

A comparative study of two diets in the treatment of primary exogenous obesity in children

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Received October 1st, 1978

One hundred four children, six to fourteen years of age, with primary exogenous obesity were randomly distributed in order to be subjected to two different diets, ketogenic (low carbohydrate) and hypocaloric, for eight weeks. Body weight, serum triglycerides, cholesterol, a glucose tolerance test, blood glucose and plasma insulin determination were performed before and after both diets. The results revealed significant differences in body weight and triglyceride concentrations with the two diets although they were more remarkable with the ketogenic diet. There were insignificant differences in the fasting insulin levels, insulinogenic index, and insulin concentration after a glucose tolerance test in the patients subjected to a ketogenic diet.

There are two main trends in the dietetic approach of primary exogenous obesity, one to reduce the net energy intake; and the other, not to limit the energy intake but to vary the composition of some nutrient liable to generate metabolic modifications producing weight reduction. In view of the few and controversial reports on these diets and taking into account the disheartening results of hypocaloric diets in obese children, we considered it necessary to evaluate the effects of the two diets on body weight, serum lipids and carbohydrate metabolism.

PATIENTS AND METHODS

One hundred four boys and girls ranging in age from six to fourteen years, whose percentual values for ideal weight for actual

height (IW/AH) exceeded 120, have been studied.

A venous blood sample was taken from each patient after an overnight fast and the serum triglyceride and cholesterol levels were determined.

The patients were distributed at random in two groups, they were given two kinds of diet for 8 weeks.

Diet A: Ketogenic ad libitum, with a carbohydrate intake under 20 g daily.

Diet B: 4.6 MJ with a proportion of 50% carbohydrate, 30% fat and 20% protein.

Urine samples were obtained daily to measure ketonuria.

A tolerance test (GTT) with 1.75 g of glucose per kg body weight was performed in 10 and 11 children selected at random, eating the diets A and B, respectively. Blood samples were taken after 0,30, 60 and 120 min. Glucose was determined with an AC-60 automatic analyzer and plasma insulin by radio-immunoassay based on the double antibody separation method of Hales and Randle [8]. The increases of the

total areas of insulin (ΔI), glucose (ΔG), and Insulinogenic Index (II) were calculated

$$II = \frac{\Delta I}{\Delta G}$$

At the end of the 8 week treatment, body weight, height, cholesterol, triglycerides and the GTT were determined again.

RESULTS

A) Weight reduction: mean IW/AH before diet A was 147.5, and after it 132, indicating a decrease of 9.15 ($p < 0.05$) (Fig. 1).

B) Serum cholesterol and triglycerides: Figure 2 shows the mean tri-

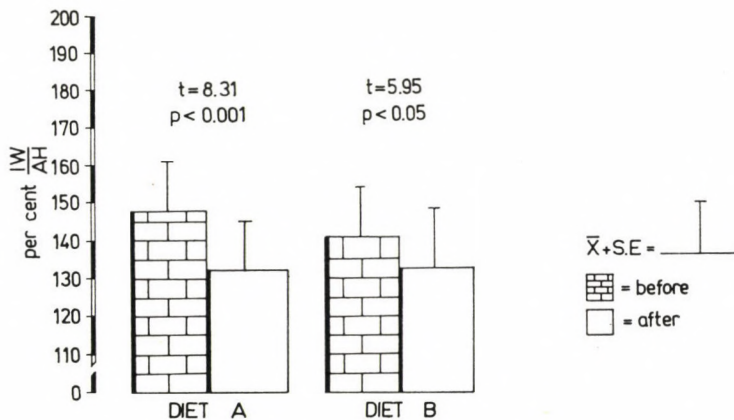


FIG. 1. Percentual values of ideal weight for actual height in obese children before and after consuming two different diets

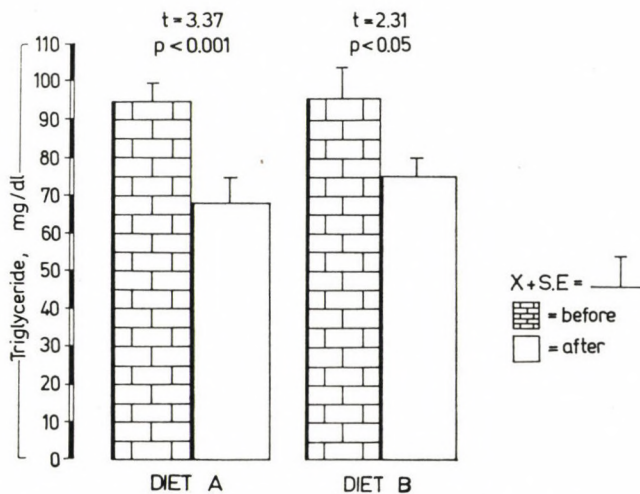


FIG. 2. Triglyceride concentrations in obese children before and after consuming two different diets

TABLE I

	Diet A			Diet B		
	Before	After	Signif.	Before	After	Signif.
Fasting Insulin	28.73 ± 3.55	7.14 ± 2.91	p < 0.001	29.47 ± 1.78	22.6 ± 1.66	p = NS
Insulinogenic Index	0.799 ± 0.188	0.289 ± 0.06	p < 0.01	0.581 ± 0.191	0.391 ± 0.106	p = NS

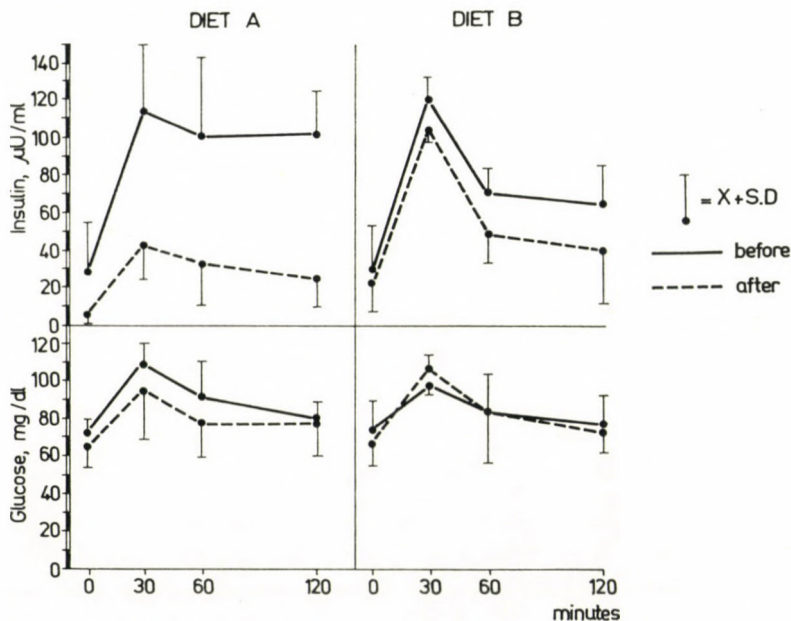


FIG. 3. Plasma insulin and glucose concentrations during a glucose tolerance test in obese children before and after consuming two different diets

glyceride concentrations in obese patients before and after the diet. Significant differences were obtained in both cases, and especially with diet A. Cholesterol values decreased slightly but the changes were not significant statistically.

C) Carbohydrate metabolism:

As can be seen in Table I and Fig. 3, diet A caused a statistically significant decrease in the fasting insulin

level and in the insulin concentration after a GTT. Those given diet B showed no significant differences. Blood glucose values were unchanged with both diets.

DISCUSSION

As can be seen in Fig. 1, the most significant decrease was achieved with diet A.

Several mechanisms have been postulated to explain the marked decrease in body weight obtained during short periods of low carbohydrate ketogenic diets. They are as follows.

Increase in basal metabolic rate [18, 19]; increased secretion of pituitary lipolytic substances [11, 13]; urinary excretion of ketone bodies which reduce the theoretical energy efficiency of lipids [14]; increased water loss due to glycogen mobilization [15].

An important advantage of ketogenic diets is the lack of quantitative limitation of food intake; this is greeted with enthusiasm by the patients.

The most common feature of diet A was anorexia, a factor which greatly contributed to the self-limitation of food intake. This is an essential element since it can be used to change the food habits and the attitude of the obese patients.

Figure 2 shows the reduction of the triglyceride level under the influence of both diets but especially of diet A. The reduction is probably related to a lower rate of triglyceride synthesis in the liver as a result of an improvement of insulin secretion [4, 9], as shown by the decrease of plasma insulin and of the II (Fig. 3, Table I).

Concerning serum cholesterol, the slight decreases obtained were non-significant in agreement with the results of Carrasco [1].

A useful way of controlling the fulfilment of the diet was to check the presence of ketonuria in the patients; it usually appeared on the third day of the diet.

Obese patients showed high fasting plasma insulin levels as seen in Table I and Fig. 3. This was not reflected in the blood glucose level. Hyperinsulinism in obese subjects has been reported by many authors, who have tried to explain it by different mechanisms: morphological [17], dietetic [7], metabolic [5], and more recently by the relative insensitivity of the insulin receptors.

Obese patients are relatively insensitive to insulin [10] and it remains to be clarified whether the hyperinsulinism is the cause or the effect of the increase of the peripheral resistance.

The role of insulin in the regulation of its receptors has been reported [3]. It seems that there is a negative connection between the insulin concentration and the number of insulin receptors.

After a glucose overload both fasting insulinaemia and the plasma insulin level decreased significantly in patients kept on diet A. The decrease might be due to two factors, a higher loss of body-weight, or an influence of the diet on insulin secretion.

REFERENCES

1. CARRASCO, B.: Dieta cetogénica como tratamiento de la obesidad. Trabajo de grado IEEM, MINSAP, Habana 1977.
2. CERASI, E.: Feedback inhibition of insulin secretion in subjects with high and low insulin response to glucose. *Diab. et Metabol.* **1**, 73 (1975).
3. CLARO, A.: El receptor a la insulina. Seminario sobre obesidad. Consejo Científico MINSAP, Habana 1977.
4. FARQUHAR, J. W., OLEFSKY, J., STERN, M., REAVEN, G. M.: Obesity, Insulin

- and Triglycerides. Obesity in Perspective. DHEW (NIH) Washington, D.C. 1973. p. 313.
5. FLATT, J. P.: Role of increased adipose tissue in the apparent insensitivity in obesity. *Amer. J. clin. Nutr.* **25**, 1189 (1972).
 6. GONZALEZ, R.: Estudio metodológico de la glicemia por el analizador automático AC-60. Centro Nacional de Información de Ciencias Médicas. Habana 1977.
 7. GROOTHOF, G., DU PLESSIS, J. P., VERSLUIS, E. E., LOUW, M. E. J., ALBERTS, A., VISAGIE, M. E., LAWBSHER, N. F., GALPIN, J. S., MARKHAM, R.: Biochemical aspects of a study of 100 obese white subjects. *S. Afr. med. J.* **49**, 893 (1975).
 8. HALES, C. M., RANDLE, P. J.: Immunoassay of insulin with insulin antibody precipitate. *Biochem. J.* **88**, 137 (1963).
 9. ICHIKAWA, K., AKANUMA, Y., KOSAKA, K., KUZUYA, N.: The effect of obesity on plasma triglycerides and fasting plasma IRI levels. Significance of Insulinogenic Index and glucose tolerance. *Horm. Metab. Res.* **9**, 429 (1977).
 10. JACOBSSON, B.: Influence of cell size on the effects of insulin and noradrenaline on human adipose tissue. *Diabetologia* **12**, 69 (1976).
 11. MUKAIDA, C. S., LICHTON, I. J.: Some dietary influences on the excretion and biological activity of an anorexigenic substance in the urine of rats. *J. Nutr.* **101**, 767 (1971).
 12. OSTLE, B.: *Statistics in Research*, 2d. ed. Iowa University Press, Ames, Iowa 1963.
 13. PAWAN, G. L. S.: Fat mobilizing substance (FMS) and body fat metabolism in man: Polypeptide hormones. Proc. fourth congress of Hungarian Society of Endocrinology Budapest 1969.
 14. PEÑA, M., PEÑA, L.: Algunas consideraciones sobre la obesidad exogena primaria en el niño. *Rev. Cuba. Pediat.* **49**, 707 (1977).
 15. RABAST, U., KASPER, H., SCHÖNBORN, J.: Obesity and low carbohydrate diets. *Nutr. and Metab.* **21**, (Suppl. 1) 56 (1977).
 16. RICHTERICH, R.: *Clinical chemistry*. Academic Press, New York 1969.
 17. SALANS, L. B.: Role of adipose cell size and adipose tissue insulin sensitivity on carbohydrate intolerance of human obesity. *J. clin. Invest.* **47**, 153 (1968).
 18. SCHÖNBORN, J., EYSSELEIN, V., RABAST, U., KASPER, H.: Vergleichende Untersuchungen des Stoffwechsels unter Kohlenhydrat- und fettreicher Reduktionkost an Normalen und Übergewichtigen. *Verh. dtsh. Ges. inn. Med.* **80**, 1224 (1974).
 19. YOSHIMURA, M., HORI, S., YOSHIMURA, H.: Effect of high fat diet on thermal acclimation with special reference to thyroid activity. *Jap. J. Physiol.* **22**, 517 (1972).

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