RELATIONSHIP BETWEEN BODY FAT MASS, CARBOHYDRATE TOLERANCE AND IRI RESPONSE DURING GLUCOSE INFUSION IN SUBJECTS WITH EARLY DIABETES

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Obesity is characterized by basal and reactive hyperinsulinism. In spite of the increased insulin secretion observed in obesity, abnormalities in glucose metabolism and increased frequency of impaired glucose tolerance are well-known to all clinicians.

The discrepancy of increased insulin secretion and disturbances in glucose metabolism is probably attributable to peripheral insulin resistance. The nature of this increased insulin resistance is still unknown and it is not yet clear in which type of tissue it is located.

Several observations have indicated the importance of adipose tissue in the development of hyperinsulinism and carbohydrate intolerance in obesity. Previous studies have shown interrelations of insulin resistance, total body fat mass, and increased adipose cell size. Moreover, a positive correlation of fasting insulin concentration, body weight, body fat and adipose cell size has been demonstrated in obesity with normal carbohydrate tolerance.

The aim of the present study was to investigate the interrelationship of total body fat mass, degree of carbohydrate intolerance and insulin response during glucose infusion in normal weight and obese subjects with early diabetes.

Key-words: Body fat mass; Carbohydrate tolerance; Early diabetes; Insulin response.

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CORRELATION OF BODY FAT MASS, GLUCOSE TOLERANCE AND IRI RESPONSE

MATERIALS AND METHODS

We have studied 17 non-obese and obese subjects suspected of having early diabetes. The definition of early diabetes includes potential, latent and asymptomatic diabetes according to the WHO definition. Glycosuria and/or disturbed carbohydrate tolerance were observed in all subjects before they were hospitalized at the Clinical Research Unit of our Institute. Each patient consumed a diet consisting of 50% carbohydrate, 30% fat and 20% protein three days before the test. Tab. 1 summarizes some relevant data regarding all subjects including age and body weight as well as the means ± SEM for non-obese and obese subjects.

The body weight of all obese subjects was more than 20% in excess of the optimal body weight as given in the tables of Möhr and Jonsen.

We carried out an i.v. glucose infusion consisting of a priming injection of 0.33 g/kg followed by a constant glucose infusion of 12 mg/kg/min in all subjects. The test was performed in conditions of bed rest following an overnight fast.

The criteria for interpretation of carbohydrate tolerance were derived from the results obtained in 115 healthy normal weight subjects without a family history of diabetes. On the basis of discriminant analysis two blood glucose criteria of the glucose infusion test were found to characterize carbohydrate tolerance:

a) - the area under the blood glucose curve for the time interval 60-120 min of the test procedure (BG area 60-120 min);
b) - the blood glucose concentration at 150 min.

The normal range of each criterion was considered as the mean + 1 SD. Values higher than the mean + 1 SD to values of the mean + 2 SD were classified as borderline range. Values higher than the mean + 2 SD were considered pathological.

Having adopted two criteria for the characterization of carbohydrate tolerance, it was necessary to define the following:

- normal carbohydrate tolerance: both criteria in the normal range or one in the borderline range;
- borderline carbohydrate tolerance: both criteria in the borderline range or one criterion 'normal' and the other 'pathological';
- pathological carbohydrate tolerance: both criteria in the pathological range or one criterion 'pathological' and the other in the borderline range.

Blood glucose concentration was estimated by autoanalyzer and plasma insulin concentration was measured radioimmunologically. Total body fat mass was calculated by the tritium dilution method.

RESULTS

The pattern of blood glucose and IRI response during the glucose infusion test is shown in fig. 1. The blood glucose and IRI concentration in the obese group were higher than in the non-obese subjects. The differences, however, were not significant. The same observation was made when areas below the glucose and IRI curves were calculated (tab. 1). The analysis of individual results of the test (tab. 1) showed a pathological carbohydrate tolerance in all obese patients. The results in the non-obese group were as follows: 2 cases with normal, 2 with borderline and 5 with pathological carbohydrate tolerance.

The data of mean body fat mass of both groups are shown in tab. 1. Significantly higher mean total body fat mass was observed in obese persons (p<0.05). There