

Role of Carbohydrate Food in Pathophysiology of Nonobese Type 2 Diabetic Subjects

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ABSTRACT

Dietary nutrients have shown overriding influence on insulin sensitivity and secretory status of diabetic subjects due to rapid lifestyle changes and urbanization. A case control study was conducted with 98 type 2 diabetic subjects and 97 healthy controls with BMI below obesity range to explore the association of dietary carbohydrate and body insulin status of nonobese diabetic subjects. Nutrient profile was taken by dietary recall method. Homeostasis Model Assessment (HOMA) was applied for measuring insulin status. Laboratory analysis and anthropometric measurements were done by standard methods. Statistical analysis was performed. Significant insulin secretory defect was found in diabetic group [Median (range), %; Control, 153 (65-285); DM, 50 (4-269)]. In diabetic group total carbohydrate [Median (range), gm/d; control, 235 (102-422); DM, 292 (151-454)], and fiber [Median (range), gm/d; control, 4.5 (2-18); DM, 6.2 (1.5-21)] consumption were found to be significantly higher than control. But no significant association was established between insulin secretions (HOMA%B) with carbohydrate consumption among the case group. So carbohydrate consumption does not influence β -cell secretory dysfunction in nonobese T2 DM patients. The possible potential effect of carbohydrate in insulin depression is not established.

Key Words: Dietary Carbohydrate, Insulin Secretion, Nonobese Type 2 Diabetics

Introduction

Diabetes mellitus is a fast expanding global health problem. The WHO Report on diabetes prevalence alarmed that diabetes is posing a serious threat to developing countries with respect to their existing health care services¹. Insulin resistance and impaired insulin secretion are usually present in patients with classic type 2 diabetes².

Basal and total 24 hour rates of insulin secretion are 3 to 4 times higher in obese subjects than in lean controls³. Again good glycemic control and duration of diabetes also influence insulin secretion in both obese and nonobese groups. Prando and his co-workers⁴ found that in well controlled groups obese persons displayed higher insulin secretion than nonobese patients especially in the early years of disease; while in poorly controlled groups obese, when compared with nonobese, displayed a similar low insulin secretion

and possibly an increased hepatic insulin resistance.

It has recently been demonstrated that a marked reduction in insulin sensitivity and a preserved insulin release characterized obese diabetic patients, while nonobese patients have a reduction in insulin release and normal insulin sensitivity⁴.

Despite possible influences of genetic and perinatal factors diet is likely to have greater and overriding influence in generation of type 2 diabetes due to rapid lifestyle changes and urbanization⁵.

There is conflicting evidence on the influence of total carbohydrate intake on insulin sensitivity⁶ and in fact a recent dietary intervention found that after six months on a low carbohydrate diet, insulin sensitivity improved among obese individuals⁷. However, the source and quality of dietary carbohydrates may differentially optimize insulin action and thereby affect the degree of

insulin resistance, which is a key underlying feature of metabolic syndrome⁸. It has been found that in subjects consuming high sucrose diet, fasting insulin concentration was higher than those consuming starch rich diet⁹. Studies in rats generally demonstrated that high intake of sucrose (18-70% of energy) or fructose (15-60% of energy) produce a decline in insulin sensitivity in the liver and later in peripheral tissues⁹. In case of complex carbohydrates, studies done in laboratory rats showed that high amylose diets appear to have beneficial effects on insulin sensitivity compared with high amylopectin diets as it is more rapidly digested than amylose¹⁰.

Ingestion of foods high in dietary fibre content appears to be associated with modest beneficial effects on insulin sensitivity. Fasting insulin concentrations are found lower among individuals reporting higher dietary fibre¹¹. Some studies report a beneficial effect on insulin sensitivity with a high consumption of dietary fibre¹², whereas other showed no effect¹³.

The glycemic index, a measure of the glycemic response to carbohydrate containing foods, has been used to physiologically classify dietary carbohydrates¹⁴. High glycemic index carbohydrates are associated with components of metabolic syndrome¹⁵. Some clinical studies have demonstrated that low glycemic index carbohydrates improve glycemic control¹⁶.

This study is an attempt to set up a causal relationship between dietary carbohydrates and the pathophysiology of diabetes in nonobese patients.

Subjects and Methods

This is a case-control study conducted in BIRDEM from July 2007 to January 2008 with 195 subjects out of which 98 were diagnosed patients of type 2 DM (case) taken from outpatient department and the remaining 97 were normal healthy subjects (control) with a BMI less than 30 and within the age limit of 30 to 50 years. The subjects were explained about the nature and purpose of the study. Their written consent, medical history was taken adequately. Clinical examination was done methodically.

Height (m), weight (kg) of the subjects was measured by standard scales and BMI was

measured by dividing the weight with square of height. Fasting plasma glucose and insulin level were measured by enzymatic method (glucose oxidase method) and ELISA respectively. β -cell secretion (HOMA B) and insulin sensitivity (HOMA S) were derived from these two parameters by Homeostasis Model Assessment (HOMA) using a particular software (HOMA-CIGMA software). Information regarding dietary carbohydrate was calculated using recall method where a questionnaire, prepared by nutritionist was introduced which dealt with dietary habits, frequency of eating and the type of cooking medium. Intakes of fibers are also calculated. The consumption was recorded on a weekly, monthly and more than a month basis. Standard sets of common utensils, utilized in our households, were used to assess the portion of food articles. Data analysis of dietary parameters was carried out by a special software. Statistical analysis was performed using SPSS-12 software.

Results

Both cases and control have BMI below obesity range (Table-I). The insulinaemic status of the cases show significant secretory defect as HOMA% B is significantly low in cases (Table-II, $p < 0.001$). Intake of both carbohydrate food and dietary fiber are significantly high in patients (Table-III). The consumptions have no effect on their insulinaemic status as HOMA% B shows no correlation with carbohydrate and fiber intake (Table-IV).

Table I: BMI of the study subjects

Variables	Control (n=97)	T2 DM (n=98)	t/p value Control vs. T2DM
BMI (kg/m ²)	22.96 \pm 2.78	23.42 \pm 2.5	1.063/0.289

Results are expressed as Mean \pm SD. Independent 't' test is performed as the test of significance at 5% level of significance n=number of subjects. BMI=Body mass index.

Table II: Insulinaemic status, β cell secretion & insulin sensitivity status of the study subjects

Variables	Control (n=97)	T2 DM (n=98)	z/p value
			Control vs. T2 DM
F. insulin (pmol/L)	75 (21-212)	76 (20-634)	0.456/0.648
HOMA % B	153 (65-285)	50 (4-269)	8.67/<0.001
HOMA % S	69 (1-247)	67 (9-256)	0.182/0.856

Results are expressed as Median (range). Mann-Whitney 'u' test was performed as the test of significance at 5% significance level n= number of subjects. F. insulin= Fasting insulin. HOMA % B= β cell function assessed by homeostasis model assessment. HOMA % S = insulin sensitivity by homeostasis model assessment.

Table III: Carbohydrate intake of study subjects

Variables	Control (n=97)	T2 DM (n=98)	z/p value
			Control vs. T2DM
CHO (g/d)	235 (102-422)	292 (151- 454)	3.008/.003
Fiber (g/d)	4.5 (2 -18)	6.2 (1.5-21)	3.794/<.001

Results are expressed as Median (range). Mann-Whitney 'u' test was performed as test of significance at 5% significance level. n= number of subjects. CHO= Carbohydrate.

Table IV: Spearman's rho correlation coefficient (r) analysis of HOMA % B with carbohydrate intake of study subjects

Variables	Control (n= 97)		T2 DM (n= 98)	
	r	p	r	P
CHO (g/d)	0.097	0.472	-0.150	0.140
Fiber (g/d)	0.215	0.109	0.095	0.354

Spearman's rho correlation coefficient (r) was performed for analysis. n= number of subjects. r= correlation coefficient. CHO= Carbohydrate.

Discussion

In this study, both carbohydrate and fiber intake was found to be significantly higher in diabetic subjects compared with the control group but no association was found between carbohydrate and dietary fiber intake and insulin secretion.

It is now evident that non obese subjects have pathophysiology of depressed insulin secretion rather insulin resistance. Their total carbohydrate and fiber consumptions are higher but not potentiate the disease pathology.

This finding claims detailed information of carbohydrate consumption on the basis of their biochemical classification or glycemic index. This will provide an organized dietary guideline for diabetic subjects regarding carbohydrate food.

The usual pathophysiological events in type 2 DM is the presence of insulin resistance followed by compensatory hyperinsulinemia but eventually β cell failure due to exhaustion. In contrast to this scenario the nonobese diabetic population in the present study did not show any hyperinsulinemia or insulin resistance (table 2). Rather they showed a highly significant reduction in insulin secretory capacity. This conforms to the previous studies in Bangladeshi population¹⁷⁻¹⁸ where a predominant secretory defect has been found in T2 DM.

The influence of carbohydrate intake on insulin status is controversial as its type and source determine its metabolic effect. The glycemic index (GI), is considered to predict the potential of carbohydrate foods to influence insulin action. Some studies showed that diets having a low GI may improve insulin secretion¹¹. In contrast, an inverse picture was found between low GI carbohydrates and the onset of frank diabetes¹⁹. Another observational study also showed that total carbohydrate intake is unrelated to fasting insulin¹¹.

A study done by raising carbohydrates resulted in improved glucose tolerance as a result of an increase in insulin secretion in older subjects²⁰ and mild diabetic subjects²¹.

Dietary fibers are claimed to have a unique role in determining insulin secretion or sensitivity. A study on middle aged and older men¹³, and postmenopausal women²² showed no effect of dietary fiber on insulin secretion. Some observational studies reported a beneficial effect on insulin sensitivity with a high consumption of dietary fiber²³.

Conclusion

The study is concluded with the finding that high intake of carbohydrate food does not affect decreased insulin secretion in non obese T2 diabetic patients.

References

1. Hussain A, Vaaler S, Sayeed MA, Mahtab H, Ali SMK, Khan AKA. Type 2 diabetes and impaired fasting blood glucose in rural Bangladesh: a population-based study. *European Journal of Public Health* (2006) 17: 291-296.
2. YKi-Jarvinen H. Role of insulin resistance in the pathogenesis of NIDDM. *Diabetology* (1995) 38: 1378-1388.
3. Polonsky KS, Given BD, Hirsch L. Abnormal patterns of insulin secretion in non-insulin dependent diabetes mellitus. *N Eng J Med* (1998) 318: 1231-1239.
4. Prando R, Cheli V, Melga P, Guusti R, Ciuchi E, Odetti P. Is type 2 diabetes a different disease in obese and nonobese patients? *Diabetes Care* (1998) 21: 1680-1685.
5. Isharwal S, Arya S, Misra A, Wasir J, Pandey R, Rastogi K. Dietary Nutrients and Insulin Resistance in urban Asian Indian Adolescents and young Adults. *Annals of Nutrition and Metabolism* (2008) 52:145-151.
6. Daly ME, Vale C, Walker M, George K, Alberti MM, Mathers JC. Dietary carbohydrates and insulin sensitivity: a review of the evidence and clinical implications. *Am J Clin Nutr* (1997) 66: 1073-1085.
7. Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J. A low carbohydrate as compared with a low fat diet in severe obesity. *N Engl J Med* (2003) 348: 2074-2081.
8. Nicola M, James B, Simin L, Edward S, Wilson P, Jacques P. Carbohydrate nutrition, insulin resistance and the prevalence of Metabolic Syndrome in the Framingham offspring Cohort. *Diabetes Care* (2004) 27:538-546.
9. Pagliassotti M, Prach PA. Quantity of sucrose alters the tissue pattern and time course of insulin resistance in young rats. *Am J Physiol* (1995) 269:641-646.
10. Higgins JA, Brand JC, Denyer GS. Development of insulin resistance in the rat is dependent on the rate of glucose absorption from the diet. *J Nutr* (1996) 126: 596-602.
11. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML. Dietary fibre, weight gain and cardiovascular disease risk factors in young adults. *JAMA* (1999) 282: 1539-1546.
12. McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. Carbohydrate nutrition, insulin resistance and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. *Diabetes Care* (2004) 27: 538-546.
13. Davy BM, Davy KP, Ho RC, Beske SD, Davrath LR, Melby CL. High fibre oat cereal compared with wheat cereal consumption favourably alters LDL-cholesterol subclass and particle numbers in middle-aged and older men. *Am J Clin Nutr* (2002) 76: 351-358.
14. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* (1981) 34: 362-366.
15. Liu S, Manson JE, Stampfer MJ, Holmes MD, Hu FB, Hankinson SE. Dietary glycemic load assessed by food frequency questionnaire in relation to plasma high density lipoprotein cholesterol and fasting plasma triacylglycerols in postmenopausal women. *Am J Clin Nutr* (2001) 73: 560-566.
16. Jimenez-Cruz A, Bacardi-Gascon M, Tumbull WH, Rosales-Grady P, Severino-Lugo I. A flexible low glycemic index Mexican-style diet in overweight and obese subjects with type 2 diabetes improves metabolic parameters during a 6 week treatment period. *Diabetes Care* (2003) 26: 1967-1970.
17. Junaid MM. Insulin resistance and insulin secretory dysfunction in obese Bangladeshi type-2 diabetic subjects M. Phil Thesis, BIRDEM Academy, University of Dhaka (2000) 60-64.
18. Al-Mahmood AK. Role of insulin deficiency and insulin resistance in the pathogenesis of type 2 diabetes in above 40 years Bangladeshi subjects (2000) M Phil Thesis, BIRDEM Academy, University of Dhaka, 56-61.
19. Marshall JA, Hoag S, Shetterly S, Hamman RF. Dietary fat predicts conversion from impaired glucose tolerance to NIDDM- The San Luis Valley Diabetes Study. *Diabetes Care* (1994) 17: 50-56.
20. Chen M, Halter JB, Porte Jr. D. The role of dietary carbohydrate in the decreased glucose tolerance of the elderly. *J Am Geriatr Soc* (1987) 35: 417-424.
21. Brunzell JD, Lemer RI, Hazzard WR, Porte Jr. D, Bierman EL. Improved glucose tolerance with high carbohydrate feeding in mild diabetes. *N Engl J Med* (1971) 284: 521-524.]
22. Juntunen KS, Laaksonen DE, Poutanen KS, Niskanen LK, Mykkanen HM. High-fibre rye bread and insulin secretion and sensitivity in healthy postmenopausal women. *Am J Clin Nutr* (2003) 77: 385-391.
23. Fukagawa NK, Anderson JW, Hageman G, Young VR, Minaker KL. High-carbohydrate, high-fibre diets increase peripheral insulin sensitivity in healthy young and old adults. *Am J Clin Nutr* (1990) 52: 524-528.