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MODULATION OF POSTPRANDIAL GLYCAEMIA AND INSULINAEMIA BY DIETARY FAT

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Abstract : The present study was designed to examine the effect of corn oil (Co) on postprandial glycaemia and insulinaemia when ingested with glucose (G), casein (Cs), cellulose (Cl) and pectin (P) in various combinations. The study was conducted on six healthy male volunteers, on each of whom six meal tolerance tests were performed. The meals were isocaloric and consisted of G; G and Co; G, Co and Cs; G, Co and P; G, Co, Cs and P; and G, Co, Cs and Cl. The meals were administered after an overnight fast. In addition to a fasting blood sample, blood was collected 0.5, 1.0, 1.5 and 2.0 h after ingestion for measurement of serum glucose and insulin levels. The glycaemic response to GCo was comparable to that to G, but the insulinaemic response was significantly lower. The glycaemic response to GCoCs was significantly lower than that to G but the insulinaemic response to both was comparable. The cellulose containing meal GCoCsCl showed a further reduction in the glycaemic response but not in the insulinaemic response. The pectin containing meals GCoP and GCoCsP gave the lowest glycaemic and insulinaemic responses, the responses to the latter being lower. Corn oil by itself has only a modest effect on the postprandial metabolic response to glucose. Addition of protein and fibre, specially pectin, leads to significant attenuation of glycaemic and insulinaemic responses.

Key words : glucose tolerance test glycaemic index postprandial glycaemia corn oil dietary fibre cellulose pectin

INTRODUCTION

Dietary fat slows gastric emptying (1) and at least some varieties of fat stimulate GIP secretion (2, 3, 4, 5). These effects of fat suggest that its coingestion might affect postprandial glycaemia. While the slowing of gastric emptying would reduce the rate of delivery of carbohydrate to the small intestine, enhanced GIP secretion would stimulate insulin secretion. Both these consequnces are likely to attenuate the glycaemic response. Coingestion of fat in a carbohydrate meal has, in fact, been shown to result in attenuation of postprandial glycaemia (6, 7, 8, 9). The glycaemic response is further blunted if both protein and fat are added to the carbohydrate meal (6, 10, 11). Dietary fibre, specially its water-soluble viscous components, are also known to attenuate the glycaemic reponse (8, 12, 13, 14) and retain this effect when given in combination with a mixed meal (15, 16). But the precise nutrient interaction on successive addition of fat, protein and fibre to a carbohydrate is not known. It is also not known whether the tendency of each of these nutrients to attenuate the glycaemic response is mathematically additive. The present study was designed to study the effect of fat on the glycaemic response to glucose, the effect of further addition of protein and fibre, and the quantitative contribution of each individual nutrient to alteration in the response, if any.

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METHODS

Subjects : The study was conducted on six healthy young male human volunteers (age, 19-21 years; body weight, 47-69 kg; height 165-176.5 cm). The subjects were on their usual diet which provided at least 250 g carbohydrate every day. They were expected to abstain from late night snacks, smoking, and alcohol on the day preceding the meal tolerance test (MTT).

Ethical considerations : The protocol of the study was apporved by the Ethics Committee of the All India Institute of Medical Sciences, New Delhi. An informed written consent was obtained prior to enlisting a subject for the study.

Meals : Each subject received six different isocaloric meals (Table I). The 100 g oral glucose tolerance test (OGTT) was performed for comparison with other meals . Except for this reference meal, all other meals had glucose and corn oil, with or without one or more other nutrient.

The meals were prepared on the morning of the test by hydration 0.5 h before ingestion. The meals were provided in a standardized 400 ml volume.

Meal Tolerance Tests : The volunteers were studied after an overnight fast on six mornings at weekly intervals. After a fasting venous blood sample had been drawn (before 10.00 AM), they were administered one of the six meals in accordance with a 6×6 Latin Square design. Each meal was consumed in 5-10 min at a steady rate. The mid-point between starting and finishing the meal was taken as zero time. Venous blood samples were drawn 0.5, 1.0 1.5 and 2.0 h after ingestion. Serum was separated within 0.5 h by clot breaking and centrifuging for 10 min at low speed.

Analysis : Each blood sample was analysed for serum glucose by the o-toluidine method and serum insulin by double antibody radioimmunoassay.

Calculations : From serial estimations of serum glucose and insulin, the following indices were derived : area under the 2-h glucose curve (AUC-G), area under the 2-h insulin curve (AUC-I), corresponding incremental area (△ AUC-G and △ AUC-I), glycaemic index (GI) and insulinaemic index, and corresponding incremental indices, △ GI and △ Insulinaemic index.

Areas under the glucose and insulin curves were calculated by using a programmable calculator (Hewlett Packard 41 CV). The glycaemic index was calculated using the formula :

$$\text{Glycaemic index} = \frac{\text{AUC-G in response to the meal}}{\text{AUC-G in response to 100 g glucose}} \times 100$$

Similarly the insulinaemic index was calculated using the formula:

$$\text{Insulinaemic index} = \frac{\text{AUC-I in response to the meal}}{\text{AUC-I in response to 100 g glucose}} \times 100$$

For the incremental indices, corresponding incremental areas were used instead of the absolute areas.

Statistical analysis : The observed and computed parameters following different meals were compared by analysis of variance (ANOVA). The points at which a significant difference between meals could be expected on the basis of ANOVA analysis were subjected to Newman-Keuls' multiple range test. Newman-Keuls' test is a rather conservative multiple range test, and therefore sometimes misses even some farily marked differences. To minimise the chances of missing genuine differences, paired comparisons by Student's t test were also made between each meal and the control (glucose meal). This was considered reasonable even in a multiple test situation because using the response to 100 g Glucose as the reference for comparison was built into the protocol of the study. Differencs were considered significant when $P < 0.05$ but marginally significant results ($0.05 < P < 0.10$) have also been reported.

RESULTS

The glycaemic and insulinaemic responses to the meals are given in Tables II and III and Figs. 1 and 2. The values of various computed indices are given in Table IV.

Coingestion of corn oil with glucose (GCo) gave a glycaemic responses comparable to G but the insulinaemic response to GCo was significantly lower than that to G at 2 h. Also AUC(I) with GCo was significantly ($P < 0.01$) lower than with G.

TABLE I : Composition of the experimental meals.

Meal	*G (g)*	*Co (g)*	*Cs (g)*	*P (g)*	*Cl (g)*	*Energy (kcal)*
1. G	100	—	—	—	—	400
2. GCo	60	18	—	—	—	400
3. GCoCs	60	9	20	—	—	400
4. GCoP	60	18	—	20	—	400
5. GCoCsCl	60	9	20	—	20	400
6. GCoCsP	60	9	20	20	—	400

G, glucose Co, corn oil Cs, casein; P, pectin; Cl, cellulose

TABLE II : Glycaemic response to the isocaloric meals tested (Mean ± SEM).

Meal	*Serum glucose (mg/dl), n = 6*					*AUG-G*	*△AUC-G*
	0 min	*30 min*	*60 min*	*90 min*	*120 min*	*(mg dl^{-1}min)*	*(mg.dl^{-1}. min)*
G	77.8±2.2	140.7±10.8	137.7±14.5	126.8±10.4	123.5±11.5	15467±1089	6127±1141
GCo	72.5±1.2	132.5±9.4	140.7±7.7	119.5±10.3	96.7±10.7	14585±744	5885±630
GCoCs	77.8±2.1	114.2±12.7	124.7±8.2	116.0±7.0	97.8±6.8	13456±709	4117±674
GCoP	77.8±1.4	116.2±5.4	101.8±6.6**	106.7±4.7	96.7±6.5	12695±329**+	3355±425**
GCoCsCl	74.7±1.5	125.2±6.7	107.7±10.8**+	97.7±10.7**+	97.7±5.4	12790±743**+	3830±675+
GCoCsP	74.2±2.9	105.7±7.8**+	90.3±3.5**+	90.0±3.6**+	88.5±3.8**+	11260±412**+	2360±393**+

+, $P < 0.05$ (by paired t test); **, $P < 0.05$ (by multiple comparisons).

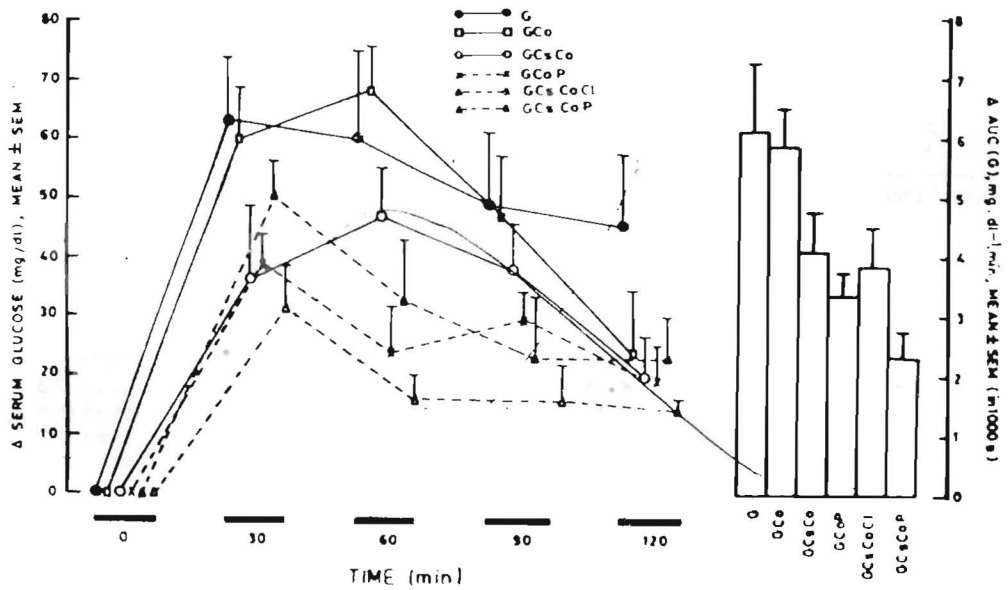

Fig. 1 : Incremental serum glucose response to the meals administered.

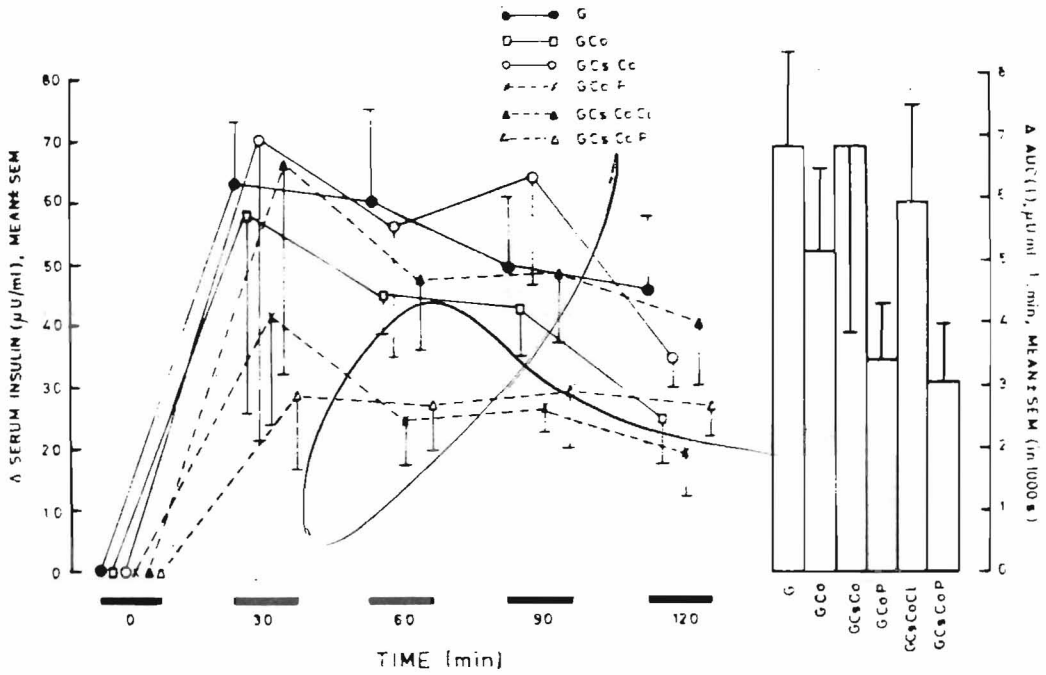

Fig. 2 : Incremental serum insulin response to the meals administered.

TABLE III : Insulin response to the isocaloric meals tested (Mean ± SEM).

Meal	*Serum insulin(μu/ml), n = 6*					*AUG-I*	*ΔAUC-I*
	0 min	*30 min*	*60 min*	*90 min*	*120 min*	($\mu g\ dl^{-1}min$)	($\mu g.dl^{-1}.\ min$)
G	3.0±1.2	65.5±24.3	58.5± 8.1	65.3±10.9	75.7±19.4	7190±1542	6830±1490
GCo	1.7±0.8	60.2±32.6	47.0± 6.6	43.7± 8.3	27.0± 6.8*+	5380±1435+	5180±1396+
GCoCs	3.0±1.0	72.7±48.4	59.2±21.3	67.7±18.7	37.7± 6.1*+	7203±3067	6843±3001
GCoP	2.3±1.0	43.8±17.6	27.2± 7.6	29.2± 4.9+	21.7± 8.0**	3703± 938+	3423± 885+
GCoCsCl	2.2±0,5	68.7±34.0	50.0±11.1	50.5±11.4	42.7±10.8**	6208±1531	5948±1566
GCoCsP	2.5±1.2	31.3±12.0	29.5± 8.2+	31.8± 9.0+	19.8± 6.3*+	3340± 962+	3040± 915+

+, P <0.05 (by paired t test); **, P <0.05 (by multiple comparisons).

TABLE IV : Indices of glycaemic and insulin response to the isocaloric meals tested (Mean ± SEM).

Meal	*GI*	ΔGI	*Insulin index*	*Δ Insulin index*
G	100.0	100.0	100.0	100.0
GCo	95.9±6.4	106.6±14.3	72.4± 7.7	73.2± 7.2
GCoCs	88.0±4.5	73.6±11.4	85.4±15.8	84.3±16.4
GCoP	83.9±5.7	62.0±10.9	56.9±12.6	55.6±12.7
GCoCsCl	83.5±4.2	64.9±10.3	86.7± 8.4	86.5± 9.0
GCoCsP	74.6±5.8	45±13.7	46.7± 6.1	44.6± 5.7

Partial isocaloric substitution of corn oil by casein (GCoCs) led to a significant reduction in glycaemic response as compared to G at 0.5 h ($P < 0.10$) and in terms of AUC (G) ($P < 0.10$). The insulin response was comparable to G, but the 2 h insulin level was significantly lower than with G.

TABLE V : Effect of individual nutrients on postprandial glycaemia and insulinaemia.

Meals compared	*Additional nutrient*	*Mean* △ AUC-G	*% change* △ AUC-I
G vs GCo	Co	− 3.9	− 24.2
GCo vs GCoCs	Cs	− 30.0	+ 32.1
GCo vs GCoP	P	− 43.0	− 33.9
GCoCs vs GCoCsP	P	− 42.7	− 55.6
GCoCs vs GCoCsCl	Cl	− 7.0	− 13.1

△AUC-G, incremental area under the 2-h glucose curve;
△AUC-I, incremental area under the 2-h insulin curve.

TABLE VI : Predictability of response to multiple nutrients.

Meals compared	*Additional nutrients*	*% change in △AUC-G*		*% change in △AUC-I*	
		Observed	*Predicted*	*Observed*	*Predicted*
G vs GCoCs	Co + Cs	− 32.8	− 33.9	+ 0.2	+ 7.9
G vs GCoP	Co + P	− 45.2	− 46.7[2]	− 49.9	− 68.9[2]
G vs GCoCsCl	Co + Cs + Cl	− 37.5	− 40.9	− 12.9	− 5.2
G vs GCoCsP	Co + Cs + P	− 61.5	− 76.7[2]	− 55.5	− 36.8[2]

[1] Predicted change is the algebraic sum of changes in response to individual nutrients indicated in Table V.
[2] The predicted contribution of pectin to the response is the mean of the two values indicated in Table V.

Addition of 20 g pectin to GCo (GCoP) led to significant reduction in glycaemic response as compared to G and GCo at 1.0 h and in terms of AUC-G, and an even more pronounced reduction in insulinaemic response, the difference being significant at 1.5 h and 2.0 h as in terms of AUC-I.

If pectin was added to the meal containing both corn oil and casein (GCoCsP), there was a further reduction in glycaemia and insulinaemia. The combination gave the lowest glycaemic and insulinaemic response among the meals studied.

If the dietary fibre added was cellulose instead of pectin (GCoCsCl), the reduction in glycaemic response was not as marked as with pectin but was still significantly lower than GCo and GCoCs at 1.0 h and 1.5 h and in terms of AUC-G. But the insulin response to GCoCsCl was quite high, being significantly lower than G only at 1 h.

DISCUSSION

The results of the present study indicate that corn oil alone has little effect on postprandial glycaemia, although it does reduce the insulinaemic response. In contrast, some earlier studies indicate that fat blunts postprandial glycaemia (6, 7, 8, 9) but not insulinaemia (6, 7). The reduced insulinaemia in our study may be due to the lower glucose content (60 g) of GCo than that of G (100 g). Shivley et al (17) concluded that insulinaemia is more sensitive than glycaemia to the amount of carbohydrate in the meal. Although the reduction in insulinaemia in our study is understandable, the lack of reduction in the glycaemic response is difficult to explain. It is possible that the reduction in insulinaemia is excessive in relation to the reduction in the ingested carbohydrate load, and hence the failure of corn oil to reduce glycaemia. Yet another reason for the unexpected result could be the individual variation in response. None of the subjects of the present study was in common with those of our previous study (8) in which we observed a reduction of 13.4% in AUC-G on coingestion of fat in contrast to only 5.7% reduction observed in the study.

The casein meal (GCoCs) reduced postprandial

glycaemia possibly by stimulating insulin secretion. As compared to GCo, this meal gave a 32.1% increase in the area under the 2-h incremental insulin curve (Table V). The insulinotropic effect of proteins has been reported earlier (10, 11, 18, 19).

The pectin meal (GCoP) reduced postprandial glycaemia while at the same time reducing insulinaemia. As compared to GCo, there was a 43.0% reduction in △AUC-G and 33.9% reduction in △ AUC-I. The effects of pectin have been reported earlier (8, 12, 13, 14) and are thought to be due to slowing down of gastric emptying because of the high viscosity of pectin (20, 21). Similar effects of pectin are seen in the presence of protein in the case of GCoCsP (Table 5). Thus in the meals studied, the hypoinsulinaemic effect of pectin dominates the insulinotropic effect of casein. If the fibre used is cellulose instead of pectin (GCoCsCl) the reduction in glycaemia as well as insulinaemia is much less. In fact, the insulin levels at 0.5 h and 1.0 h, as well as AUC-I and △AUC-I are quite comparable to the corresponding parameters in response to G and GCsCo. The insulinotropic effect of cellulose has been observed by us earlier (22) and explained tentatively on the assumption that terminal glucose units of the cellulose molecules possibly stimulate GIP secretion in the same way as glucose itself.

It is interesting to examine whether the changes in glycaemic and insulinaemic response attributable to individual nutrients (Table V) are of value in predicting the responses to meals having multiple nutrients (Table VI). For example, if we compare G with GCoCs, Co may be expected to lower △AUC-G by 3.9% and Cs by 30.0% (Table V). Thus the total predicted reduction is 3.9 + 30.0 = 33.9%. The observed reduction, 32.8%, is quite close to the predicted reduction (Table VI). Prediction of glycaemic response from nutrient composition could have considerable applied value. But since real foods have many nutrients other than those studied by us, and even non-nutrient and anti-nutrient substances, such prediction is not possible. Thus rice, potato and green gram gave glycaemic responses very different from synthetic meals of equivalent respective nutrient composition (23).

In short, corn oil by itself has only a modest effect on the postprandial metabolic response to glucose. Addition of protein and/or dietary fibre alters the response significantly. Casein as well as cellulose reduce postprandial glycaemia significantly, at least partly by enhancing insulin secretion. Pectin contributed the most to reduction in postprandial glycaemia and insulinaemia. The lowest glycaemic and insulinaemic response was seen when glucose was coingested with corn oil, casein and pectin.

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