Fibre and Irritable Bowels

SIR,—I read with interest your leading article (1 June, p. 457) which referred to recent work on this disorder. I had just completed a survey of most of the medical journals of Africa south of the Sahara from 1960 to 1974 and had found no mention of this disease in Africans. In 1960 I had suggested as a tentative hypothesis that the rarity of non-infective colonic diseases in Africans was due to diets which contained large amounts of fibre-rich starchy carbohydrate. This may be called the "fibre hypothesis," those words being employed at that time. There appeared to be rarity of constipation, irritable bowel disease, diverticular disease, appendicitis, ulcerative colitis, haemorrhoids, polyps, and carcinoma of the large bowel and rectum.¹ The position does not seem to have changed much in the intervening 14 years. My clinical observations, based on some 30 years' medical experience in East Africa, have been considerably amplified by my Uganda colleague, Mr. Denis Burkitt, and the studies of Mr. Neil Painter. There is a possibility that irritable bowel disease often precedes diverticular disease. It is therefore of interest that we are presenting shortly much new information, necropsy, radiological, and clinical, concerning the great rarity of diverticular disease in many countries in Africa and Asia.²

At medical boards I had frequently retired European government officials for incurable irritable bowel disease, having satisfied myself that no significant and detectable tropical infection was present. In 1960 I could not remember ever having diagnosed an African as having suffered from irritable bowel disease so I suggested that a placid temperament and high-carbohydrate diets rich in dietary fibre protected them against constipation and irritable bowel disease. Doubtless many risk factors occur and the recorded death rates from diseases which are seldom fatal may be an unreliable source of evidence concerning irritable bowels or diverticular disease. Hospital admissions also for either disease are highly selective³ and should not be used by themselves to prove or disprove the relationship of dietary fibre to either disorder. It is also suggested that we produce a serious confusion of thought if we refer to the dietary fibre hypothesis as the bran hypothesis³ for many Africans do not consume cereals or bran but remain almost free of these complaints.—I am, etc.,

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1 Trowell, H. C., *Non-infectious Disease in Africa*, pp. 217-222. London, Arnold, 1960.

2 Trowell, H. C., Painter, N. S., and Burkitt, D. P., *American Journal of Gastroenterology*. In press.

3 Eastwood, M. A., et al., *Lancet*, 1974, 1, 1029.

Withdrawal of Rifamide

SIR,—A brief note appeared in the *Pharmaceutical Journal* of 5 January 1974 as follows: "Rifocin-M (Lepetit) to be discontinued from U.K. market when present stocks are exhausted." It is commonplace for pharmaceuticals to be replaced by newer agents of greater efficacy and when this occurs there can be little room for complaint. However, in this case there does

not appear to be any suitable alternative available in this country.

Rifocin-M (rifamide) is one of the rifamycin group of antibiotics which has a wide range of activity including *Mycobacterium tuberculosis*, staphylococci, and bacteroides,¹ and pharmacological properties which make it valuable in treating infections of the biliary tract.² Rifampicin is a different rifamycin which has a similar antibacterial spectrum and is widely used for treatment of tuberculosis, but it is given by mouth, has different pharmacological properties, and cannot replace rifamide. The three clinical uses of rifamide which will be lost if this drug is withdrawn are as follows.

In biliary infections the organisms mainly responsible are Gram-negative bacilli, and though rifamide shows lower activity against Gram-negative than against Gram-positive bacteria, the high levels of several thousand $\mu\text{g/ml}$ attained in bile³ with this compound make it extremely valuable in biliary infections.⁴

Resistant strains of staphylococci are common in hospital, and strains resistant to cloxacillin or fucidin or lincomycin, kanamycin, and older agents are frequently isolated. The activity of rifamide against staphylococci could prove very useful in controlling severe infections with staphylococci showing multiple antibiotic resistance. Determinations of the minimum inhibitory concentration of rifamide against 232 strains of staphylococci showed that 95% of strains were sensitive to 0.1 $\mu\text{g/ml}$ or less and only 4% were resistant to 5 $\mu\text{g/ml}$ or more. We have not used rifamide for treating staphylococcal infections but it has been held as a reserve compound.

Finally, there are the treatment problems associated with bacteroides infection, which is receiving increasing attention in medical journals. Several studies have shown that bacteroides is reliably sensitive to two antibiotics—rifamide and clindamycin.⁵ When there are contraindications to the use of clindamycin or when parenteral therapy is necessary in cases of severe bacteroides infection rifamide might be used to initiate therapy.

There are objections to the use of rifamycins because of their value in tuberculosis, but the number of patients falling into the categories outlined above is small and the amount of rifamycin used in general infections is minute compared with that used in tuberculosis, so that the contribution to the development of resistance by *M. tuberculosis* is negligible.

The withdrawal of this drug creates a gap which cannot readily be filled even though rifamide is mainly held as a reserve antibiotic. Pharmaceutical companies willingly go through detailed studies and prepare lengthy reports in order to have a drug accepted by the Committee on Safety of Medicines. I wonder if some relatively simple procedure could be fol-

lowed before drugs are removed from the market?—I am, etc.,

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- 1 Pallanza, R., et al., *Arzneimittelforschung*, 1965, 15, 800.
- 2 Stratford, B. C. and Dixon, S. *Medical Journal of Australia*, 1966, 1, 1.
- 3 Accocella, G., et al., *Gut*, 1966, 7, 380.
- 4 Bevan, P. G., and Williams, J. D., *British Medical Journal*, 1971, 3, 284.
- 5 Kislak, J. W., *Journal of Infectious Diseases*, 1972, 125, 295.
- 6 Nastro, L. J., and Finegold, S. M., *Journal of Infectious Diseases*, 1972, 126, 104.

Rapid Weight Loss in Children

SIR,—Rapid weight loss in children presents problems of a type similar to those reported by Dr. J. Runcie and Mr. T. E. Hilditch (18 May, p. 352), but lean body changes in children are highlighted by changes in height velocity. We admitted seven boys and 13 girls aged 1½-16½ years to hospital for treatment of obesity with a 350-kcal diet. Water-soluble vitamins were given and unrestricted water was allowed. Energy expenditure was largely voluntary and was uncontrolled. The weight loss in the group (table I) confirms the change in slope documented by Drs. Runcie and Hilditch. None of the following factors had any effect on the rate of weight loss: age of the child, degree or duration of obesity, or the size or number of adipose cells.

TABLE I—Mean Weight Loss ($\pm$ S.D.) Expressed as Percentage of Previous Week's Weight

Weeks in Hospital	No. of Patients	Mean Weight Loss ($\pm$ S.D.)
1	20	5.55 $\pm$ 1.68
2	19	2.87 $\pm$ 1.16
3	19	1.68 $\pm$ 1.16
4	19	2.86 $\pm$ 1.33
5	15	2.16 $\pm$ 0.74
6	14	2.67 $\pm$ 1.15
7	12	1.85 $\pm$ 1.07
8	11	1.54 $\pm$ 1.09
9	4	2.85 $\pm$ 0.52
10	4	2.10 $\pm$ 0.55
11	4	1.78 $\pm$ 0.66
12	3	2.30 $\pm$ 1.30

Body composition was measured by skin-fold thicknesses¹ in 14 of the inpatient group and in six outpatients who lost comparable amounts of weight over a much longer period on 600-800-kcal diets (table II).

Height was measured in the same children by the same observer before and after weight loss. The change in height was recorded over the periods of treatment and converted into a yearly height velocity.² This was compared with the mean expected height velocity calculated from 3-monthly measurements allowing for seasonal variations.³ The individual variations over a 3-6-month period in normal children are wide and the contribution made by measurement errors over so short a period correspondingly great, but

TABLE II—Mean Changes ($\pm$ S.D.) in Body Composition during Weight Loss

Number of Children	% Body Weight as Fat before Diet	Duration of Weight loss (Years)	% Body Weight as Fat after Diet	% Adipose Mass Lost	% Lean Body Mass Lost
14 inpatients	39.6 $\pm$ 4.2	0.21 $\pm$ 0.08	28.8 $\pm$ 4.3	41.9 $\pm$ 11.3	8.3 $\pm$ 5.6
6 outpatients	36.1 $\pm$ 5.3	0.55 $\pm$ 0.13	23.4 $\pm$ 3.3	45.8 $\pm$ 8.7	2.5 $\pm$ 1.9
P	N.S.	<0.001	<0.02	N.S.	<0.05