

Sucrose or honey at breakfast have no additional acute hyperglycaemic effect over an isoglucidic amount of bread in Type 2 diabetic patients

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Summary. Exclusion of simple sugars from the diabetic diet is not always followed by patients and may not even be as crucial as was hitherto thought. We tested three types of mixed breakfasts (400 kcal, 50 g HCO) including an isoglucidic amount either of white bread (30 g), honey (20 g) or sucrose (15 g), at the critical morning period i.e. for breakfast, in a group of 21 Type 2 (non-insulin-dependent) diabetic patients (6 well- and 15 badly controlled). Mean plasma glucose and insulin levels were comparable on the three occasions: respectively with bread, sucrose and honey, peak glucose values were 18 mmol/l, 17.7 mmol/l and 17.5 mmol/l in the uncontrolled group versus 13.9 mmol/l, 12.8 mmol/l and 12.7 mmol/l in the well-controlled group. Peak insulin values were 33.6 mU/l, 34.0 mU/l and 36.3 mU/l (p>0.05) in uncontrolled patients against 57.5 mU/l, 54.8 mU/l and 52.5 mU/l

in well-controlled subjects (p>0.05). The mean increment in peak plasma glucose values for the three breakfasts was as follows: 6.9 mmol/l, 6.3 mmol/l and 6.2 mmol/l for the uncontrolled group against 7.2 mmol/l, 5.9 mmol/l and 6.2 mmol/l in well-controlled subjects; the mean increment in peak plasma insulin levels was 21.8 mU/l, 22.0 mU/l and 24.2 mU/l in the controlled group versus 38.2 mU/l, 32.0 mU/l and 34.7 mU/l in the well-controlled subjects, all values being non-significantly different (p>0.05). We conclude that, in acute conditions, simple sugars have no additional hypergly-caemic effect over an isoglucidic amount of bread in well- and in badly controlled Type 2 diabetic patients, even at breakfast.

Key words: Sucrose; honey, simple sugar, diabetic diet, Type 2 diabetes, plasma glucose, plasma insulin.

Exclusion of simple sugars from the diabetic diet is recommended by almost all international diabetic associations [1-3]. This recommendation is known – but to a lesser extent followed – by most diabetic patients [4] and is based on the hypothesis that mono- and disaccharides induce faster and higher blood glucose levels than starch. However, several studies, some over a decade old, have shown that certain sugars, even when eaten alone are less hyperglycaemic than some starch-containing foods in normal subjects [5, 6] and in diabetic patients [7-11]. We have recently shown that 20 g sucrose at the end of a regular mixed meal at lunch had no additional acute hyperglycaemic effect over an isocaloric amount of starch in well controlled Types 1 and 2 diabetic patients [12].

We decided to evaluate the acute metabolic effects of two current simple sugars in Type 2 diabetics at a critical period of the day, i.e. in the morning [13], in both uncontrolled and well-controlled Type 2 diabetics.

Subjects and methods

Twenty-one Type 2 diabetic patients were studied whose characteristics are given in Table 1. Fifteen (group A) were hospitalized to improve bad metabolic control [fasting and 2-h post prandial plasma

glucose levels for the 3 days of experimentation were above 8 mmol/l, and mean (\pm SEM) HbA_{1c} 9.7 \pm 0.5%] (normal range: 4 to 6%). Six others, carefully matched for age, BMI and diabetes duration, and considered as reasonably well-controlled (group B) (fasting and 2-h post prandial lunchtime plasma glucose values below 8 mmol/l for the 3 days of experimentation; mean HbA1c 7.1 \pm 0.4%) accepted a short hospitalization for the study. Body weight, diet, physical activity and therapy were stable for at least 1 week before testing, their usual therapy being continued throughout the experiment and taken before the test meal as usual where indicated.

All gave informed consent to participate in the study (approved by the Hospital Ethical Committee).

Each patient, serving as his or her own control, was tested on 3 consecutive days, in a random order permitting a Latin square study. At 08.00 hours after an overnight fast, patients ate a mixed breakfast of 70 g white bread, 10 g butter, 30 g camembert cheese, 250 ml of decaffeinated coffee and either an additional 30 g white bread, 15 g sucrose of 20 g honey. Each breakfast (400 kcal) thus contained 50 g carbohydrate, 16 g fat and 12 g protein according to reference tables [14]. The composition of the three breakfasts is summarized in Table 2. Each breakfast was taken in 10 min. Blood samples were drawn from an indwelling catheter every 15 min between -30 and +60 min after the beginning of the meal and then every 30 min until 180 min after the start of the meal. Samples were immediately centrifuged and plasma frozen at 18 °C for ultimate assay. Plasma glucose was assayed using a glucose oxidase method (Beckman Autoanalyzer II, Beckman instr., Fullerton, USA) and insulin using a radio-immuno assay with charcoal separation. All samples were assayed in the same batch. Insulin intra assay reproducibility was 6%. HbA1c was assayed according to Trirelli.

Table 1. Clinical characteristics of the patients

Subjects	Sex	Age (years)	BMI (Kg/m²)	Diabetes	HBA _{1c} f	Fasting plasma	Antidiabetic drugs	
				duration (years)	(%)	glucose ^c (mmol/l)	Glibenclamide (mg/day)	Metformin (mg/day)
Group A ^a								
1. GUE	M	53	29.4	10	14.5	13.9 ± 0.2	0	0
2. COU	M	57	24.1	20	13.0	11.3 ± 0.4	15	1700
3. JER	M	57	30.5	17	7.8	11.6 ± 0.4	0	1700
4. MAZ	M	49	27.1	2	8.1	8.3 ± 0.2	0	0
5. ROU	F	41	32.7	2	7.6	10.3 ± 0.1	0	1700
6. LAJ	M	59	19.6	10	8.9	10.1 ± 0.3	15	0
7. MOK	M	50	24.3	13	9.4	12.3 ± 0.6	15	1700
8. MER	M	65	26.5	20	9.9	12.7 ± 0.9	15	1700
9. BEN	M	38	24.4	8	7.1	9.7 ± 1.2	15	1700
10. AUD	M	33	28.4	2	7.5	11.4 ± 0.5	0	0
1. KRA	M	33	29.9	0.75	8.3	11.9 ± 0.8	0	0
12. COE	F	51	38.9	5	10.2	8.2 ± 0.2	0	0
13. MAI	F	67	26.4	10	10.9	14.6 ± 0.5	0	0
14. AUB	M	67	20.8	7	11.7	14.9 ± 0.5	7.5	0
15. VAN	M	67	20.9	25	10.2	13.1 ± 0.8	15	1700
Mean ± SEM	M/F 12/3	52.5 ± 3.1	27.2 ± 1.4	10.1 ± 1.9	9.7 ± 0.5	11.3 ± 0.4 ^d		_
Group B ^b								
1. NAD	M	63	31.70	12	7.5	6.6 ± 0.0	0	0
2. NAN	M	38	22.50	0.20	7.3	7.1 ± 0.2	0	0
3. THO	F	56	24.60	14	8.3	7.5 ± 0.2	15	1700
1. VAH	M	62	30.10	12	7.1	7.2 ± 0.2	0	850
5. LEC	M	60	28.30	3	6.2	6.2 ± 0.2	15	0
5. BAS	M	62	21.80	20	6.2	5.3 ± 0.6	6.25	1700
Mean ± SEM p value (AvsB) ^e	5/1	56.8±3.9 NS	26.5 ± 1.7 NS	10.0 ± 2.0 NS	7.1 ± 0.4 p < 0.05	6.5 ± 0.2 $p < 0.001^{d}$		

^a Group A: 15 uncontrolled subjects at the time of experiment; ^b Group B: 6 well controlled subjects; ^c mean \pm SEM calculated from the 3 fasting values on the 3 days of experimentation; ^d mean \pm SEM calculated from the 45 (Group A) or 18 (Group B) fasting values; ^e p= statistical significance (Student's t test) NS = non significant (p> 0.05); ^f normal range 4-6%

Table 2. Composition of the three breakfasts

Type of	Carbohydrate (g)				Protein (g)	Energy (kcal)
breakfasts	Polysaccharides	Disaccharides	Monosaccharides			
Bread						
White bread 100 g	51	-	_	1.15	8.20	247
Cheese 30 g	_	0.55^{a}	-	6.85	5.60	86
Butter 10 g	_	0.05^{a}		8.10	0.05	_73
		51.6		16.10	13.85	406
Sucrose						
White bread 70 g	35.5	_	-	0.85	5.75	173
Sucrose 15 g	_	15 ^b	_	_	-	60
Cheese 30 g	-	0.55^{a}	_	6.85	5.60	86
Butter 10 g	. –	_0.05a	= ,	8.10	0.05	$\frac{73}{392}$
24.00 10 8		51.1		15.80	11.40	392
Honey						
White bread 70 g	35.5	-	_	0.85	5.75	173
Honey 20 g	_		16.60°	_	0.05	67
Cheese 30 g	_	0.55a	_	6.85	5.60	86
Butter 10 g	_	0.05^{a}	_	8.10	0.05	73
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^a Lactose; ^b saccharose; ^c fructose 53% – glucose 47%

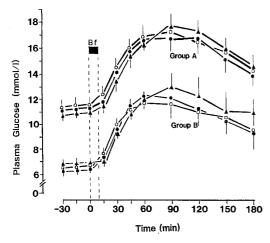


Fig. 1. Mean plasma glucose profiles in 15 uncontrolled (group A) and 6 well-controlled (group B) Type 2 diabetic patients receiving at breakfast (Bf) either 20 g honey (●—●), 15 g sucrose (○—○), an extra 30 g white bread (▲——▲). Bars indicate 1 SEM

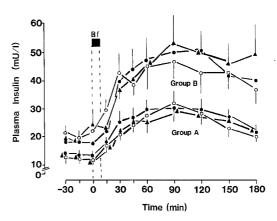


Fig. 2. Mean plasma insulin profiles in 15 uncontrolled (group A) and 6 well-controlled (group B) Type 2 diabetic patients receiving at breakfast (Bf) either 20 g honey (●—●), 15 g sucrose (○—○), an extra 30 g white bread (▲——▲)

Table 3. Characteristics of mean plasma glucose variations within 180 min of start of breakfast

	a	Bread	Sucrose	Honey	Significance ^b
Fasting plasma glucose (mmol/l)	Group A Group B Significance ^c	$ \begin{array}{ccc} 11.2 \pm & 0.6 \\ 6.7 \pm & 0.3 \\ p < 0.001 \end{array} $	$ \begin{array}{ccc} 11.4 \pm & 0.6 \\ 6.5 \pm & 0.5 \\ p < 0.001 \end{array} $	$ \begin{array}{ccc} 11.3 \pm & 0.7 \\ 6.4 \pm & 0.5 \\ p < 0.001 \end{array} $	NS ^d NS
Peak plasma glucose (mmol/l)	Group A Group B Significance	18.0 ± 0.8 13.9 ± 0.9 $p < 0.01$	$ \begin{array}{rrr} 17.7 \pm & 0.9 \\ 12.8 \pm & 0.8 \\ p < 0.005 \end{array} $	$ \begin{array}{rrr} 17.5 \pm & 0.8 \\ 12.7 \pm & 0.9 \\ p < 0.001 \end{array} $	NS NS
Δ Peak plasma glucose (mmol/l)	Group A Group B Significance	6.9 ± 0.7 7.2 ± 0.9 NS	6.3 ± 0.6 5.9 ± 0.7 NS	6.2 ± 0.3 6.2 ± 0.9 NS	NS NS
Peaking time (min)	Group A Group B Significance	86 ± 6 95 ± 12 NS	91 ± 9 75 ± 16 NS	82 ± 7 77 ± 11 NS	NS NS
Areas increment above starting value $(\text{mmol} \cdot l^{-1} \cdot \text{min}^{-1})$	Group A Group B Significance	807.4 ± 101.2 796.3 ± 160.2 NS	740.0 ± 83.4 755.1 ± 176.2 NS	705.7 ± 83.0 730.7 ± 158.0 NS	NS NS

^a Group A: n=15; badly controlled diabetics; Group B: n=6; well controlled diabetics; ^b significance: analysis of variance; ^c significance: Student's t test for unpaired data; ^d NS=non significant (p>0.05)

Table 4. Characteristics of mean insulin plasma variations within 180 min of start of breakfast

	a	Bread	Sucrose	Honey	Significance ^b
Fasting insulin levels (mU/l)	Group A Group B Significance ^c	$ \begin{array}{ccc} 11.9 \pm & 2.0 \\ 24.2 \pm & 2.0 \\ p < 0.005 \end{array} $	$ \begin{array}{rrr} 11.8 \pm & 1.0 \\ 21.5 \pm & 4.0 \\ p < 0.005 \end{array} $	12.7 ± 2.0 17.2 ± 4.0 NS	NS ^d NS
Peak insulin values (mU/l)	Group A Group B Significance	33.6 ± 4.0 57.5 ± 10.1 p < 0.02	34.0 ± 4.5 54.8 ± 6.4 p = 0.02	36.3 ± 4.4 52.5 ± 6.3 NS	NS NS
△ Peak values (mU/l)	Group A Group B Significance	21.8 ± 3.0 38.2 ± 7.0 $p < 0.02$	22.0 ± 3.0 36.0 ± 5.0 p < 0.02	24.2 ± 3.0 34.7 ± 3.0 NS	NS NS
Peaking time (min)	Group A Group B Significance	99 ± 10 105 ± 17 NS	94 ± 9 80 ± 16 NS	86 ± 13 95 ± 18 NS	NS NS
Areas increment above starting value $(mU \cdot l^{-1} \cdot min^{-1})$	Group A Group B Significance	2324 ± 388 4655 ± 829 $p < 0.005$	2313 ±355 4087 ±404 p<0.02	2306 ± 397 4585 ± 447 $p < 0.005$	NS NS

^a Group A: n=15; badly controlled diabetics; Group B: n=6; well controlled diabetics; ^b significance: analysis of variance; ^c significance: Student's t test for unpaired data; ^d NS=non significant (p>0.05)

Statistical analysis used the analysis of variance with three factors (subjects, breakfast content and order) and the Student's test for unpaired data as indicated. Results are given as mean \pm standard error of the mean (m \pm SEM).

Results

Figure 1 and Table 3 give the mean plasma glucose profiles after each breakfast containing a supplement of white bread, sucrose or honey in the 15 uncontrolled Type 2 diabetics (group A) and in the 6 well-controlled patients (group B).

In group A mean starting fasting plasma glucose values were comparable before the extra white bread, sucrose and honey breakfasts, i.e. respectively: 11.2 ± $0.6 \,\mathrm{mmol/l} \,(201 \pm 12 \,\mathrm{mg/100 \,ml}); \,11.4 \pm 0.6 \,\mathrm{mmol/l}$ $(206 \pm 12 \text{ mg}/100 \text{ ml})$; $11.3 \pm 0.7 \text{ mmol/l}$ $(204 \pm 13 \text{ mg/ms})$ 100 ml) (m \pm SEM; p > 0.05). The mean peak valwere respectively $18.0 \pm 0.8 \, \text{mmol/l}$ $(325 \pm$ $15 \, \text{mg} / 100 \, \text{ml}$); $17.7 \pm 0.9 \, \text{mmol/l}$ $(319 \pm 16 \,\mathrm{mg}/$ 100 ml); 17.5 ± 0.8 mmol/1 (315 ± 15), p > 0.05 and the mean areas of increment above starting values were 807.4 ± 101.2 , 740.0 ± 83.4 and 705.7 ± 83.0 mmol $\times 1^{-1} \times \text{min}$, F=0.89, NS. Peak times were also comparable after the three breakfasts respectively: 86 ± 6 ; 91 ± 9 and 82 ± 7 min (p > 0.05).

Concerning plasma insulin levels (Table 4), we were there again unable to discover any significant difference between the three breakfasts, i. e. respectively and in the same order (bread, sucrose, and honey) for fasting insulin levels: $11.9\pm2.0\,\mathrm{mU/l}$; $11.8\pm1.0\,\mathrm{mU/l}$; $12.7\pm2.0\,\mathrm{mU/l}$ (F=0.99, p>0.05); likewise mean peak insulin values were $33.6\pm4.0\,\mathrm{mU/l}$; $34.0\pm4.5\,\mathrm{mU/l}$; $36.3\pm4.4\,\mathrm{mU/l}$ (NS) and mean areas of insulin increment above starting values were 2324 ± 388 , 2313 ± 355 and $2306\pm397\,\mathrm{mU}\times1^{-1}\times\mathrm{min}$ (F=0.41, NS). Peaks ocurred again at comparable times in the three groups: 99 ± 10 , 94 ± 9 and $86\pm13\,\mathrm{min}$ (NS).

In group B results were similar. Mean fasting (starting) plasma glucose values were also comparable in the three situations: 6.7 ± 0.3 mmol/l (120 ± 5 mg/100 ml) with the white bread supplement; 6.5 ± 0.5 mmol/l (117 ± 9 mg/100 ml) with sucrose; 6.4 ± 0.5 mmol/l (115 ± 9 mg/100 ml) with honey (F=0.73, NS). Mean peaking values were 13.9 ± 0.9 mmol/l (250 ± 16 mg/100 ml); 12.8 ± 0.8 mmol/l (230 ± 14 mg/100 ml); 12.7 ± 0.9 mmol/l (238 ± 16 mg/100 ml) (NS).

Mean areas of glucose increment above starting levels were 796.3 ± 160.2 , 755.1 ± 176.2 and $730.7 \pm 158.0 \,\mathrm{mmol} \times 1^{-1} \times \mathrm{min}$ (F=1.46; NS). Peaking time was respectively 95 ± 12 , 75 ± 16 and $77 \pm 11 \,\mathrm{min}$ (NS).

As for plasma insulin levels, fasting levels were $24.2 \pm 2.0 \text{ mU/l}$; $21.5 \pm 4.0 \text{ mU/l}$; $17.2 \pm 4.0 \text{ mU/l}$ (F= 0.45; NS), peak insulin levels reached $57.5 \pm 10.1 \text{ mU/l}$; $54.8 \pm 6.4 \text{ mU/l}$; $52.5 \pm 6.3 \text{ mU/l}$ (NS) and mean areas of insulin increment above starting values were respectively 4655 ± 829 ; 4087 ± 404 ; $4585 \pm 447 \text{ mU} \times 1^{-1}$

 \times min (F=0.58; NS). Lastly, peaking time was: 105 ± 17 min, 80 ± 6 min and 95 ± 18 min (NS).

Discussion

Contrarily to what might have been expected but consistent with what we have already found in Type 1 and in Type 2 diabetic patients [12], exchanging an isoglucidic isocaloric amount of bread for sugar (sucrose) or honey (glucose and fructose) has no additional hyperglycaemic effect in Type 2 diabetics whether well- or uncontrolled.

Indeed plasma glucose curves were almost perfectly superimposed in each sub-group of patients (group A: poorly controlled, group B: well controlled subjects).

If we consider the increment above starting plasma glucose values, we can see that this increment is comparable for each type of breakfast in the two subgroups of patients (Table 2) which means that the hyperglycaemic effect of a meal (i. e. the rise above pre-meal value) seems not to correlate to the degree of diabetic control.

This might be due in part to a greater renal excretion of glucose in the badly controlled subjects where the renal threshold is probably often attained [15] (though this hypothesis has not be verified in these patients), and in particular to a continuous and excessive endogenous production of glucose in greatly insulin-deficient diabetic patients which is not suppressed or altered by meal intake as shown by Pehling et al. [16].

If we now consider plasma insulin fluctuations, we can see that, as expected, insulin secretion is much lower in the uncontrolled diabetics, and their mean increment of insulin secretion above starting values was also significantly lower.

Our study confirms that the breakfast period is the worst one for blood glucose regulation since post-breakfast plasma glucose was above the 2-h post-mid-day meal value (which is the most copious meal in our country), both in the badly and in the "well"-controlled subjects even after relatively light breakfasts such as these

We conclude that, in acute conditions, substitution of part of a mixed meal by a reasonable amount of simple, currently available sugars has no deleterious effect on blood glucose regulation and insulin secretion in Type 2 diabetic patients. To what extent such sugars could be allowed in Type 2 diabetic patients, very often obese, is to be discussed cautiously. They could at least be put to the test when negotiating with the patients a better commitment to the compulsory hypocaloric diet. The validity of these conclusions now have to be confirmed on long-term follow-up studies.

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