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EDITORIALS

Continuing Low Colon Cancer Incidence in African Populations

In all big towns and cities in developing populations there are ongoing transitions in environmental factors—in dietary composition, physical activity, smoking practice, alcohol consumption, and stress. Contemporaneously, changes have occurred in the patterns of health/ill-health experienced. In South Africa, apart from major falls in the morbidity and mortality of young children, the urban African population has raced ahead of the white population in respect to the prevalence of obesity in women, hypertension, and diabetes (1). Yet, in remarkable contrast, minimal changes have occurred in the incidence of colonic diseases—diverticular disease, inflammatory bowel disease, and colorectal cancer. Furthermore, colorectal polyps, the precursor of most colorectal cancers, are nearly absent (2, 3). The age-standardized incidence rate per 100,000 population for colorectal cancer is 2.2; it is 18.7 for the local white population (4). Yet in African-Americans, the incidence rate of colon cancer alone, 32.8, is almost the highest in the world (5).

There have been reports of alterations in bowel behavior in local Africans, in regard of stool weight, defecation frequency, and whole gut transit time, although not fecal pH value (6, 7). Values are now intermediate between those previously observed in rural populations, and such reported in western populations. The dietary fiber intake of urban Africans has decreased from approximately 25–35 g (two generations ago) to 15–20 g daily (8). The staple cereal remains maize meal; however, it is usually refined (about 70% extraction rate). The intake of bread has increased, but this is mainly white bread. Additionally, because of their costs, consumption of vegetables and fruit are relatively low, about 2,5 helpings daily; *i.e.*, the diet is low in antioxidants. Yet, very importantly, although the dietary fiber content of maize meal has fallen, its content of "resistant" starch remains high, at 18 g/100 g (9).

Resistant starch fermentation produces more butyric acid than the fermentation of dietary fiber (10). Butyric acid is believed to reduce cancer risk by stimulating apoptosis, differentiation of cancer cells, and by inhibiting colon cancer cell invasion (10).

The large intestine is inhabited by a diverse population of anaerobic bacteria, which interact profoundly with the human host, thereby influencing his/her early development, quality of life, aging, and the promotion of a number of chronic degenerative diseases, including colorectal cancer (11).

The indigenous bacterial flora of the colon comprises about 400 different microbial species including bacteriodes, bifidobacteria, lactobacilli, and streptococci. The bacteria ferment unabsorbed carbohydrate to short chain fatty acids (SCFA), which are rapidly absorbed and to the gases carbon dioxide, hydrogen, and methane. The role of the microflora in both the cause and prevention of colon cancer has provoked much debate. The protective role of butyrate suggests that manipulation of the flora by diet could help prevent colonic disease. In the normal fetus, the gastrointestinal tract is sterile up to birth. Subsequently, the colon becomes colonized with bacteria that it encounters from the environment. After 4 days, the flora present are dependent on the type of feeding; breast-fed infants have mostly bifidobacteria and lactobacilli, which produce mainly acetic and lactic acids, whereas formula-fed infants have flora more like that of the adult, which produces, at this stage, mainly acetic and propionic acids with very little butyrate (12, 13). The colonic flora matures slowly during weaning, and many bacterial metabolic activities do not reach adult levels until the age of 2 yr (14). The adult microflora, in contrast, are very stable unless disrupted by antibiotics or provided with very large doses of individual dietary fibers or starch (15, 16). In rats it has been shown that dietary fiber fed at weaning can influence the ability of the rat microflora to ferment that fiber in adult life (17). In African children it has been shown that, before the age of 3 yr, their microflora is able to ferment carbohydrate to SCFA more readily than is the case with white children (18, 19). The differences in the behaviors described may be due to qualitative and quantitative differences in their microflora and, in the case of African children, may be attributable to their higher intake of "resistant" starch.

Thus, the low occurrence of "Western" bowel diseases would seem due, in large part, to a diet with continuing high "resistant" starch content, associated with effective colonic microflora acquired in early infancy.

Accordingly, attempts to prevent bowel diseases should begin in early childhood, with emphasis on the role of the diet mentioned. Currently, about a one-fourth of African primary schoolchildren lie under the fifth centile of US NCHS Reference Standards for height and weight for age (1). It is noteworthy that it has recently been emphasized that excessive early energy intake has promotive consequences with regard to several major cancers (20). Furthermore, the view has been expressed that it is lesser energy intake, "but only early in life," which is protective against the development of colon cancer (21).

As to the outlook, in developing populations, the proportion of the well circumstanced is very small, so that the masses of populations will continue to be relatively impoverished. Accordingly, undernutrition in childhood will continue, and possibly even worsen in some countries. In Southern Africa, dietarily, major reliance on maize as a staple food will continue. There is no likelihood of a rise in risk attributable to the consumption of red and processed meat (22).

The observations made raise the issue of why colon cancer remains very uncommon not only in other developing populations in Africa, but in those elsewhere. In the Gambia, in Western Africa, where the incidence of colon cancer is very low, mean daily intakes of carbohydrate foods by adult men have been reported to be 375 g starch and 25 g of nonstarch polysaccharides. However, the amount of resistant starch consumed daily was not determined (23). In Madras, India, colon cancer incidence is very low, at 1.5 per 100,000 (5). The staple cereal, rice, is reported to be low in nonpolysaccharide content (24). Unfortunately, its content of resistant starch was not reported, but may well be the protective factor. Clearly, more information is needed on the resistant starch content of the diets of the many populations who are at very low risk for colon cancer.

In brief, environmental factors that control colonic microflora in early childhood, linked with a diet high in resistant starch, are believed to be important factors in promoting a healthy large bowel in youth and later life. An additional protective factor may be undernutrition in childhood. It would seem that these are the primary factors responsible for the very low occurrence of colorectal adenomas in African populations.

Isidor Segal, F.R.C.P. (UK)
African Institute of Digestive Diseases
Chris Hani Baragwanath Hospital,
Johannesburg, South Africa

Christine A. Edwards, Ph.D.
Department of Nutrition
Glasgow University
Glasgow, United Kingdom

Alexander R. P. Walker, D.Sc.
South African Institute for Medical Research
Johannesburg, South Africa

REFERENCES

1. Walker ARP. The nutritional challenges in the New South Africa. Nutr Res Rev 1996;9:33–65.
2. Walker ARP, Segal I. Effects of transition on bowel diseases in Sub-Saharan Africans. Eur J Gastroenterol Hepatol 1997; 9:207–10.
3. Marcovic S, Boytchev H, Oettle GJ. The changing incidence of large bowel cancer in the Black population of the Witwatersrand. S Afr J Surg 1998;36:154.
4. Sitas F, Blaauw D, Terblanche M, et al. Cancer in South Africa, 1992. Johannesburg: National Cancer Registry, South African Institute for Medical Research, 1997.
5. Parkin DM, Whelan SL, Ferlay J, eds. Cancer incidence in five continents, Vol. VII (IARC Scientific Publication No. 143). Lyons: International Agency for Research on Cancer, 1997.
6. Oettle GJ, Segal I. Bowel function in an urban Black African population. Dis Colon Rectum 1985;278:717–20.
7. Veitch AM, Hassan H, Kelly P, et al. Orocaecal and colonic transit times in an urban Black African population. S Afr Med J 1995;85:921.
8. Bourne LT, Langenhoven ML, Steyn K, et al. Nutrient intake in the urban African population of the Cape Peninsula, South Africa. The Brisk study. Centre Afr J Med 1993;39:238–47.
9. Heneker T, Edwards CA, Preston T, et al. Digestion and colonic fermentation of maize porridge using a $^{13}CO_2$ breath test. Presented at South African Gastroenterology Congress, 1998 (in press).
10. Gibson PR, Mailler I, Kagelari O. Contrasting effects of butyrate on differentiation of normal and neoplastic colonic epithelial cells in vitro. J Gastroenterol Hepatol 1992;7:165–72.
11. Savage DC. Microbial ecology of the gastrointestinal tract. Ann Rev Microbiol 1977;31:107–33.
12. Edwards CA, Parrett AM, Balmer SE, et al. Faecal SCFA in breast fed and formula fed babies. Acta Paediatrica 1994;83: 459–62.
13. Parrett AM, Edwards CA. In vitro fermentation of carbohydrate by breast fed and formula fed infants. Archives Dis Child 1997;76:249–53.
14. Parrett AM, Lokerse E, Edwards CA. Colonic fermentation in vitro: Development during weaning is slower for complex carbohydrates than for sugars. Am J Clin Nutr 1997;65:927–33.
15. Bornside GH. Stability of the human fecal flora. Am J Clin Nutr 1971;31:S141–4.
16. MacFarlane GT, Cummings JH. The colonic flora, fermentation and large bowel digestive function. In: Phillips SF, Pemberton JH, Shorter RG, eds. The large intestine: Physiology, pathophysiology and disease. New York: Raven Press, 1991: 51–92.
17. Armstrong EF, Eastwood MA, Edwards CA, et al. The effect of weaning diet on the subsequent colonic metabolism of dietary fiber in the adult rat. Br J Nutr 1992;68:741–51.
18. Edwards CA, McLaughlin KA, Hassan H, et al. In vitro fermentation of carbohydrate by faecal bacteria of young children in Glasgow and young urban black children in Johannesburg. Proc Nutr Soc 1996;55:239A.
19. Edwards CA, Hepburn IC, Segal I, et al. Colonic fermentation capacity in young children from South African populations of low and high cancer risk. In: Guillon F, Amado R, Amaral C, et al., eds. Functional properties of non-digestive carbohydrates. European Union Commission DGXII, 1998:222–4.
20. Caygill CPJ, Charlett A, Hill MJ. Relationship between the intake of high fiber foods and energy and the risk of cancer of the large bowel and breast. Eur J Cancer Prev 1998;7(suppl 2):S11–7.
21. Albanese D. Height, early energy intake and cancer. Br Med J 1998;317:1331–2.
22. Cummings JH, Bingham SA. Diet and the prevention of cancer. Br Med J 1998;317:1636–40.
23. Hudson GJ, Englyst HN. Dietary intakes of starch and nonstarch polysaccharides in a West African village. Br J Nutr 1995;73:655–66.
24. Englyst HN, Kingman SM. Dietary fiber and resistant starch. A nutritional classification of plant polysaccharides. In: Kritchevsky D, Bonfield C, Anderson JW, eds. Dietary fiber. New York: Plenum Press, 1990:49–65.

Reprint requests and correspondence: Isidor Segal, F.R.C.P. (UK), African Institute of Digestive Diseases, Chris Hani Baragwanath Hospital, PO Bertsham, 2013, Soweto, South Africa.
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