization of a diet from recall in the near past is liable to considerable error; still more so must this apply to eating practices prevailing several years previously. The same reservation applies even more so to the recollection of bowel habits. There are, moreover, additional problems; thus, not all persons who believe themselves to be constipated have long transit times, and *vice versa*.[26]

Although attempts to throw light on the aetiology of colonic cancer have been very unrewarding,[1-3] research obviously must be continued. Undoubtedly, the most urgent information required is that on particular minorities of populations *within a western context* who are known to have low prevalences of the disease. Extrapolation of the experience of primitives to western populations has limited application.[27] What we urgently need to know, for example, is what hinders Scandinavian rural communities[9] from developing colonic cancer, since among such people its occurrence is only about one-third of that in the cities?

Finally, regarding the fourth question raised, what are the chances of lowering the present high mortality from colonic cancer? It is imperative to recognize that in so far as diet is crucial, sophisticated diets are here to stay; the likelihood of returning to a primitive eating pattern is practically nil. Thus, it is only necessary to consider how well known are the risk factors for coronary heart disease; yet there is minimal evidence of their recognition, as measured by the proportion of the adult population who endeavour to make the recommended changes in diet and manner of life.[28,29] The same inertia is apparent in respect of cigarette smoking and lung cancer. Briefly, if a high prevalence of colonic cancer is implicit in the consumption of western diets as presently constituted, then a fall in the frequency of the disease cannot be expected.

REFERENCES

1. Higginson, J. (1966): J. Nat. Cancer Inst., **37**, 527.
2. Wynder, E. L. and Shigematsu, T. (1967): Cancer (Philad.), **20**, 1520.
3. Wynder, E. L., Kajitani, T., Ishimawa, S., Dodo, H. and Takano, A. (1969): *Ibid.*, **23**, 1210.
4. Burkitt, D. P. (1969): Lancet, **2**, 1229.
5. Annotation (1938): *Ibid.*, **1**, 628.
6. Leading Article (1969): Brit. Med. J., **1**, 589.
7. Oettlé, A. G. (1964): J. Nat. Cancer Inst., **33**, 383.
8. Robertson, M. A. (1969): S. Afr. Med. J., **43**, 915.
9. Ringertz, N. (1967): *National Cancer Institute Monograph* No. 25, p. 219. Bethesda, Maryland: U.S. Public Health Service.
10. Walker, A. R. P. (1947): S. Afr. Med. J., **21**, 590.
11. *Idem* (1949): Nature (Lond.), **164**, 825.
12. *Idem* (1961): S. Afr. Med. J., **35**, 114.
13. Walker, A. R. P. and Walker, B. F. (1969): Brit. Med. J., **3**, 238.
14. Walker, A. R. P., Walker, B. F. and Richardson, B. D. (1970): *Ibid.*, **3**, 48.
15. Hinton, J. M. (1967): Proc. Roy. Soc. Med., **60**, 215.
16. Bremner, C. G. (1964): S. Afr. J. Surg., **2**, 119.
17. Connell, A. M., Hilton, C., Irvine, G., Lennard-Jones, J. E., and Misiewicz, J. J. (1966): Proc. Roy. Soc. Med., **59**, 11.
18. Antonis, A. and Bersohn, I. (1962): Amer. J. Clin. Nutr., **11**, 142.
19. Aries, V., Crowther, J. B., Draser, B. S., Hill, M. J. and Williams, R. E. O. (1969): Gut, **10**, 334.
20. Walker, A. R. P. (1969): S. Afr. Med. J., **43**, 768.
21. Leading Article (1969): Brit. Med. J., **1**, 591.
22. Gregor, O., Toman, R. and Prusova F. (1969): Gut, **10**, 1031.
23. Malhotra, S. L. (1967): *Ibid.*, **8**, 361.
24. Reichenbach, D D. (1967): Arch. Path., **84**, 81.
25. Shaper, A. G. and Jones, R. W. (1962): Lancet, **2**, 1305.
26. Halls, J. (1965): Proc. Roy. Soc. Med., **58**, 859.
27. Walker, A. R. P. (1964): Circulation, **29**, 1.
28. *Idem* (1966): Amer. Heart J., **72**, 721.
29. Editorial (1970): S. Afr. Med. J., **44**, 369.