Glucose Intolerance (Diabetes and IGT) In a Selected South Indian Population With Special Reference To Family History, Obesity And Lifestyle Factors - The Chennai Urban Population Study (CUPS 14)

V Mohan, CS Shanthirani, R Deepa

Abstract

Aim of the study: The aim of the study was to assess the impact of family history of diabetes, obesity and lifestyle factors particularly physical activity on glucose intolerance in a selected south Indian population.

Materials and Methods: The Chennai Urban Population Study (CUPS) is an epidemiological study involving two residential areas in Chennai in South India representing the middle and lower socio-economic group. Of the total of 1399 eligible subjects (age \geq 20 years), 1262 (90.2%) participated in the study. A detailed questionnaire was used to collect details on medical history, family history of diabetes, family income and physical activity. All the study subjects underwent a glucose tolerance test (GTT) and were categorized as having normal glucose tolerance (NGT), impaired glucose tolerance (IGT) or diabetes using WHO consulting group criteria. Obesity and abdominal obesity were defined using the new Asia Pacific guidelines.

Results: The overall prevalence of diabetes in the study population was 12.0%, (age-standardized -9.3%), which included 7.2% of known diabetic subjects and 4.8% undiagnosed diabetic subjects, while the prevalence of impaired glucose tolerance was 5.9% (age-standardized prevalence 5.0%). The prevalence of glucose intolerance (Diabetes + IGT) was significantly higher among subjects with both parents diabetic (55%) compared to those with one parent diabetic (22.1%, p=0.005) and those with no family history (15.6%, p<0.0001). Prevalence of glucose intolerance was significantly higher among subjects who had light grade physical activity (23.2%) compared to moderate (17.5%, p = 0.04) and heavy grade activity (8.1% p < 0.00001). Subjects belonging to higher socio-economic status (SES) and who also had a positive family history of diabetes had five times greater prevalence of glucose intolerance compared to subjects from lower socioeconomic status and no family history (p < 0.0001). Regression analysis revealed age (p<0.0001), waist circumference (p<0.0001), body mass index (p<0.0001), waist-hip ratio (p< 0.0001), systolic blood pressure (p<0.0001), diastolic blood pressure (p<0.0001), family history of diabetes (p < 0.0001), higher SES (p < 0.0001), moderate (p = 0.001) and light (p < 0.001) grade physical activity to be associated with glucose intolerance. Multiple logistic regression analysis showed that even after adjusting for variables like age and family history of diabetes, physical activity showed a significant association with glucose intolerance

Conclusion: The prevalence of glucose intolerance is high in this selected urban south Indian population. Lifestyle factors and family history have a synergistic effect on increasing the risk for diabetes in this population.

value of the presents a spectrum of metabolic disorders, which has become a major health challenge worldwide.¹

MV Diabetes Specialities Centre and Madras Diabetes Research Foundation, Gopalapuram, Chennai, India. Received : 9.7.2003; Accepted : 12.7.2003 The unprecedented economic development and rapid urbanization in Asian countries, particularly India has led to a shift in health problems from communicable to noncommunicable diseases. Of all the non-communicable diseases, diabetes and cardiovascular diseases lead the list. It is predicted by the World Health Organization that India would contribute nearly 57 million people to the global burden of diabetes by the year 2025.¹ Recent reports suggest that these figures are based on conservative estimates and do not include rise in diabetes-related risk factors like obesity and aging of the population. Hence, the original numbers projected may be too low and the actual figures may be around 80 million by the year 2030.

Diabetes is fortunately one of the most preventable of all non-communicable diseases. Primary prevention strategies can be formulated based on the known risk factors for diabetes. However, as the risk of diabetes varies between different ethnic groups,^{1,2} it is possible that the risk factors could also differ between different populations. Hence, it is necessary to have population-based data in different regions of the world to identify the risk factors for diabetes. This forms the basis of the present study where we have attempted to determine the risk factors for diabetes in native Indians, who are considered to be at high risk for diabetes.³

Methods

The data is from the Chennai Urban Population Study (CUPS). Methodological details and other data of the CUPS have already been published.^{4,5} Briefly, this study commenced in 1996, in two residential colonies in Chennai (formerly Madras), the fourth largest city in India. The colonies selected for the study were at Tirumangalam and T.Nagar and were chosen as they represent different socioeconomic status and also because of their geographic convenience, social differences and the local support available which would facilitate future incidence studies. All individuals aged ≥ 20 years were invited to participate in the study. The study had a response rate of 90.2% (1262/1399 participants). The aim of the study was to determine the prevalence and risk factors of diabetes and associated disorders.

Information on gender, age, anthropometric measurements including height, weight, waist and hip measurement were obtained using a standardised questionnaire by a structured interview. The body mass index (BMI) was calculated using the formula weight (Kg) / height (m²). Waist and hip were measured using standard techniques and the mean of two measurements was taken for calculating the waist-hip ratio (WHR). Blood pressure was recorded in the sitting position in the right arm to the nearest 2mm Hg with a mercury sphygmomanometer (Diamond Deluxe BP apparatus, Pune, India). Two readings were taken 5 minutes apart and the mean of the two was taken as the blood pressure.

A fasting blood sample was collected for biochemical investigations after an overnight fast of atleast 10 hours. Biochemical analysis were done on Ciba Corning Express Plus Auto Analyser (Corning, Medfield, MA, USA) using kits supplied by Boehringer Mannheim (Mannheim, Germany). Serum cholesterol (CHOD-PAP method) and serum triglycerides (GPO-PAP method) were measured using kits supplied by Boehringer Mannheim, Germany. High-density lipoprotein (HDL) cholesterol was estimated by CHOD-PAP method after precipitating low-density lipoprotein and chylomicron fractions by the addition of phosphotungstic acid and magnesium chloride. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula.⁶

Definitions and diagnostic criteria

Diabetes was diagnosed based on drug treatment for diabetes (insulin or oral hypoglycaemic agents) and/or criteria laid by the WHO consultation report i.e. fasting plasma glucose (FPG) \geq 126 mg/dl or 2 hr post-glucose value \geq 200 mg/dl. Impaired glucose tolerance (IGT) was diagnosed if FPG was <126 mg/dl and 2 hr post-glucose value (140 mg/dl and <200 mg/dl.⁷

Family history of diabetes was considered as positive if either or both the parents had diabetes. Physical activity level was graded as light, moderate and heavy based on a physical activity questionnaire, which included job-related and leisure time activities and specific questions on exercise.⁵ The monthly income of the family was recorded which was the combined income of the husband and wife taken as a single unit.

Obesity and abdominal obesity was defined using the revised criteria for Asian Indians⁸: underweight : BMI < 18.5 Kg/m², normal range : BMI 18.5 -22.9 Kg/m², overweight : at risk : BMI 23 - 24.9 Kg/m², obese I : BMI 25 - 29.9 Kg/m², obese II : BMI \geq 30 Kg/m² for both males and females, Abdominal obesity - waist circumference \geq 90 cm for males and \geq 80 cm for females.

Statistical Analysis

All the data were computed on FoxPro database and statistical analyses were done using SPSS PC Windows version 10.0 (Chicago, IL). Student's t tests was used for comparison of means and Chi-squared tests and Fisher's exact test for comparison of frequencies. The crude prevalence rates obtained from the study were age-adjusted based on the 1991 Chennai population census. Subjects with diabetes and IGT were grouped together as glucose intolerance for regression analysis. Variables like age, sex, waist, body mass index, waist-hip ratio, systolic and diastolic blood pressure, family history of diabetes, physical activity and socio-economic status (SES) were used as independent variables for the regression analysis.

RESULTS

Prevalence of diabetes

Out of the 1262 study subjects 152 were diagnosed to have diabetes, which included 91 (7.2%) known diabetic subjects, and 61 (4.8%) undiagnosed (not previously known) diabetic subjects. The crude prevalence of diabetes in the study population was 152/1262 (12.0%), while the age-standardised prevalence rate was 9.3%.

The prevalence of diabetes at age <30 years was 0.6%, at age 31-40 years : 4.8%, at 41-50 years : 15.2%, at 51-60 years : 22.9%, at 61-70 years : 34.2% and in those >70 years of age, 22.4% had diabetes. Prevalence of diabetes thus increased with increase in age until 70 years (trend chi square - 119.4,

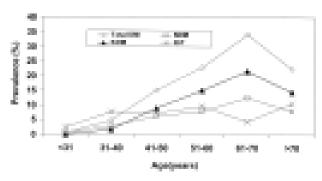


Fig. 1 : Age-wise prevalence of diabetes and IGT in the study bobulation

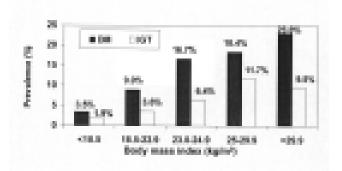


Fig. 2 : Prevalence of diabetes and IGT in relation to body mass index

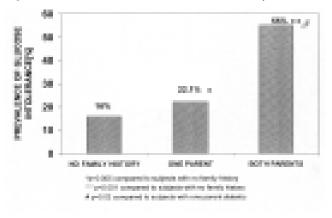


Fig. 3 : Prevalence of glucose intolerance in relation to family history of diabetes

p<0.001). The prevalence of undiagnosed diabetes and known diabetes also increased with increase age until 70 years (Fig. 1).

The total study population was categorized into quartiles based on body mass index and prevalence of diabetes in each quartile was computed. In the first, second, third and fourth quartiles of body mass index, prevalence of diabetes was observed to be 2.9%, 8.1%, 17.6% and 19.5% respectively. Diabetes showed significantly increasing trend with increasing quartiles of BMI (trend chi square - 52.1, p < 0.001).

Prevalence of diabetes was computed according to BMI classification according to the Asia Pacific guidelines.⁸ Diabetes was higher among subjects who were overweight; at risk (16.7%, p=0.007), obese I (18.4%, p<0.001) and obese II (23%, p<0.001) compared to normal subjects (Fig. 2).

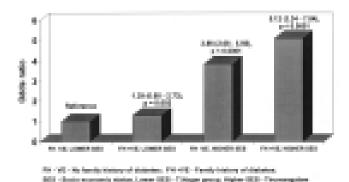


Fig. 4 : Cumulative effect of family history of diabetes and

socioeconomic status on risk of glucose intolerance

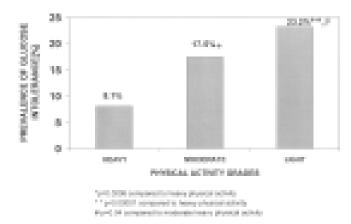


Fig. 5 : Prevalence of glucose intolerance in relation to physical activity

Prevalence of diabetes in subjects with abdominal obesity was significantly higher compared to those without abdominal obesity (27.8% vs 9.0%, p <0.001).

Prevalence of impaired glucose tolerance

Seventy four subjects (5.9%) were diagnosed to have impaired glucose tolerance (IGT). The age-standardised prevalence of IGT was 5.0%. At age <30 years, 2.8% of the population had IGT; age 31-40 years : 7.7%, 41-50 years : 7.2%, 51-60 years : 9.6%, 61-70 years : 4.5% and in those >70 years of age : 10.5% had IGT. Prevalence of IGT increased until 40 years and plateaued thereafter until 60 years of age (Fig. 1).

Prevalence of IGT was observed to be 2.2%, 3.2%, 5.9% and 12.1% in the first, second, third and fourth quartiles of BMI respectively. The increase was statistically significant (Trend chi square: 29.9, p<0.001). Prevalence of IGT among subjects with normal BMI was 3.6%, overweight at risk : 6.4%, obese I :11.7% and obese II :9.5% (Fig. 2). Prevalence of IGT in subjects with abdominal obesity (15.0%) was significantly higher compared to subjects without abdominal obesity (2.6%) p<0.001.

Risk factors for glucose intolerance

Table 1 shows the clinical profile of the study subjects. Subjects with diabetes were older, had higher body mass index, waist circumference, waist-hip ratio, systolic and diastolic blood pressure compared to those with normal glucose tolerance (p<0.05). Subjects with IGT were older and had higher body mass index compared with normal glucose tolerance subjects (p<0.05). Serum cholesterol, triglycerides and LDL cholesterol were significantly higher among subjects with diabetes and IGT compared to subjects with NGT. There was no significant difference in HDL cholesterol between the groups.

Table 2 reveals the proportion of various risk factors in

Table I: Clinical and bio	hemical profile of	of study groups
---------------------------	--------------------	-----------------

Variable	NGT (n = 1036)	IGT (n = 74)	DM (n = 152)
	()	· /	(/
Age (yrs)	40.5 ± 14.4	48.8 ± 13.3*	55.5 ± 11.9*@
Waist (cm)	74.8 ± 13.2	83.4 ± 12.4*	86.4 ± . *
Waist-hip ratio (%)	0.84 ± 0.09	0.87 ± 0.09	0.89 ± 0.08*
Body mass index	22.0 ± 4.4	24.9 ± 4.0*	24.9 ± 4.1*
(kg/m ²)			
Systolic Blood	120.3 ± 16.1	128.7 ± 14.0*	3 .4 ± 4.9*
pressure (Hg/mm)			
Diastolic blood	77.9 ± 9.4	83.6 ± 9.8*	84.0 ± 10.1*
pressure (Hg/mm)			
Fasting plasma	76 ± 14	90 ± 14*	160 ± 85 *@
glucose (mg/dL)			
Total cholesterol	164.6 ± 44.4	192.5 ± 35.4*	198 ± 53.4*
(mg/dl)			
Serum triglycerides	108.9 ± 70.2	35. ± 74*	64.9 ± 94.8*@
(mg/dl)			
HDL cholesterol	38.5 ± 11.8	40.0 ± 10.1	36.8 ± 11.1
(mg/dL)			
LDL cholesterol	107 ± 32	126 ± 32*	133 ± 38*
(mg/dL)			

*p<0.05 compared to NGT, @ p<0.05 compared to IGT

NGT- Normal glucose tolerance, IGT-Impaired glucose tolerance, DM-Diabetes mellitus

subjects with glucose intolerance. Proportion of subjects with obesity and abdominal obesity were significantly higher among subjects with glucose intolerance (diabetics and IGT subjects) compared to normal glucose tolerance (p<0.001). Prevalence of hypertension, hypercholesterolemia and hypertriglyceridemia were also significantly higher among subjects with glucose intolerance (diabetes and IGT subjects) compared to normal glucose tolerance subjects (p < 0.001), while prevalence of low HDL levels showed no significant differences between the groups. Family history of diabetes was higher among subjects with IGT and known diabetes compared to normal glucose tolerance (p < 0.001).

Family history of diabetes

Two hundred and forty eight subjects had positive family history of diabetes (either or both parents). Two hundred and nine individuals were unaware of their family history these individuals were clubbed with no family history of diabetes for further analysis. The prevalence of diabetes and IGT in relation to family history of diabetes was analysed. Prevalence of diabetes was higher among subjects who had positive family history of diabetes (18.2%) compared to subjects without a family history of diabetes (10.6%, p =0.0015). 9.3% of subjects with family history of diabetes had IGT compared to 5.0% of subjects without family history (p = 0.016). Due to small numbers, subjects with diabetes and IGT were grouped together as glucose intolerance. Prevalence of glucose intolerance among subjects who had both parents diabetic (55%) was significantly higher than with one parent diabetic (22.1%, p=0.005) and those with no family history (15.6%, p<0.0001) (Fig. 3). Subjects with glucose intolerance and family history of diabetes were younger (subjects with family history: 48 ± 13 years vs no family history: 55 (12 years, p<0.001)), had significantly higher body mass index $(25.7 \pm 3.6 \text{ Kg/m}^2 \text{ vs } 24.6 \pm 4.2 \text{ Kg/m}^2, \text{ p=0.059}), \text{ waist}$

	0			
Parameters	Normal glucose tolerance (n=1036)	Impaired glucose tolerance (n = 74)	Newly diagnosed diabetic subjects (n=61)	Known diabetic subjects (n=91)
Body mass index (%)				
\geq 25 kg/m ²	23.6	54.1*	34.4*	56.6*
Waist circumference (%)				
For Male \geq 90 cm	23.5	62.2*	50.8*	62.6*
Female \geq 80 cm				
Hypertension (%)				
SBP (140 mm Hg and/or	16.7	47.2*	47.5*	46.I*
DBP (90 mm Hg and/or				
Known hypertension				
Hypercholesterolemia (%)				
(Total cholesterol : ≥ 200 mg/dl)	19.0	43.2*	37.7*	46.2*
Hypertriglyceridemia (%)				
(Serum triglycerides : \geq 150 mg/dl)	17.6	31.1@	52.5*	42.9*
Low HDL levels (%)	36.5	35.1	41.0	37.4
(HDL cholesterol				
Males < 40 mg/dl				
Females < 50 mg/dl)				
Family history of diabetes (%)				
(Either or both parents)	17.4	31.1*	19.7	36.4*

Table 2 : Association of risk factors with glucose intolerance

* p < 0.001 compared to NGT, @ p = 0.006 compared to IGT

Table 3 : Univariate regression of determinants of
glucose intolerance

	0			
Variables	β	S.E.	p value	OR (95% CI)
Age	0.056	0.005	<0.0001	1.058
				[1.047-1.069]
Sex	0.136	0.147	NS	1.146
(Male = 1,				[0.858-1.529]
Female = 0)				
Waist	0.070	0.007	<0.0001	1.073
				[1.058-1.087]
Body mass index	0.143	0.017	<0.0001	1.154
				[1.116-1.193]
Waist-hip ratio	0.224	0.039	<0.0001	1.231
(1 unit = 0.5)				[1.159-1.350]
Systolic blood	0.036	0.005	<0.0001	1.037
pressure				[1.027-1.046]
Diastolic blood	0.058	0.007	<0.0001	1.059
pressure				[1.044-1.075]
Family history of	0.716	0.167	<0.0001	2.046
diabetes				[1.476-2.836]
(Yes = I, No = 0)				
SES	1.421	0.155	< 0.0001	4.14
(Higher SES : Tirum	angalam =	1)		[3.0-5.6]
Lower SES : T.Naga	r = 0)			
Income	0.776	0.161	< 0.001	2.174
(Income > Rs. 5000	= 1,			[1.585-2.98]
Income \leq Rs. 5000	= 0)			
	-			

OR - Odds ratio; CI - Confidence interval; SES - Socioeconomic status

 Table 4: Impact of physical activity on glucose intolerance

Physical activity grades	Dependent v	Dependent variable - glucose intolerance			
	OR	95% CI	p value		
Unadjusted					
Heavy	Reference				
Moderate	2.41	1.47 - 3.96	0.001		
Light	3.44	2.15 - 5.50	< 0.0001		
Adjusted for age					
Heavy	Reference				
Moderate	2.52	1.51 - 4.21	< 0.0001		
Light	2.43	1.49 - 3.95	< 0.0001		
Adjusted for age & famil	y history of dia	abetes			
Heavy	Reference				
Moderate	2.21	1.31 - 3.72	0.003		
Light	2.07	1.26 - 3.39	0.004		

circumference $(89.4 \pm 11.9 \text{ cm vs } 83.7 (11.9 \text{ cm, p} = 0.001)$ and waist to hip ratio $(0.91 \pm 0.08 \text{ vs } 0.87 \pm 0.09, \text{ p}=0.004)$ compared to subjects without family history.

Table 3 represents the results of univariate regression analysis using glucose intolerance as dependent variable. Age (OR-1.05, p<0.0001), waist circumference (OR-1.07, p<0.0001), body mass index (OR-1.15, p<0.0001), waist hip ratio (OR - 1.23, p<0.0001), systolic blood pressure (OR-1.03, p<0.0001), diastolic blood pressure (OR-1.05, p<0.0001), family history of diabetes (OR-2.04, p<0.0001), high income (OR -2.174, p<0.0001) and higher SES (OR - 4.14, p<0.0001) were found to be associated with glucose intolerance.

Socioeconomic status [SES] and family history of diabetes

To determine the synergistic effect of family history of diabetes and socioeconomic status on glucose intolerance, the odds ratios for glucose intolerance were computed using no family history of diabetes and lower SES as reference category. Based on colony of residence, subjects were categorised as Tirumangalam - higher SES (middle income group) and T.Nagar - lower SES (lower income group) due to the following reasons : The mean monthly income of a family at Tirumangalam was Rs. 8075 while that of a T.Nagar family was Rs. 1399. The Tirumangalam study group consisted of businessmen, professionals, executives and clerical workers while the T.Nagar population consists mostly of manual labourers, maid servants and some clerical workers.5 Subjects with negative family history of diabetes but belonged to higher SES had higher risk for glucose intolerance compared to subjects of lower SES and no family history (OR - 3.86, p < 0.0001). Similarly those with higher SES and positive family history of diabetes had five times high prevalence of glucose intolerance compared to subjects of lower SES and no family history (p < 0.0001, Fig. 4).

Physical activity and diabetes

Prevalence of diabetes, IGT and glucose intolerance was computed against grades of physical activity. Prevalence of diabetes was significantly higher among subjects with light grade activity (17.0%) compared to moderate grade (9.7%, p=0.001) and heavy grade activity (5.6%, p<0.0001) (Figure 5). The prevalence of IGT was higher among subjects with moderate (7.8%, p=0.005) and light grades of physical activity (6.2%, p=0.03) compared to heavy grade activity (2.5%). Overall, the prevalence of glucose intolerance was significantly higher among subjects who had light grade activity (23.2%) compared to moderate (17.5%, p=0.04) and heavy grade activity (8.1% p<0.00001).

Regression analysis was done using glucose intolerance as dependent variable and physical activity (heavy grade activity as reference) as independent variable. Moderate (OR -2.41, p=0.001) and light grade activity (OR -3.44, p < 0.0001) showed a strong association with glucose intolerance. This association persisted even after adjusting for variables like age and family history of diabetes (Table 4).

DISCUSSION

Prevalence of Glucose Intolerance (Diabetes and IGT)

Until 1970, the prevalence of type 2 diabetes was considered to be low in India. The projection from the WHO in the year 1998 has highlighted that India would lead the world in the prevalence of diabetes.¹ Studies done in Western countries has confirmed that the prevalence of diabetes among migrant Indians is significantly higher than the host populations.^{1,4} This might be explained by a higher genetic susceptibility to the development of diabetes among Asian Indians or stronger environmental factors eg. decreased physical activity.

Studies on native Indian population during the last 30

years have shown rising trends in the prevalence of diabetes. In the year 1972 Ahuja et al⁴ reported a prevalence of 2.1% among Urban residents, this rose to 8.0% in 1982 and is 12% in the present study. In a recent national survey of diabetes conducted in six major cities in India, Ramachandran et al ³ showed that the overall prevalence of diabetes in urban cities was 12.1%. The contribution of urbanisation to diabetes is evidenced from various studies, which have compared the prevalence of the disease was clearly indicated among the urban residents compared to their rural counterparts.^{5,9,10}

The prevalence of IGT in this study population is 5.9%, which on age standardisation is reduced to 5.0%. High prevalence of IGT in the urban and rural populations has been reported in India, which assumes great importance as it represents a large group of subjects at high risk of developing diabetes.¹¹ The ratio of IGT to diabetes was 0.5, surprisingly lower than that reported in recent Indian surveys.³ The low prevalence of IGT in this study could probably be attributed to the large number of subjects belonging to the lower SES, who are more physically active being recruited to this study. Moreover, this study is based on purposive sampling in two residential colonies in Chennai and hence is not representative of the general population.

Risk factors for glucose intolerance

Though there are several reports that highlight the high prevalence of diabetes and IGT in Indians, the exact reasons for the epidemic of diabetes in this ethnic population are not completely understood. Many epidemiological studies on Western population have highlighted various risk factors which includes increasing age, body mass index, waist circumference, waist to hip ratio, family history of diabetes, sedentary lifestyle etc.

Evidences both from prospective and cross-sectional epidemiological studies suggest obesity to be strongly linked to diabetes.^{12,13} However, measures for obesity differ between studies with some using body mass index, others waist to hip ratios and some waist circumference. In earlier studies, body mass index was measured to determine obesity and these studies have shown body mass to be a risk factor for diabetes.12,13 The mean BMI of the present study population was 22.6 kg/m², which is much lower compared to Western populations. Despite this, body mass index still showed a strong association with glucose intolerance both in univariate and multiple logistic regression analysis. Moreover, even at low BMI categorised as low risk according to WHO guidelines, the prevalence of diabetes was still high. This shows that South Indians have a high risk for diabetes even at a low BMI. Earlier studies have revealed that migrant Indians have higher body fat and high insulin resistance even at a low BMI compared to their European counterparts.¹⁴ A comparative study on native Indians and Mexican Americans reported that Asian Indians with low BMI had a waist to hip ratio similar to that reported in Mexican Americans with high rates of BMI.15

Recent studies have emphasised the importance of

abdominal obesity, which is measured using waist to hip ratio or waist circumference. In a prospective study, Ohlson *et al*¹⁶ demonstrated that a high waist to hip ratio is a predictor of diabetes independent of the degree of obesity. In the present study, the subjects with glucose intolerance had increased waist circumference and waist to hip ratio compared to the normals (Table1).

Familial and lifestyle risk factors

Subjects with family history had 2 - 3 times higher risk of developing glucose intolerance. The role of heritability has long been known in diabetes.^{3,17,18} It has been shown that subjects with family history of diabetes develop diabetes earlier compared to subjects without family history. In the present study, glucose intolerant subjects with family history were 7 years younger than subjects without a family history of diabetes. Obesity parameters like BMI and waist circumference were also significantly higher among subjects with family history of diabetes. Though there are several studies, which have looked at the association of lifestyle factors like physical activity with glucose intolerance,¹⁹ very few studies have made an attempt of looking at the cumulative effect of these factors particularly with family history of diabetes, which confers potentially high risk for developing glucose intolerance.17,18,20

In the present study subjects who had family history of diabetes and belonged to a higher SES had higher risk for glucose intolerance compared to those of lower SES. Subjects who performed moderate to light grade physical activity had higher risk for glucose intolerance compared to those who performed heavy activity. Furthermore, the strong association of physical activity with glucose intolerance persisted even after adjusting for age and family history of diabetes. This observation is similar to that reported in the National Urban Diabetes Study,⁵ wherein the subjects with sedentary lifestyle had higher prevalence of diabetes compared to subjects who performed heavy activity. The same study also demonstrated that physical activity showed an association with diabetes even after adding family history in the multiple logistic regression analysis.⁵ Overall, lifestyle factors and family history of diabetes seem to have a synergistic impact on the risk of glucose intolerance confirming an earlier study by Ko et al.²⁰ However, this study on 2847 Chinese subjects with high risk for glucose intolerance, including positive family history of diabetes showed that low SES to be an additional risk factor for diabetes. Our study result contradicts this as we found higher SES being an additional risk factor to family history of diabetes for diabetes in native Indians. As Asian Indians have higher degree of heritability for diabetes compared to their European counterparts,^{17,18} this along with lifestyle changes might be one of the reasons for the diabetes epidemic in India.

In summary, our study shows a high prevalence of diabetes and impaired glucose tolerance in a selected native South Indian population. The study also demonstrates that age, body mass index, decreased physical activity and family history of diabetes are associated with glucose intolerance. Furthermore, synergistic effect on increasing the risk for diabetes by lifestyle factors and family history of diabetes was observed in this study. With high degree of heritability and increased urbanisation, diabetes could become a major health hazard in India and this underscores the fact that prevention of diabetes must be one of the important health targets for the nation in this century.

Acknowledgement

We are grateful to Dr. Manjula Datta, Professor and Head, Department of Epidemiology, The Tamil Nadu Dr. MGR Medical University, Chennai for providing us the statistical tools and help for the present study

REFERENCES

- King H,Aubert RE,Herman WH. Global burden of diabetes, 1995 - 2025 - Prevalence, numerical estimates and projections. Diabetes Care 1998; 21:1414-31.
- 2. Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. J Diabetes Complications 2003;17:39-58.
- Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, Rao PV, Yajnik CS, Prasanna Kumar KM, Nair JD. High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. Diabetologia 2001;44:1094 - 1101.
- Pradeepa R, Deepa R, Mohan V. Epidemiology of diabetes in India - current perspective and future projections. J Indian Med Assoc 2002;100:144 - 148.
- Mohan V, Shanthirani S, Deepa R, Premalatha G, Sastry NG, Saroja R. Intra-urban differences in the prevalence of the metabolic syndrome in southern India — the Chennai Urban Population Study (CUPS No. 4). Diabet Med 2001;18:280 -287.
- Friedewald WT,Levy RI,Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem 1972; 18:499-502.
- Alberti KGMM,Zimmet PZ. Definition diagnosis and classification of diabetes mellitus and its complications. Part I: Diagnosis and classification of diabetes mellitus provisional report of a WHO Consultation. Diabet Med 1998; 15: 539 - 553.
- The Asia Pacific perspective: Redefining obesity and its treatment. Regional Office for the Western Pacific of the World Health Organization. World Health Organization, International Association for the Study of Obesity and International Obesity Task Force. Health Communications Australia Pty Limited, 2000: 22- 29.

- Ramachandran A, Jali MV, Mohan V, Snehalatha C, Viswanathan M. High prevalence of diabetes in an urban population in South India. Br Med J 1988; 297: 587 - 590.
- Ramachandran A, Snehalatha C, Dharmaraj D, Viswanathan M. Prevalence of glucose intolerance in Asian Indians. Urbanrural difference and significance of upper body adiposity. Diabetes Care 1992; 15: 1348-1355.
- Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, Haffner SM, Pettitt DJ, Sorkin JD, Muller DC, Collins VR, Hamman RF. Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. Diabetes 1997; 46:701 -710.
- Ferrannini E, Camastra S Relationship between impaired glucose tolerance, non-insulin-dependent diabetes mellitus and obesity. Eur J Clin Invest 1998; 28:3 - 6.
- Motala AA, Pirie FJ, Gouws E, Amod A, Omar MA. High incidence of Type 2 diabetes mellitus in South African Indians: a 10-year follow-up study. Diabet Med 2003; 20:23 - 30.
- 14. Joshi SR. Metabolic Syndrome Emerging clusters of the Indian phenotype. J Assoc Physicians India 2003;51: 445 446.
- Ramachandran A, Snehalatha C, Viswanathan V, Viswanathan M, Haffner SM. Risk of noninsulin dependent diabetes mellitus conferred by obesity and central adiposity in different ethnic groups: a comparative analysis between Asian Indians, Mexican Americans and Whites. Diab Res Clin Pract 1997;36:121 -125.
- Ohlson LO, Larsson B, Bjorntorp P, Eriksson H, Svardsudd K, Welin L, Tibblin G, Wilhelmsen L. Risk factors for type 2 (non-insulin-dependent) diabetes mellitus. Thirteen and onehalf years of follow-up of the participants in a study of Swedish men born in 1913. Diabetologia 1988;31:798-805.
- Mohan V, Sharp PS, Aber VR, Mather HM and Kohner EM. Family histories of Asian Indian and Europeans non-insulindependent diabetic patients. Practical Diabetes 1986; 3: 254-256.
- Viswanathan M, Mohan V, Snehalatha C, Ramachandran A. High prevalence of type 2 (non-insulin-dependent) diabetes among the offspring of conjugal type 2 diabetic parents in India. Diabetologia 1985;28:907 - 910.
- Mensink M, Feskens EJ, Saris WH, De Bruin TW, Blaak EE. Study on lifestyle intervention and impaired glucose tolerance Maastricht (SLIM): preliminary results after one year. Int J Obes Relat Metab Disord 2003;27:377 - 384.
- Ko GT, Chan JC, Yeung VT, Chow CC, Tsang LW, Cockram CS. A low socio-economic status is an additional risk factor for glucose intolerance in high risk Hong Kong Chinese. Eur J Epidemiol 2001;17:289 - 295.