Original Article

Role of Dietary Macronutrient Composition and Fibre Intake in Development of Double Diabetes in Indian Youth

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Abstract

Introduction: Insulin resistance (IR) and obesity are common presentations of double diabetes (DD) in subjects with type-1 diabetes (T1D). There is evidence that dietary composition has an impact on developing IR. Objectives were to assess the impact of macronutrient and fibre intake on glycaemic control and the role of macronutrient composition of diet in the development of DD in subjects with T1D. **Methods:** This cross-sectional study included 77 young adults (10–25 years) with T1D. Data related to demography, anthropometry, biochemistry and body composition were collected. Dietary data was collected by fourteen-day food diary. IR was calculated using eGDR, SEARCH and CACTI equations, and metabolic syndrome (MS) was diagnosed using the International Diabetes Federation Consensus Definition. **Results:** Subjects at risk of DD had higher age, leptin levels, percentage carbohydrate consumption in diet and IR. A positive association of insulin sensitivity with fibre intake and %protein intake was noted. Poor glycaemic control, adiponectin/leptin ratio, fibre intake and insulin/carbohydrate ratio were significant negative predictors of IR. Addition of dietary factors to the regression model improved the R square and percentage of subjects identified correctly. Inclusion of dietary parameters significantly improves the prediction of the risk of development of DD in subjects with T1D. **Conclusion:** Good glycaemic control and increased intake of dietary fibre may prevent the development of IR in subjects with T1D and reduce the burden of DD.

Keywords: Double diabetes, fibre, glycaemic control, insulin resistance, macronutrients

Introduction

As per the International Diabetes Federation (IDF) atlas 2021, India has the highest prevalence as well as incidence per year of type-1 diabetes (T1D) in individuals aged less than 20 years.[1] 'Double diabetes' (DD), a recently coined term, refers to cases in which a patient demonstrates features of both, type-1 and type-2 diabetes (T2D).[2] A large epidemiological study found that 25.5% of individuals with T1D also demonstrated features of the metabolic syndrome (MS).[3] They also report DD as an independent risk factor to develop macrovascular and microvascular complications in T1D. A similar Indian study found 7% prevalence of DD in patients with youth onset diabetes. Moreover, the rate of complications even in a well-controlled DD subgroup is reported to be higher than in all those with T1D without MS, regardless of glycaemic control.[4,5] Insulin resistance (IR) and obesity are regarded as common presentations of DD in subjects with T1D. Due to the lack of awareness of metabolic comorbidities in T1D, identification and treatment of MS in T1D are extremely difficult. Lifestyle changes like dietary modification are being considered for prevention as well as management of DD.[6]

A study demonstrates various degrees of IR in individuals with long-standing T1D.[7] Liver fat and abnormal lipid profile are implied as causes of IR in these individuals, and it results in subsequent increase in the risk of cardiovascular disease.[8] Adipokines like leptin and adiponectin play an important role in food intake as well as in glucose and energy homeostasis; thus, adiponectin/leptin ratio is an important biomarker for

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the development of obesity, IR and MS.[9] It is recognized that obesity impairs insulin action.[10] Obesity is central to the development of DD and dietary patterns play a critical role in the development of obesity. Moreover, diet is considered an important modifiable risk factor in the development of IR in T1D.[11] Due to changes in eating behaviour with increased fast-food consumption by individuals with T1D, a major impact of dietary fat on IR and body weight has been noted.[12] A recent review article concluded that in the presence of various challenges in managing T1D, development of IR should also be considered. IR is likely to be made worse by the recent obesity epidemic for which lifestyle interventions of medical nutrition therapy and exercise as well as adjunctive therapies to insulin in youth with T1D may prove beneficial.[13]

Growing evidence suggests that dietary composition has a marked impact on the risk of developing IR. However, a clear elucidation of its mechanistic connections with different eating habits and food components has not yet been demonstrated.[14] A longitudinal study identified clinically meaningful modifiable factors like insulin regimen and non-modifiable like gender which were predictive of insulin requirements and glycaemic control in youth with T1D and suggested that anticipatory insulin adjustments may improve glycaemic control.[15] In a previous study, the author's group reported age, gender, body mass index (BMI) and estimated glucose disposal rate (eGDR) as significant predictors of the likelihood of MS in Indian children and youth with T1D. However, the study did not take into account the dietary intake of study participants.[16] Therefore, the objectives of this study were to assess the impact of macronutrient and fibre intake on glycaemic control of subjects with T1D and to assess the role of macronutrient composition of diet in addition to other clinical and biochemical parameters in the development of DD in subjects with T1D.

Methods and Materials

Study design and subjects: Adolescents and young adults aged 10–25 years having T1D for more than one year who were attending the diabetes clinic at a tertiary care hospital in Pune, India were included in this cross-sectional, observational study.

Clinical history and examination: Data on age of the participant, age at onset of diabetes, duration of diabetes, current medications, family and personal medical history, type of insulin regimen and total dose of insulin per day were recorded using a questionnaire administered to the participant or primary caregiver and confirmed from clinic records. All participants were examined by paediatric endocrinologists.

Anthropometry and Body Composition: Height (Seca Portable stadiometer, Hamburg, Germany up to 0.1 cm accuracy) and body weight (Seca 876 Flat scale, Hamburg, Germany, up to 100 g accuracy) were measured using standard protocols. BMI was computed using the following formula: BMI = weight (kg) ÷ height (m^2). Waist circumference (WC) and hip circumference were measured using the World Health Organization (WHO) guide to physical measurements.[17] Subsequently, the height, weight, WC and BMI were converted to z scores using Indian reference data.[18,19] Waist/hip ratio (WHR) was calculated as WC divided by the hip circumference. Body composition (fat mass, fat-free mass and total body water) was assessed using Bioelectrical Impedance Analyzer (BIA), (Tanita Model BC420MA) after a minimum of 3 hours of fasting and voiding before measurements in standing position.[20] Z scores for fat percentage and muscle mass percentage were calculated using Indian reference data.[21]

Blood pressure (BP): BP was measured on the right arm with the participant lying down quietly. The cuff was leak-tested prior to commencement of the measurement. All air was removed from the cuff, and it was wrapped snuggly and neatly around the upper arm to allow one finger under the cuff. The cuff was placed 2–5 cm above the elbow crease. All the measurements were performed manually with the same oscillometric non-invasive BP (NIBP) device (Goldway™ Multipara Monitor—Model Number GS20).

Biochemical Assessments: Six to eight ml of blood was drawn by an experienced phlebotomist after a minimum of an eight-hour fast. Fasting blood samples were then assessed for lipid profile (total cholesterol, triglycerides and HDL-C) using the enzymatic method and low-density lipoprotein-cholesterol (LDL-C) concentrations were calculated by the Friedewald formula.[22] Glycaemic control was evaluated by measuring glycosylated haemoglobin (HbA1C) using high-performance liquid chromatography (HPLC, BIO-RAD, Germany). Thyroid-stimulating hormone concentrations (TSH) were measured by chemiluminescent microparticle immunoassay (CMIA). Creatinine was measured by enzymatic method, phosphorous by ultraviolet (UV) method and 25(OH) D by HPLC. Microalbumin in spot urine was detected by immunoturbidimetry, creatinine by Jaffe w/o deproteinization and albumin/creatinine ratio (ACR) by Jaffe method. Serum leptin and adiponectin were measured by enzyme immunoassay (TiterZyme EIA kit, Assay Designs' Inc, USA)

Metabolic Syndrome (MS): As per the IDF Consensus 2017 MS in children may be diagnosed with abdominal obesity and the presence of two or more other clinical features, viz. elevated triglycerides, low HDL-cholesterol, high blood pressure and increased plasma glucose. Abdominal obesity is defined as WC >90^{th} centile for age and gender in children or WC > 80 cm in adult females or >90 cm in adult males. Other parameters were defined as follows: raised triglycerides: ≥150 mg/dl (1.7 mmol/L), reduced HDL-cholesterol: <40 mg/dl (1.03 mmol/L) in males and <50 mg/dl (1.29 mmol/L) in females, raised blood pressure: systolic ≥130 mmHg or diastolic ≥85 mmHg and impaired fasting glycaemia ≥100 mg/dl (5.6 mmol/L).[23] All the participants in this study had elevated fasting blood sugar (FBS). Thus, participants who had two or more criteria as per the definition of MS (apart from elevated FBS) were termed to have DD while those with a single criterion (except elevated FBS) were termed at risk of having DD.

The American Diabetes Association (ADA) has suggested the following target values for HbA1c in relation to age: <8.0% at age 6–12 years, <7.5% at age 13–18 years and <7.0% at age 19 + years. Individuals who met the ADA target were classified as 'good' control; those with HbA1c ≥9.5% regardless of age were classified as 'poor' control, and those with HbA1c values between the definition of 'good' and 'poor' control were classified as 'intermediate' control.[24]

IR was calculated using the formulae of estimated insulin sensitivity (eIS)[25-28]:

1. EDC = 24.31 − 12.22× (Waist/hip ratio) −3.29× (hypertension 0 = No; 1 = Yes) −0.57× (HbA1c, %)
2. SEARCH = exp (4.64725 – 0.02032 (waist, cm) – 0.09779 (HbA1c, %) – 0.00235 (Triglyceride, mg/dl)
3. CACTI-excluding adiponectin = exp (4.1075 − 0.01299× (waist, cm) −1.05819× (insulin dose, UI/kg/day) −0.00354× (Triglyceride, mg/dL) −0.00802× (Diastolic BP, mmHg))

Dietary Data: Dietary data was collected by a fourteen-day food diary. Participants were trained to record their fourteen-day food intake. Aspects related to the recording of names of foods, serving measures and sizes of all foods were included in the training protocol. Nutritive values of meals consumed were analysed using the C-Diet software which uses a cooked food database.[29] The average nutrient content for 14 days was computed for each participant. Dietary macronutrient intake was computed as a percentage of total energy intake (TEI). The insulin/carbohydrate ratio is roughly calculated using the formula 500 ÷ total daily insulin dose.[30] It determines the amount of carbohydrates (grams) covered by one unit of insulin which was calculated individually in each subject based on their carbohydrate intake and total daily dose of insulin.[31]

Physical activity data was recorded using validated activity questionnaires adapted for Indian children.[32]

Statistical Analyses: All statistical analyses were carried out using the SPSS for Windows software program, version 26 (SPSS, Chicago, IL, USA). All outcome variables were tested for normality using Q-Q plots and Kolmogorov–Smirnov and Shapiro–Wilk tests before performing statistical analyses. Differences in means were tested using two-tailed Student's *t*-test for parametric data after examining the significance of Levene's test for equal variances and Mann–Whitney U test for non-parametric data. Correlation analysis was performed using Spearman's correlation coefficient. McNemar's test for comparison of related samples and Chi-square test and Cramer's V were used for correlation analysis of categorical variables. Receiver operating characteristic (ROC) curves were used to identify the best cut-off point of each IR index using Youden formula (sensitivity + specificity − 1).[33] For testing relationships between dichotomous-dependent variables and continuous predictors, binary logistic regression analysis was carried out. *P* values < 0.05 were considered as statistically significant.

Ethical Aspect

The study was approved by the institutional ethics committee named as 'Ethics Committee, Jehangir Clinical Development Center Pvt Ltd.' vide letter no NA (our ethics committee does not provide an approval number) on 22 July 2020. Written informed consent was obtained for participation in the study and use of the patient data for research and educational purposes from participants and their parents. The procedures follow the guidelines laid down in Declaration of Helsinki 2013.

Results

Of the 92 participants, nine were not included in the current study as they did not collect their diet data at all. Further, six subjects were excluded from analysis as their dietary data was recorded for less than ten days. Thus, final results have been presented on a total of 77 participants, of which 41 (53.2%) were males. A post hoc power of 0.8 was calculated for logistic regression with a sample size of 77 and a 0.05 level of significance.

The mean age of the participants was 15.8 ± 2.8 years, and their mean duration of diabetes was 6.2 ± 2.3 years, respectively. The mean glycated haemoglobin (HbA1c) concentrations and insulin requirement of study population were 9.6 ± 1.6% and 1.02 ± 0.2 U/kg/day. Only eight (10.4%) subjects were overweight or obese according to the Indian Academy of Pediatrics 2015 classification.[18] We observed that 26 (33.8%) subjects from the study population had at least one abnormal component of MS (with the exception of high blood sugar) and hence were considered at risk for the development of DD. Of these, four subjects (5.2%) had two or more criteria as per the definition of MS (apart from elevated FBS) and were termed to have DD.

Table 1 presents a comparison of anthropometric, demographic, laboratory, body composition and dietary intake data of participants in only T1D group (no DD) and DD (risk and confirm) group. Subjects of DD group had significantly higher age, diastolic blood pressure, urine albumin/creatinine ratio, percentage carbohydrate consumption, serum leptin levels and IR by SEARCH and CACTI, excluding the fasting and adiponectin formulae, as compared to subjects with only T1D. They also had significantly reduced sleep duration, HDL-C concentrations, lean body mass, total body water percentage and protein intake. Contrary to expectation, subjects of DD group had lower fat intake and lower percentage of fat consumption in diet. As noted in Table 2, subjects with poor control had higher mean energy intake including carbohydrates, fat and protein. However, there were no significant differences in macronutrient composition of diet and fibre intake between the two groups.

As there are no cut-offs to define IR using eIS equation in subjects with T1D, we performed the ROC curve analysis to find the best equation suggestive of IR and found the CACTI equation to be the best marker of IR (Area under curve [AUC]

Table 1: Comparison of demographic, anthropometric, laboratory, body composition and dietary consumption of subjects included in the study

Parameter	Type-1 diabetes	Double diabetes (confirm and at risk)	*P*
	Median (IQR)	Median (IQR)	
DEMOGRAPHIC			
Age in years*	14.7 (3.7)	17.3 (4.4)	0.001
Duration of illness in years	5.8 (3.8)	6.8 (2.7)	0.221
Systolic blood pressure (mmHg)	110 (8)	112 (12)	0.393
Diastolic blood pressure (mmHg)*	72 (6)	76 (28)	0.019
Sleep in hours per day*	8.5 (1)	8 (.8)	0.025
ANTHROPOMETRIC			
Height z score	-0.7 (1.7)	-0.6 (1.3)	0.69
Weight z score	-0.7 (1.2)	-0.4 (1.7)	0.33
Body mass index z score	-0.5 (1)	-0.1 (1.2)	0.134
Waist circumference z score	-1.9 (1.8)	-1.5 (1.9)	0.102
Waist/hip ratio	0.8 (0.1)	0.8 (0)	0.143
BIOCHEMICAL			
Leptin* (ng/ml)	4.4 (8.8)	9.2 (12.2)	0.032
Adiponectin (mcg/ml)	18 (10.3)	16.6 (14.1)	0.48
Creatinine (mg/dl)	0.7 (0.1)	0.7 (0.1)	0.26
Total cholesterol (mg/dl)	139 (39)	126 (50)	0.154
Triglyceride (mg/dl)	67 (28)	71 (36)	0.455
HDL-C* (mg/dl)	49 (7)	45.5 (8.3)	0.002
LDL-C (mg/dl)	73.4 (36.8)	67.4 (41.5)	0.465
Very low-density lipoprotein (mg/dl)	13.4 (5.6)	14.2 (7.1)	0.455
Urine albumin/creatinine ratio* (mcg/mg)	6.6 (10.6)	14.2 (14.6)	0.005
GLYCAEMIC CONTROL			
HbA1c %	9.6 (3.1)	9.7 (1.6)	0.894
Average glucose mg/dl	209.5 (87)	195 (73)	0.28
Insulin requirement U/kg/day	0.9 (0.2)	0.9 (0.5)	0.479
BODY COMPOSITION			
Total body water %*	61.5 (11)	51.7 (9.7)	0.005
Basal metabolic rate	1235 (234)	1306 (192)	0.386
Fat % z score	-0.3 (0.9)	0.1 (1.5)	0.183
Lean body mass z score*	-2.6 (0.9)	-3.6 (1.3)	0.001
DIET			
Energy (kcal/kg/day)	34 (12)	30 (10)	0.063
Protein intake (g/kg/day)*	0.9 (0.4)	0.7 (0.4)	0.019
Fat intake (g/kg/day)*	1 (0.4)	0.9 (0.5)	0.019
Carbohydrate intake (g/kg/day)	5 (1.9)	4.8 (1.5)	0.094
Fibre intake (g/day)	23.8 (8.8)	21.5 (7.9)	0.23
Carbohydrate intake (% TEI)*	61.4 (3.8)	62.6 (4.4)	0.015
Protein intake (% TEI)	10.6 (1.1)	10.4 (1.5)	0.123
Fat % intake (% TEI)*	27.9 (3.3)	27.3 (4.4)	0.023
INSULIN RESISTANCE			
Insulin sensitivity index	11.4 (6.6)	11.2 (5.1)	0.298
Insulin/carbohydrate ratio	34.4 (19.9)	33.7 (15.2)	0.298
EGDR (mg/kg/min)	8.8 (2)	8.6 (1.7)	0.371
SEARCH* (mg/kg/min)	9.1 (3.9)	8.2 (3.4)	0.05
CACTI* (mg/kg/min)	3.9 (1.9)	3 (1.8)	0.015

*Statistically significant at $P < 0.05$

=0.674, $P < 0.05$) [Figure 1]. We obtained the highest Youden Index of 0.331 with a cut-off of 3.05 mg/kg/min to obtain a sensitivity of 56% and specificity of 77.1%. Using these cut-offs, we found that 34.2% subjects had IR. On performing

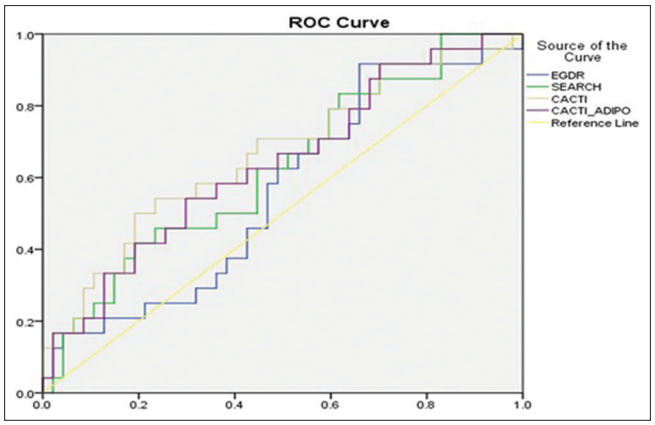

Figure 1: ROC curves of insulin sensitivity equations (eGDR, SEARCH and CACTI) predicting the risk of double diabetes in the study participants

Spearman's correlation test, we found significant positive association of eIS with fibre intake (g/kg/day) and protein intake (% of TEI). The highest association was noted with fibre intake (rho = 0.341 and $P < 0.05$) as seen in Figure 2.

Out of a total of 26 individuals in DD (risk and confirm) group, 56% had IR which was significantly higher ($P < 0.05$) than subjects with only T1D. On performing the related sample McNemar's test, risk for the development of DD in subjects with T1D was higher if they had IR. However, overweight/obesity in subjects with T1D did not show association with the development of DD ($P > 0.05$). Cramer's V showed statistically significant ($P < 0.05$) correlation of 0.331 between IR and development of DD. The Chi-square test showed that the odds ratio for the development of DD in subjects with T1D with IR was 4.3 (95% confidence interval 1.5–12.1) while the relative risk of development of DD in subjects with T1D with IR was 1.8 (95% confidence interval 1.1–2.8).

Binary logistic regression analysis was performed to develop a model to predict IR in participants with T1D with dependent variable as presence or absence of IR. The independent variables used to predict metabolic risk were glycaemic control, overweight/obesity based on BMI, duration of illness of T1D, adiponectin/leptin ratio and total body water percentage (used as fat z score had linear relation with BMI, adiponectin and leptin and in obese individual fat is increased at cost of total body water), and dietary factors like fibre intake, percentage of protein content in diet and insulin/carbohydrate ratio. Binary logistic regression analysis showed that poor glycaemic control was a significant positive predictor while adiponectin/leptin ratio, fibre intake and insulin/carbohydrate ratio were significant negative predictors of IR. We found that the addition of dietary factors to the model significantly improved Nagelkerke R square and the percentage identified correctly from 0.2 and 73.2% to 0.7 and 88.7%, respectively. The variables used in the regression analysis are shown in Table 3.

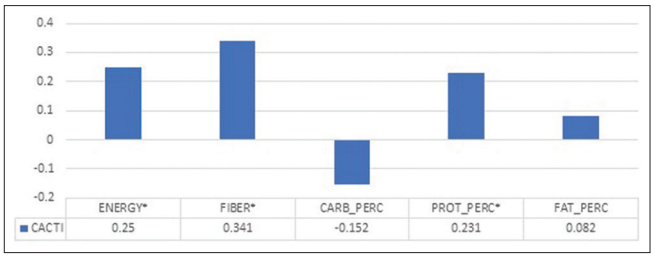

Figure 2: Correlation of dietary parameters with estimated insulin sensitivity

Table 2: Comparison of dietary intake based on glycaemic control

Dietary Parameter	Good control ($n=38$)		Poor control ($n=39$)	
	Mean	SD	Mean	SD
Energy intake (kcal/kg/day)*	31	8	36	10
Protein intake (g/kg/day)*	0.8	0.2	1	0.3
Fat intake (g/kg/day)*	1	0.3	1.1	0.4
Carbohydrate intake (g/kg/day)*	4.8	1.2	5.5	1.5
Carbohydrate intake (% TEI)	61.8	3.3	61.8	2.7
Protein intake (% TEI)	10.4	1	10.6	0.9
Fat intake (% TEI)	27.8	2.9	27.8	2.5
Fibre intake (g/day)*	24	6.9	22.8	4.5

*Statistically significant at $P < 0.05$, TEI- Total energy intake, SD- standard deviation

Table 3: Binary logistic regression for the development of insulin resistance in subjects with T1D

Variables	B	S.E.	Wald	df	Sig.	Exp (B)
Duration of illness	-0.1	0.2	0.1	1	0.7	0.9
Total body water %	0	0.1	0.3	1	0.57	1
Glycaemic control	2.4	1.1	5.2	1	0.02	11.1
Overweight/obese	1.6	1.5	1.1	1	0.3	4.8
Adiponectin/leptin ratio	-0.1	0	5.5	1	0.02	0.9
Fibre intake (g/kg/day)	-0.2	0.1	4.1	1	0.04	0.9
Protein intake (% TEI)	-0.5	0.5	0.9	1	0.36	0.6
Insulin/carbohydrate ratio	-0.3	0.1	12.7	1	<0.01	0.7

TEI- Total energy intake, df- degree of freedom, Sig- significance, S.E.- standard error

Discussion

We report a 5.2% prevalence of DD (MS in T1D) among our study population of young adults with T1D, and additionally, 28.6% of participants were at risk (at least one feature of MS except high sugar) of development of DD. Subjects with DD and risk of DD had higher age, leptin levels, percentage carbohydrate consumption in diet and IR. Using the CACTI equation cut-offs, we found that 34.2% subjects with T1D had developed IR and we found a significant correlation between development of IR and DD. The odds ratio and relative risk for the development of DD in subjects of T1D with IR were 4.3 and 1.8, respectively. A significant positive association of insulin sensitivity with fibre intake and protein intake (%

TEI) in diet was noted. The addition of dietary factors to the regression model improved the R square and percentage of subjects identified correctly. Besides, glycaemic control, adiponectin/leptin ratio, fibre intake and insulin/carbohydrate ratio were significant predictors of IR.

The authors in a previous publication have described 4.5% prevalence of MS in subjects with T1D similar to our result of 5.2%.[16] Preliminary results from a study conducted in Italy on 161 Caucasian subjects with diabetes found a 4.96% prevalence of DD.[34] Merger *et al*.[3] noted a 25.5% prevalence of DD; however, the study was conducted in adults of greater than 18 years of age. Moreover, higher prevalence in Caucasian population may also be attributed to variation in genetic and geographic factors. Similar to our results, an Indian study on subjects with T1D of age 3–32 years noted 7% prevalence of DD and found that subjects with DD were older and obese.[5]

The accelerator hypothesis attributes IR as an important factor in the causation of DD as well as a common symptom along with obesity.[35] IR has been described in adolescents with poor glycaemic control by various studies.[36] It has also been observed that hyperglycaemia contributes to the observed IR in patients with T1D.[37] A review on IR in T1D has enlisted uncontrolled glycaemia as one of the factors causing IR in T1D. They also report that poor glycaemic control in T1D is associated with hepatic IR, while IR in turn has been proposed as one of the reasons for suboptimal glycaemic control in T1D.[38] It is known that both leptin and adiponectin are involved in the regulation of lipolysis which is central to the pathogenesis of IR in T1D. A decrease in the adiponectin/leptin ratio may alter this process. A recent study has demonstrated that adiponectin concentrations are positively correlated with insulin sensitivity in T1D patients.[39] It has also been noted that the adiponectin/leptin ratio correlates with IR better than adiponectin or leptin alone and is significantly reduced in patients with MS as was seen in our study.[40,41]

Similar to our study, Katz *et al*.[42] also observed higher fat intake and lower fibre intake in subjects with T1D having poor control. It has been noted that intake of dietary fat, particularly saturated fat, appears to be associated with IR.[43] However, we did not find such an association in our study. Various studies have demonstrated the beneficial role of dietary fibre in improving insulin sensitivity while some studies show no effect.[44,45] A study has shown improvement in insulin sensitivity by consumption of dietary fibre while total carbohydrate intake had no role, with intakes assessed using a food frequency questionnaire (FFQ).[46] In contrast to our results, a study has reported that long-term dietary protein intake affects glucose metabolism. It increases glucagon and insulin stimulation, possibly reducing insulin sensitivity.[47] However, the study was performed on non-diabetic adult subjects.

To the best of our knowledge, ours is the first study to explore role of diet in developing IR in young adults with T1D. The strength of our study is that we collected a comprehensive dietary history using the fourteen-day food record method and studied role of biochemical markers like adiponectin/leptin ratio in the development of DD. The single centre cross-sectional nature of the study with lack of inclusion of glycaemic index in carbohydrate intake, inability to perform measurement of IS by euglycaemic–hyperinsulinemic clamp technique, lack of pubertal assessment and modest sample size are our limitations. Longitudinal follow-up to assess improvement in IS after making necessary modifications may strengthen the observations made by the present study.

In conclusion, subjects with poor glycaemic control had higher intake of energy, fat, protein and carbohydrates although the macronutrient composition was similar to that in subjects with good control. Diet is an important modifiable risk factor in the development of IR in subjects with T1D; high fibre, high protein, low fat and optimum carbohydrate diet may lead to an improvement in IR. Good glycaemic control and increased intake of dietary fibre may prevent the development of IR in subjects with T1D and thereby reduce the burden of DD.

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Author contributions

All the authors were involved in designing and planning the project, data collection, manuscript writing and final approval of the manuscript, data analysis and interpretation of the data.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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