the index is incomplete, and at present it probably provides only an underestimate of opiate use. One way in which the index might be modified to reflect more accurately the prevalence of all the serious forms of drug addiction would be to extend compulsory notification to include all self-injectors. Although this might have improved the accuracy of the index at the time of our survey (over 80% of addicts had injection marks), there is evidence that self-injection is becoming less common.[14] This continuous, rapid change in the pattern of drug-taking behaviour makes any rigid system of notification, particularly one dependent on doctor-participation, unlikely to succeed in isolation as an epidemiological monitor, and it is in this context that the value of a survey such as ours in coroners' courts can be most easily appreciated. Repeated surveys not only in coroners' courts but in prisons, clinics, and accident and emergency departments would be a much more sensitive way to monitor drug misuse and its morbidity and mortality.

Nevertheless, a central, integrated record of all notifications and of the results of different surveys is clearly of value, and the Home Office index can fulfil this role. Moreover, even in the present study the figures suggest that some deaths of addicts in London were known to the Home Office but had not been notified to a coroner, perhaps because the addict was not taking drugs at the time of death. Again, this emphasises that no one technique is ever likely to provide all the answers and that complementary research methods are essential.

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Guar crispbread in the diabetic diet

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Summary and conclusions

Nine diabetic patients who were receiving various treatments supplemented their normal home diets (two patients) or metabolic ward diets (seven patients) with guar crispbread for five days. Their mean urinary glucose excretion fell significantly by 38% during the last two days. A significant fall in fasting blood glucose concentration of 1·1±0·4 mmol/l (19·8±7·2 mg/100 ml) was seen only in those who took guar after the control period. Over eight weeks' treatment insulin dosage was reduced by 21% in five patients, and home testing showed that glycosuria was reduced by 68% in six patients.

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Guar crispbread is likely to be a useful adjunct to diabetic treatment irrespective of the type of treatment or insulin dosage used.

Introduction

Certain forms of dietary fibre (unabsorbable plant polysaccharides) have been used successfully to treat diabetes[1-3] and have permitted withdrawal of insulin or reduction of the dose in patients whose original dose was low (<30 U/day).[1] Comparison of six unabsorbable carbohydrates or synthetic fibres showed guar to be the most effective in reducing postprandial glycaemia.[4] Its high viscosity, however, precluded its use in long-term diabetic treatment until the recent development of a palatable guar crispbread. We have therefore looked at the effect on total urinary glucose output and fasting blood glucose concentrations of incorporating guar crispbread into both the metabolic ward and home-based diets of diabetics.

Patients and methods

We studied nine diabetics (four men and five women; see table I). After a preliminary three to four days in hospital seven of the patients began two five-day metabolic study periods, the order of which was randomised such that guar was added to the diets of three patients in the first period and to the diets of the remaining four in the last. During the preliminary days in hospital we reduced the patients' normal daily insulin dosages by a mean of 8 U (table I) and increased carbohydrate intake by 40-50 g daily to prevent hypoglycaemic episodes.[2,3] In five cases the studies ran from Monday to Saturday morning, patients being replaced on their outpatient treatment at

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weekends. In both the remaining cases the two five-day periods ran consecutively, starting with a control period. The home-based studies (two patients) also consisted of two five-day periods (Monday to Saturday) extending over two consecutive weeks, guar being taken during the first week in one case and during the second in the other.

We used two-day rotating menus for the guar and control metabolic ward diets, which were identical except that 14-26 slices of guar crispbread containing 1 g guar per slice were substituted for 14-26 control slices (see table I). In the two home-based studies a five-day record of weighed intakes was made during the first period and the exact daily menu repeated during the second,[2] only the crispbread being changed. Each patient ate up to 26 crispbreads (depending on his ability) during the early part of each meal as divided doses throughout the day—for example, at breakfast, six; mid-morning, two; at lunch, six; at tea, two; at dinner, six; and at supper, three.

During the study periods in all nine patients we made complete 24-h urine collections (7 am to 7 am) for glucose analysis[2 5] into 2-l plastic bottles containing 10 ml concentrated HCl as a preservative. Blood samples for glucose estimation[6] were taken in the morning after overnight fasts at the beginning and end of each five-day period.

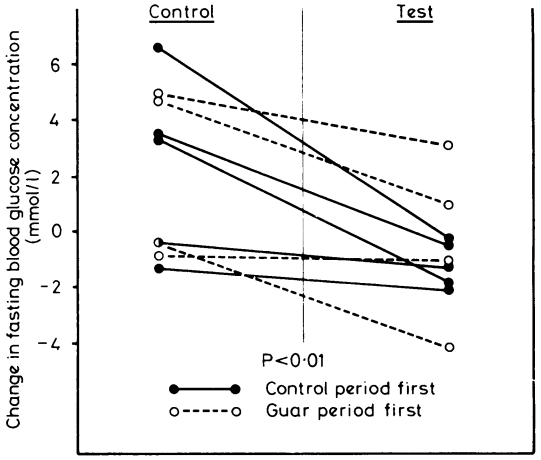
We obtained limited follow-up data on patients who continued to take guar (see table II) and so could compare the most recent results with those obtained before the patients began the trial.

Results are expressed as means ± SE of mean, and the significance of differences was calculated using Student's *t* test for paired data.

Results

Urinary glucose loss—The mean five-day urinary glucose output of all nine patients was 538 ± 128 mmol/24 h (97 ± 23 g/24 h). This fell on taking 23 g guar daily to 344 ± 61 mmol/24 h (62 ± 11 g/24 h), which represents a mean reduction of 27 ± 10% ($P < 0.025$; table I). The mean percentage changes in urinary glucose outputs for each day of guar treatment were: day 1, +3·9 ± 16·0% (not significant (NS)); day 2, −18·2 ± 12·9% (NS); day 3, −23·9 ± 20·3% (NS); day 4, −42·7 ± 10·7% ($P < 0.01$); and day 5, −28·3 ± 6·8% ($P < 0.01$), giving an overall reduction in urinary glucose loss over the last two days of 38 ± 8% ($P < 0.002$; table I). Because the percentage falls while patients were taking guar did not reach significance until day 4 the results of the last two study days for each patient are more likely to be representative of the longer-term effect of guar and are shown in table I.

Fasting blood glucose concentrations—The mean blood glucose concentration in all nine patients before the control period was 13·3 ± 1·4 mmol/l (240 ± 25 mg/100 ml), which rose during this period by 2·2 ± 1·0 mmol/l (40 ± 18 mg/100 ml). When patients took guar a



Individual changes in fasting blood glucose concentrations over control and test periods.

Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml.

TABLE I—*Results of 10-day studies*

Case No:	1*	2	3	4	5	6*†	7†	8†	9†	Mean ± SE of mean
Details of patients										
Age	44	32	66	26	37	42	33	64	34	42 ± 5
Sex	F	F	F	M	M	M	F	M	F	
Duration of diabetes (years)	6	1·5	44	3	21	20	12	10	22	16 ± 4
% desirable weight	105	97	96	114	93	101	144	98	105	106 ± 5
Blood glucose (mmol/l)‡	12·3	9·0	16·9	19·0	5·4	18·8	15·3	8·8	18·7	13·8 ± 1·7
Daily treatment before study	C, 750 mg	G, 25 mg	I, 26 U	I, 58 U	I, 52 U	I, 36 U	I, 56 U	I, 40 U	I, 36 U	43 ± 6 U§
Daily treatment during study	C, 750 mg	G, 25 mg	I, 20 U	I, 52 U	I, 48 U	I, 36 U	I, 32 U	I, 32 U	I, 32 U	36 ± 6 U§
Daily dietary intakes										
No of crispbreads	20	14	22	25	25	26	25	25	25	23 ± 3
Fat (%)	45	39	21	46	35	10	43	40	37	34 ± 4
Protein (%)	29	27	24	18	17	29	24	21	21	23 ± 1
Available carbohydrate (%)	26	30	48	37	48	61	33	34	38	41 ± 4
Energy (kcal)‖	1685	1225¶	2025¶	3425	3715	1730	1345	2305¶	2350¶	2190 ± 275
Urinary glucose outputs										
During control period** (mmol/24 h)	799	289	178	688	1681	477	394	327	433	583 ± 150 } P < 0·002
During test period** (mmol/24 h)	627	105	111	372	477	189	289	266	450	322 ± 61 }

C = Chlorpropamide. G = Glibenclamide. I = Insulin.
*Home study. †Guar treatment first. ‡Fasting value measured on admission. §Insulin doses only. ‖1000 kcal ≈ 4·18 MJ. ¶Excess energy measured as sorbitol. **Mean of values in last two days.
Conversion: SI to traditional units—Blood glucose: 1 mmol/l ≈ 18 mg/100 ml. Urinary glucose: 1 mmol/24 h ≈ 180 mg/24 h.

TABLE II—*Effect of long-term administration of guar on insulin dosage and mean urinary glucose concentrations, and duration of administration*

Case No	Before taking guar			After taking guar			Duration of treatment (weeks)
	Insulin (U/24 h)	Weight (kg)	Mean urinary glucose (mmol/l)*	Insulin (U/24 h)	Weight (kg)	Mean urinary glucose (mmol/l)*	
2		43	83·2		42·7	5·5	8
3	26	52	16·6	20	51·7	0	5
4	58	75·8	27·7	48	76·0	22·2	7
5	52	70	27·7	34	71·3	16·6	11
6	36	70	111·0	36	70·4	27·7	8
7	56	76	111·0	40	77·1	22·2	8
Mean ± SE of mean	46 ± 6	64·5 ± 5·6	61·0 ± 16·6	36 ± 5	64·9 ± 5·8	16·6 ± 5·5	7·8 ± 0·8
P†				< 0·05		< 0·05	

*Mean of fasting, pre-lunch, pre-dinner, and pre-bed urine tests.
†Significance of mean absolute difference from control values.
Conversion: SI to traditional units—Urinary glucose: 1 mmol/l ≈ 0·018 g/100 ml.

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fall of 0·8±0·7 mmol/l (14±13 mg/100 ml) occurred from a pre-guar mean of 14·7±1·3 mmol/l (265±23 mg/100 ml). Although these changes were not significant, the five patients who took guar after the control period showed a significant mean fall of 1·1±0·4 mmol/l (20±7 mg/100 ml) in fasting blood glucose concentrations ($P < 0·05$), with a corresponding rise over the control periods of 2·3±1·4 mmol/l (41±25 mg/100 ml). The figure shows the individual changes over the control and guar periods. A significant relative reduction in fasting blood glucose concentrations of 3·1±0·7 mmol/l (56±13 mg/100 ml) ($P < 0·01$) was seen when the results were expressed as the difference between changes in blood glucose concentrations over control and guar periods.

Long-term effects—Table II shows the individual results in patients who continued to take guar for a mean of eight weeks after discharge from hospital. The mean insulin dosage was reduced from 46 U by 21±6% ($P < 0·05$), and the mean daily urinary glucose concentration was reduced from 62·7 to 18·3 mmol/l (1·13 to 0·33 g/100 ml)—that is, by 68±13% ($P < 0·01$). There was no change in body weight.

TABLE III—*Mean (±SE of mean) daily urinary glucose outputs during control periods before (five cases) and after (four cases) patients took guar expressed as percentage of output on day 5*

Day:	1	2	3	4	5
Pre-guar control	109±14	112±9	113±15	121±18	100
Post-guar control	69±15	80±11	101±8	88±3	100

Discussion

Adding guar in crispbread form to the diets of diabetics reduced the urinary glucose loss and resulted in a relative fall in fasting blood glucose concentrations. Guar gum is derived from the cluster bean and is a galactomannan, a major dietary fibre constituent of certain leguminous seeds. Because of its viscous nature guar is widely used in low concentration in the food industry as a thickener and stabiliser of emulsions. It noticeably reduces postprandial glycaemia in normal[6] and diabetic volunteers[7] and reduces urinary glucose loss in diabetics.[2] Nevertheless, its practical application in the treatment of diabetes has been limited by the difficulty of making a therapeutic dose edible. The first palatable guar formulation therefore represents an important advance in food technology.

The overall five-day urinary glucose loss decreased by 27% when patients took guar crispbread; this was further reduced to 38% during the last two days of the study. The only other workers who carried out a study of this type and reported sequential measurements in diabetics placed on high-fibre diets also noted the progressive nature of the effect,[1] with the insulin dosage being reduced and finally withdrawn in four out of five diabetics needing less than 28 U/day. In our follow-up studies we found that after eight weeks' guar treatment the insulin dosage had been reduced by 21%, while urinary glucose concentrations had fallen by 68%, as judged by home testing.

The fibre used in the studies of Kiehm *et al*[1] was derived largely from wheat. Comparison of the effects of various fibres and fibre analogues in flattening the peak rise in glucose concentration when added to 50 g glucose loads showed guar to be 230% more effective than wheat bran.[4] Much of the effect of the fibre in the cereal-based study may therefore have been due to an interaction of the fibre with the increased carbohydrate and decreased fat contents of the unrefined foods used. It is interesting that raw-vegetable high-fibre (vegan-type) diets have permitted withdrawal of insulin from diabetics,[8] while work on whole and fibre-depleted foods (potatoes and apples) has further emphasised the importance of food form in modifying postprandial glycaemia[9] and insulin response.[10] Supplementing diabetic diets with 17 g crude fibre (largely as cellulose) also reduced mean plasma glucose concentrations throughout the day.[3] To prevent hypoglycaemic episodes[2 3] we reduced the insulin and increased the carbohydrate intakes of all patients during their preliminary period in hospital. In this way control blood glucose concentrations were raised and urinary glucose outputs increased sufficiently for hypoglycaemic episodes not to occur when patients were taking guar. This may have resulted in higher blood glucose concentrations during the first five-day metabolic period and be the reason why significant falls of fasting blood glucose concentrations were seen only when guar was taken in the second five-day period.

The mechanism of action of guar is probably related to its ability to increase the viscosity of gastrointestinal contents, slow gastric emptying, and act as a barrier to diffusion by increasing the width of the unstirred layer. There is no evidence, however, that it causes malabsorption of carbohydrate,[6] though an increased loss of fat and bile salts in the stool has been reported.[11] No reduction in blood concentrations of the metal ions Ca^{++}, Mg^{++}, and Fe^{++} was seen over two weeks during which patients took 36 g guar daily.[12]

Our results also show an important "carry-over" phenomenon associated with guar treatment. Thus the mean daily urinary glucose outputs in the pre-guar control period (five patients) showed no trend, while in the post-guar control period (four patients) the mean over the first two days (322±33 mmol/24 h (58±6 g/24 h)) was significantly lower than that over the last three days (416±33 mmol/24 h (75±6 g/24 h); $P < 0·02$; table III). The only patient (case 9) who appeared to be unresponsive to guar in our studies had already been taking it for three months before her five-day control period. Although in previous studies of the effects of guar flatulence has sometimes been a problem, this was minimal with the guar in crispbread.

We conclude that guar crispbread has a useful part to play in the dietary treatment of diabetes, irrespective of the type of hypoglycaemic regimen.

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