Ind. J. Physiol. Pharmac., Volume 33, Number 2, 1989

# MODULATION OF POSTPRANDIAL GLYCAEMIA AND INSULINAEMIA BY PECTIN IN MIXED NUTRIENT COMBINATIONS

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( Received on January 15, 1989 )

**Summary :** The present study was designed to examine the effect of pectin (P) on postprandial glycaemia and insulinaemia when ingested with glucose (G), casein (Cs) and corn oil (Co) in various combinations. The study was conducted on five healthy male volunteers, on each of whom five meal tolerance tests were performed. The meals were isocaloric and consisted of G; G and P; G, Cs and P; G, Co and P; and G, Cs, Co and P. 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 and insulinaemic response to GP did not differ significantly from that to G. All the other meals, viz. GCsP, GCoP and GCsCoP, gave a significant reduction in postprandial glycaemia as compared to G. The corn oil containing meals, viz. GCoP and GCsCoP, in addition, gave a significant reduction in post-prandial insulinaemia as compared to G Pectin alone is not a dependable dietary constituent for reducing postprandial glycaemia. Its combination with protein and fat significantly lowers the postprandial glycaemic as well as insulinaemic response to orally administered glucose.

**Key words :** pectin glucose tolerance test glycaemic index

## INTRODUCTION

Fibre is a heterogenous group of substances displaying various metabolic effects arising largely from their physicochemical properties. Perhaps the earliest study which demonstrated the effect of fibre on postprandial glycaemic response was by Jeffrys (1) indicating that not all forms of fibre are effective in blunting the response Several studies indicate that guar gum (2, 3, 4, 5) and pectin (2, 3, 6, 7) have a significant attenuating effect on postprandial glycaemia and insulinaemia. This effect has been largely attributed to the viscosity of these fibres (3, 8) because cellulose, which is non-viscous, was much less effective in attenuating the postprandial glycaemia and insulinaemia (3, 7). Although the effect of pectin on postprandial glycaemia has been studied, its interaction with other nutrients in this respect is not clearly understood. Pectin is thought to reduce postprandial glycaemia by slowing gastrointestinal transit (3). We do not know how far the slowing in gastric emptying brought about by fats would modify the effect of pectin. Proteins release insulin (9, 10, 11, 12), but we do not know how their presence in food would influence the effect of pectin. The present study was designed to explore the effect of pectin, singly or in combination with macronutrients, on postprandial glycaemia and insulinaemia.

## METHODS

The study was conducted on five healthy male volunteers (age 19–21 years, weight 47–63 kg, height

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160–177 cm). The subjects reported after an overnight fast before 10.00 a.m. on five occasions at weekly interval. A fasting venous blood sample was drawn and one of the five isoenergetic meals (Table I) was provided in accordance with a Latin Square Design. The meals were formulated using glucose (G) (Glucose-D, Glindia Ltd., Bombay), casein (Cs) (SISCO Research Laboratories, Bombay), corn oil (Co) (Cornola, Ballarpur Industries, Chandrapur) and pectin (P) (SISCO Research Laboratories, Bombay). The meals were constituted on the morning of the test by hydrating half an hour prior to ingestion. The meals were provided in a standardized 400 ml volume.

TABLE I : Composition of the experimental meals.

| *Meal* | *Glucose (g)* | *Casein (g)* | *Corn oil (g)* | *Pectin (g)* | *Energy (kcal)* |
|---|---|---|---|---|---|
| G | 100 | — | — | — | 400 |
| GP | 100 | — | — | 20 | 400 |
| GCsP | 60 | 40 | — | 20 | 400 |
| GCoP | 60 | — | 18 | 20 | 400 |
| GCsCoP | 60 | 20 | 9 | 20 | 400 |

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

Each meal was consumed in 5 min at a steady rate. The midpoint between starting and finishing the meal was taken as zero time. Venous blood samples were drawn at 0.5, 1.0, 1.5 and 2.0 h. All blood samples were analysed for glucose concentration by the o-toluidine method and for insulin concentration by double antibody radioimmunoassay.

Serial estimations of serum glucose and insulin were further used for deriving the following indices: area under the 2-h glucose curve (AUC–G) and area under the 2-h insulin curve (AUC–I), corresponding incremental areas (△ AUC-G and △ AUC-I), glycaemic index (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

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

*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 fairly marked differences. To minimize the 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. Differences were considered significant when $P<0.05$.

*Ethical considerations*

The experimental protocol of the study had the

previous approval of the Ethics Committee of All India Institute of Medical Sciences. The participation was strictly voluntary and the subject could withdraw from the study at any stage. Each volunteer gave a written consent before participating in the study.

## RESULTS

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

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

| *Meal* | *Serum glucose (mg/dl), n = 5* | | | | | *AUC(G)* ($mg.dl^{-1}.min$) | *△AUC(G)* ($mg.dl^{-1}.min$) |
|---|---|---|---|---|---|---|---|
| | *0 min* | *30 min* | *60 min* | *90 min* | *120 min* | | |
| G | 79.0±2.8 | 118.0±5.8 | 93.0±3.5 | 95.2± 8.9 | 85.2±3.1 | 12030±390 | 2550±540 |
| GP | 81.6±2.6 | 114.4±5.1 | 97.8±7.3 | 99.4±10.8 | 95.2±7.6 | 12276±684 | 2484±558 |
| GPCs | 77.2±1.3 | 109.8±4.4 | 81.0±8.0 | 76.2± 5.6 | 80.0±4.7 | 10632±403+ | 1368±270 |
| GPCo | 77.6±1.6 | 91.8±5.0**+ | 88.4±7.6 | 90.4± 5.1 | 83.4±1.7 | 10666±499 | 1354±501+ |
| GPCsCo | 82.0±1.7 | 95.0±4.9**+ | 93 8±6.1 | 80.2± 6.2 | 81.0±2.1 | 10514±520 | 674±407 |

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

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

| *Meal* | *Serum insulin (μU/ml), n=5* | | | | | *AUC(I)* ($\mu U.ml^{-1}.min$) | *△AUC(I)* ($\mu U.ml^{-1}.min$) |
|---|---|---|---|---|---|---|---|
| | *0 min* | *30 min* | *60 min* | *90 min* | *120 min* | | |
| G | 2.4±0 8 | 19.8±5 8 | 13.6±2.7 | 11.8±3.4 | 12.0±1.8 | 1680±355 | 1392±387 |
| GP | 1.8±0.6 | 13.4±2.9 | 13.4±2 3 | 11.6±2.5 | 12.8±1.7 | 1414±250 | 1198±246 |
| GPCs | 1.4±0.5 | 15.8±4.0 | 10.0±2.7 | 9.0±1.7 | 9.2±2.6 | 1298±215 | 1130±234 |
| GPCo | 1.2±0.3 | 8.0±2.0 | 16.0±6.7 | 13.2±2.9 | 6.4±1.1**+ | 1256±268+ | 1112±263+ |
| GPCsCo | 1.6±0.7 | 10.2±3.8 | 15.8±3.6 | 12.8±4.9 | 7.0±1.9+ | 1322±388 | 1130±336 |

+, 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* | *△ GI* | *Insulinaemic index* | *△ Insulinaemic index* |
|---|---|---|---|---|
| G | 100.0 | 100.0 | 100.0 | 100.0 |
| GP | 102.4±6.3 | 117.0±28.3 | 94.4±18.0 | 103.1±26.2 |
| GPCs | 88.6±3.4 | 85.1±45.5 | 84.8±12.3 | 90.5±12.5 |
| GPCo | 97.1±7.5 | 49.4±14.8 | 80.6±12.7 | 90.5±17.8 |
| GPCsCo | 88.0±6.0 | 50.5±33.8 | 86.9±21.3 | 97.8±26.0 |

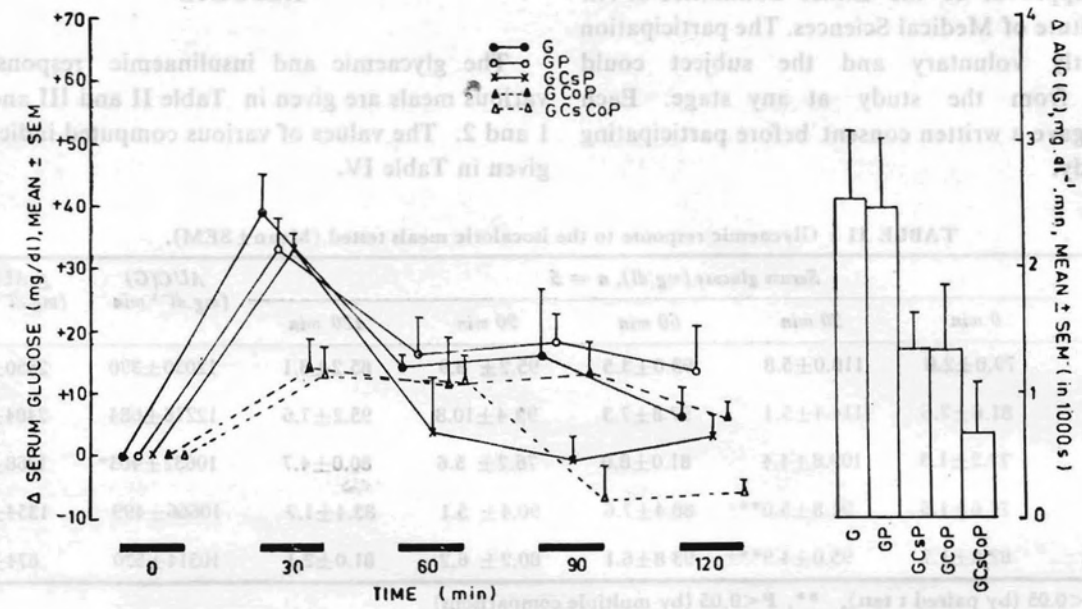


Fig. 1 : Serum glucose response to the meals administered.

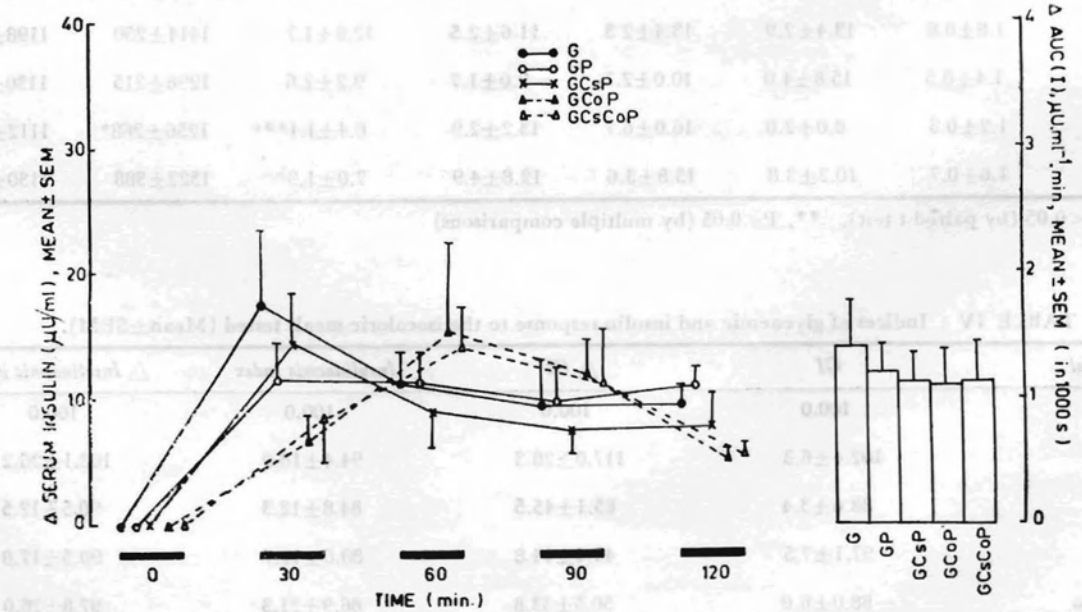


Fig. 2 : Serum insulin response to the meals administered.

When 20 g pectin was coingested with 100 g glucose (GP), both glycaemic and insulinaemic response did not significantly differ from the response to G.

Partial isoenergetic substitution by 40 g casein in the pectin containing meal (GCs P) gave a lower AUC (G) as compared to G ($P<0.05$). As compared to G, the peak glucose level did not show a significant difference, but the 1.0 h glucose level indicated considerable lowering ($P<0.10$). The glucose curve was almost a plateau between 1.0 h and 2.0 h. Although the insulin response was lower than the response to G, the difference was not significant.

On 18 g corn oil substitution (GCoP) the glycaemic and insulinaemic responses were markedly blunted. The 0.5 h glucose level was significantly lower than G ($P<0.01$), GP ($P<0.05$) and GCsP ($P<0.05$). The AUC (G) was also significantly lower than G ($P<0.05$). The insulin response indicated a delayed peak at 1.0 h and hence the 0 5 h value was lower ($P<010$) than G. The 2.0 h insulin value was significantly lower than G ($P<0.01$) and GP ($P<0.05$). The AUC (I) was also significantly lower than G ($P<0.01$).

When both 20 g casein and 9 g corn oil were substituted for 40 g glucose in the pectin-containing meal (GCsCoP) the glycaemic response was the lowest. The 0.5 h glucose level was significantly lower than G ($P<0.01$), GP ($P<0 05$) and GCsP ($P<0.05$). The mean 1.5 and 2.0 h values fell even below the fasting level reducing AUC (G) markedly. The insulin response was comparable to GCoP with a peak at 1.0 h which was higher than G ($P<0 10$) However, the 2.0 h value was significantly lower than G ($P<0.05$). The AUC(I) was comparable to GCsP and GCoP.

Table V compares the incremental glycaemic and insulinaemic response to different pairs of meals so as to bring out the contributions of pectin, casein and corn oil.

TABLE V : Effect of casein, corn oil, or both in the pectin containing meals.

| *Meals compared* | *Mean % lowering* | |
|---|---|---|
| | *△ AUC (G)* | *△ AUC (I)* |
| G vs GP | 2.6 | 13.5 |
| GP vs GCsP | 44.9 | 5.7 |
| GP vs GCoP | 45.5 | 7.2 |
| GP vs GCsCoP | 72.9 | 5.7 |

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

## DISCUSSION

Coingestion of 20 g pectin with glucose did not reduce the postprandial glycaemia significantly. This is in marked contrast with most of the previous studies including one from our laboratory (2, 3, 6, 7). Similar ineffectiveness has been reported for another viscous fibre, guar gum (13) but this fibre became effective in reducing postprandial glycaemia once it was intimately mixed with the meal (14). This explanation cannot possibly be given for our results because the technique of preparing the meals was the same as in the previous study (7). Another possible reason is the difference in the properties of the pectin used. Although the pectin used in the present study was from the same manufacturer as the one used in our previous study (7), different batches could differ in molecular weight or degree of methylation. Such differences are known to affect physical properties, and consequently, physiological

characteristics (15). 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 the previous study.

All the other meals of the present study gave a significant reduction in postprandial glycaemia as compared to G. This may be partly due to the lower glucose content of these meals (Table I). But at least some af the effect is likely to be due to the nutrients added because the glycaemic responses to 50 g and 100 g glucose do not differ significantly in normal individuals (16).

The casein containing meal (GCsP) gave a significantly lower glycaemic response which is consistent with previous reports (17). However, the insulinotropic effect of proteins (9, 10, 11, 12) was not seen, which could be because of presence of pectin in the meal which tends to reduce insulin secretion. The corn oil containing meals (GCoP and GCsCoP) reduced both the glycaemic and insulinaemic response significantly, which may be because of the tendency of fats as well as pectin to slow down gastric emptying. Attenuation of postprandial glycaemia by high fat meals has been reported earlier (18, 19).

Comparison of various pairs of meals (Table V) in terms of their effect on incremental glucose and insulin areas shows that pectin alone reduces the insulin response by 13.5%. Replacing part of the glucose in GP by casein, corn oil, or a mixture of both further brings about only a marginal reduction in the insulin response. However, the effect of these nutrients on glycaemic response is pronounced. Casein and corn oil accounted for more than 40% reduction in the response when present in GCsP and GCoP respectively. In GCsCoP the amount of casein and corn oil is half that in GCsP and GCoP respectively (Table I). Hence if the reduction in glycaemic response were to be predicted mathematically, GCsCoP (as compared to GP) may be expected to give a $\frac{1}{2}(44.9 + 45.5)\% = 45.2\%$ reduction in glycaemic response. But the reduction actually observed is 72.9%. That protein and fat together give a greater reduction in postprandial glycaemia than the sum of individual effects of both confirms the observations reported earlier (20).

In short, pectin alone plays only a minor role in reducing postprandial glycaemia Secondly, maximum reduction in glycaemia is seen with meals composed of all major nutrients, i.e. carbohydrates, proteins, fat and viscous dietary fibre.

## ACKNOWLEDGEMENTS

The present study was supported by a research grant and fellowship to SS from the Indian Council of Medical Research. The authors would like to thank Ms. Promila Kapoor and Mr. B.R. Arya for efficient technical assistance, and the volunteers for their cooperation.

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