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Rising prevalence of NIDDM in an urban population in India

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Summary A survey conducted in 1988–1989, in the city of Madras, South India, showed that the prevalence of diabetes mellitus in adults was 8.2 % and prevalence of impaired glucose tolerance (IGT) was 8.7 %. The present survey was another cross-sectional study conducted 5 years later in the same urban area to study the temporal changes in the prevalence of diabetes and IGT. The two sample populations surveyed were similar in age structure and socioeconomic factors. In the second survey in 1994–1995, a total of 2183 subjects, 1081 men and 1102 women, with a mean age of 40 ± 12 years were tested by an oral glucose tolerance test; fasting and 2-h post-glucose plasma glucose were measured. Anthropometric measurements, details of physical activity and clinical history of diabetes were recorded. Age-standardised prevalence of diabetes had increased to 11.6 % from 8.2 % in 1989 and IGT was 9.1 %, similar to 8.7 % in 1989. Multiple regression analysis showed age, waist : hip ratio, body mass index (BMI) and female sex were correlated to diabetes. Family history of diabetes showed interaction with age and BMI. Prevalence of IGT correlated to age, BMI and waist : hip ratio. This study highlights the rising trend in the prevalence of non-insulin-dependent diabetes (NIDDM) in urban Indians. The persistent high prevalence of IGT may also be a predictor of a further increase in NIDDM in the future. No significant differences in the anthropometric data were noted in this compared to the previous study. [Diabetologia (1997) 40: 232–237]

Keywords NIDDM, impaired glucose tolerance, epidemiology, India, urban population.

Asian Indians who have migrated from the Indian subcontinent are reported to have a high prevalence of non-insulin-dependent diabetes mellitus (NIDDM) [1–10]. In the last decade studies have indicated that native Indians in the urban population have a similar high prevalence of NIDDM [10–13]. Rural Indian populations have a significantly lower prevalence than their urban counterparts [10, 13, 14]. Studies in migrant Indians, and Indians in urban and rural southern India illustrate the strong influences of genetic and environmental factors in the development of NIDDM [1, 13, 14]. It has also been suggested that the prevalence of NIDDM has been increasing [1, 15–17]. There are no data on the temporal change in the prevalence of NIDDM in native Indians. In 1992, we reported the high prevalence of NIDDM in urban South Indians based on a survey carried out in 1988–1989 [13]. Here we report the change in the prevalence of diabetes and impaired glucose tolerance (IGT) in an urban population based on a second cross-sectional survey carried out in 1994–1995 in the same population. The sample populations studied in the two surveys were similar in age structure and socioeconomic factors, although the same individuals were not studied in both surveys.

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Abbreviations: NIDDM, Non-insulin-dependent diabetes mellitus; IGT, impaired glucose tolerance, WHR, waist : hip ratio; PARF, population attributable risk fraction; OR, odds ratio; CI, confidence interval.

Subjects and methods

The study population was located in the city of Madras in southern India. A detailed description of the population has been given previously [13]. The majority of subjects were Hindus and their staple food was rice. The population was literate and belonged to various socioeconomic strata, there were businessmen, traders, factory workers, clerical staff, professionals and their families. Three clusters of defined areas were chosen to represent the various socioeconomic groups in the city. We conducted a preliminary door-to-door survey of all the households in the area with details of name, sex and age of all the inhabitants of age 20 years and over. We obtained the support of the local medical practitioners as well as voluntary welfare organisations for conducting the survey.

Survey procedure. The procedure was similar to our previous survey and was based on World Health Organisation (WHO) recommendations [18]. After registration, a fasting venous blood sample was taken and the subject was given 75 g anhydrous glucose in 250 ml of water. A second blood sample was drawn 2 h later. In all the known diabetic subjects a glucose load was administered after antidiabetic drugs had been withdrawn for 2 days before the test. During the waiting period, a form was filled in with the following details: name, year of birth, sex, occupation, level of physical activity, family income, number of family members and family history of diabetes. Physical activity was assessed based on type of occupation and time spent on leisure-time physical activities. In the questionnaire, details of occupation, periods spent on desk work, and manual labour were ascertained.

Similarly, time spent on house work, outdoor activities and exercise, especially for unemployed individuals, was assessed. All subjects were finally divided into four categories of physical activity based on the occupation and exercise programmes, namely, sedentary (executives and elderly and those taking no regular exercise), light (housewives and clerks and no regular exercise), moderate (skilled workers and/or regular exercise), and heavy (manual labourers and agricultural workers or those engaged in strenous exercise programmes). Weight, height, and waist and hip measurements were recorded. The waist and hip measurements were made with subjects standing wearing thin clothes. The waist was defined as the smallest girth between the costal margin and iliac crests and hip as the circumference at the level of the greater trochanters. The mean of two readings taken by two independent nutritionists was recorded for each individual. Body mass index (weight in kg/height in m^2) (BMI) and the waist : hip ratios (WHR) were calculated. About 40–50 subjects were tested on a single survey day. The project was conducted during 1994–1995.

Blood glucose measurements were made in the fasting state and 2 h following a 75 g glucose load. Blood was collected in oxalate fluoride mixture and transported on ice for measurement of plasma glucose in the laboratory. The measurements were done within 8 h, using the glucose-oxidase peroxidase method (Boehringher Mannheim reagents; Mannheim, Germany) using a Hitachi 704 autoanalyser (Mannheim, Germany).

The coefficients of variation of glucose estimations were less than 2 %, as estimated by the internal quality control check carried out at intervals of 30 tests.

Measurement of fasting and 2 h plasma insulin responses, were performed in 1020 individuals. The radioimmunoassay kit supplied by the Bhabha Atomic Research Centre, Bombay was used. It used a modified procedure of Herbert et al. [19], using a second antibody and polyethylene glycol mixture for separation of the bound and free labelled compound. The lowest detection limit was 2 μU/ml. Inter and intra assay coefficients of variation were less than 6.5 and 5 %, respectively.

Statistical analysis

Prevalence of diabetes and IGT were age-adjusted separately for men and women, to the age distribution of the population in Madras as per the 1991 census by the direct standardisation method [16]. In the previous study, the standardisation was performed using the census figures for 1981. For comparison, the 1989 figures were also age-standardised using the census figures for 1991. Plasma insulin values were corrected for BMI by using linear regression. Comparison between mean values in groups was done by the Student's *t*-test or one-way analysis of variance (ANOVA) as appropriate and the proportions were tested by the chi-square test.

Trend chi square test was performed to assess the combined effect of BMI and WHR in tertiles of those parameters. Multiple logistic regression analysis was done to study the association of various parameters to the development of diabetes and IGT separately. The analyses were done separately for men and women. Population attributable risk fractions (PARF) were calculated by the formula:

$$\text{PARF} = \frac{100\,\text{P}\,(\text{R}-1)}{[\text{P}(\text{R}-1)+1]}$$

where P was the proportion of the exposed individuals in the total population, and R was the relative risk estimate of the exposure [20].

Results

Prevalence of diabetes and IGT compared with the previous survey. A total of 2183 subjects were tested and the mean response rate was 84 % (78, 86 and 88 % in 3 areas). Table 1 shows the characteristics of the populations studied in the two surveys. A larger number was studied in the present survey. The mean age of the population in this survey was higher than the 1989 survey. No other significant differences were seen.

Age-standardised prevalence (1981 census) of diabetes was 8.2 % and IGT was 9.1 % in 1989. Table 2 shows the prevalence of diabetes and IGT in the present study in comparison with the prevalence in 1989 (both figures age-standardised to 1991 census).

The prevalence of diabetes has increased to 11.6 % from 8.3 %. A 40 % increase in the prevalence of NIDDM was present in the total population during the 6 years ($\chi^2 = 6.77$, $p = 0.009$, $df = 1$). Increase in the prevalence of diabetes was significant in women ($\chi^2 = 7.5$, $p = 0.006$, $df = 1$). Women showed a slightly higher prevalence of diabetes and IGT than men, although the difference was statistically non-significant. Prevalence of IGT also marginally increased (from 8.7 to 9.1 %), even more in women. The ratio of known to new cases of diabetes was 1.18 (162 and 137, respectively) (in men 1.54, in women 0.93).

Comparison of the age-specific prevalence in the two surveys showed that in the 1995 survey there

Table 1. Details of the population samples of the 1989 and 1995 surveys

	1989	1995
Number	900	2183
Men : women	457 : 443	1081 : 1102
Age (years)		
Total	38 ± 12	40 ± 12[a]
Men	40 ± 11	42 ± 12[a]
Women	37 ± 12	39 ± 12[a]
Body mass index (kg/m^2)		
Total	23.0 ± 4.0	22.3 ± 4.3
Men	22.5 ± 3.5	21.7 ± 3.8
Women	23.4 ± 4.0	22.9 ± 4.5
Waist girth (cm)		
Total	81.0 ± 11.2	78.9 ± 11.1
Men	84.5 ± 10.7	80.8 ± 10.6
Women	77.4 ± 10.5	77.0 ± 11.2
Waist : hip ratio		
Total	0.86 ± 0.07	0.87 ± 0.07
Men	0.91 ± 0.1	0.90 ± 0.07
Women	0.81 ± 0.07	0.84 ± 0.07
Obesity (%) (BMI ≥ 27 and ≥ 25 for men and women respectively)		
Total	22.0	18.1
Men	10.0	8.0
Women	33.0	28.0
Family income in RS		
Men	3151 ± 1624	3513 ± 1858
Women	3121 ± 1621	3336 ± 2020

Values are mean ± SD
[a] $p < 0.01$ vs 1989 values, by *t*-test

Table 2. Temporal change in the age-adjusted prevalence of diabetes and IGT in urban Indians

	Diabetes (%)		IGT (%)	
	1989	1995	1989	1995
Total	8.3 (6.5–10.1)	11.6[a] (10.6–12.6)	8.3 (6.5–10.1)	9.1 (8.1–10.1)
Men	8.3 (5.8–10.8)	10.4 (8.4–12.4)	8.6 (6.1–11.1)	8.8 (7.1–10.5)
Women	7.6 (5.1–10.1)	12.7[b] (10.7–14.7)	8.3 (5.7–10.4)	9.5 (7.8–11.2)

All values are age-adjusted to the 1991 census; Values in brackets show confidence intervals, [a] $\chi^2 = 6.77$, $p = 0.009$; [b] $\chi^2 = 7.5$, $p = 0.006$

was a significantly higher prevalence of diabetes in the age groups 35 to 44 years (8.5 to 11.5 % $p = 0.05$) 45 to 54 (16.8 to 26.4, $p = 0.0003$) and 55 to 64 (20.0 to 32.7 %, $p = 0.02$ χ^2).

Analysis of variables associated with diabetes and IGT in the present survey. Mean ages ± SD of men with IGT (45.9 ± 12.2 years) and diabetes (49.6 ± 8.3 years) were higher ($p < 0.001$) than men with normoglycaemia (39.6 ± 12.3 years). Similarly women with IGT (44.7 ± 11.4 years) and diabetes (47.8 ± 10.3 years) had a higher mean age than normal women (36.9 ± 11.6 years). BMI in normal men compared to

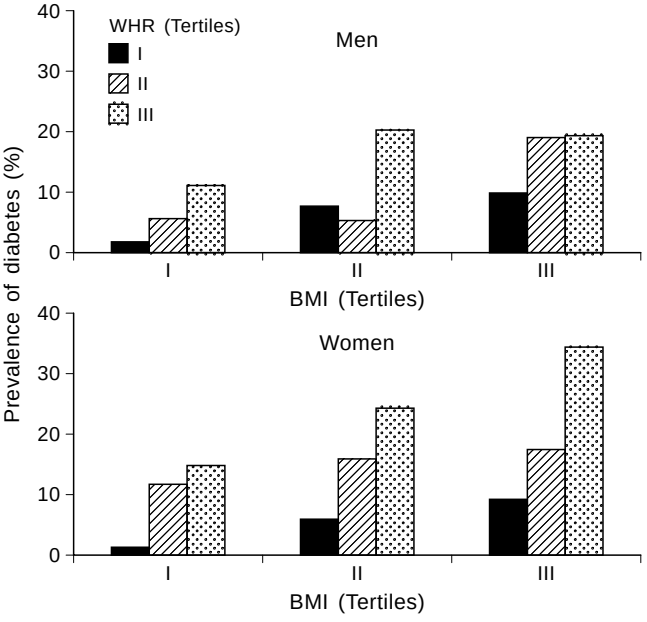

Fig. 1. Age-standardised prevalence of diabetes within crossed tertiles of body mass index and waist : hip ratio in men (upper panel) and women (lower panel)

those with IGT and diabetes were 21.2 ± 3.8, 23.0 ± 3.4 and 23.3 ± 3.5 kg/m^2 and the corresponding values in women were 22.3 ± 4.5, 24.5 ± 4.7 and 24.8 ± 4.1 kg/m^2. The corresponding WHR in men were 0.89 ± 0.07, 0.91 ± 0.05 and 0.93 ± 0.06 and women 0.83 ± 0.07, 0.85 ± 0.07 and 0.88 ± 0.06 kg/m^2.

BMI and WHR were higher ($p < 0.001$) in IGT and diabetes, in both men and women. Percentage with positive family history of diabetes was similar in normal (26.2 %) and IGT (26 %) groups whereas it was significantly more in those with diabetes (47.2 %, diabetes vs IGT $\chi^2 = 23.3$, $p < 0.001$, diabetes vs normal $\chi^2 = 52.2$, $p < 0.001$, $df = 1$).

The interactions of BMI and WHR in diabetes in men and women are shown in Figure 1. Trend chi square was used for comparison between tertiles. In men, an increase in WHR in the first and second tertiles of BMI produced a significant increase in the prevalence of diabetes ($p = 0.003$ and $p = 0.0002$, respectively) but in the third tertile of BMI, the effect was evident only up to the second tertile of WHR. In women, an increase in WHR for all the tertiles of BMI produced a significant increase in the percentage of diabetes ($p < 0.001$), and the effect of BMI was most prominent in the first tertile of WHR ($p < 0.001$).

PARF for NIDDM in men was 59 % for an increase in BMI above the first tertile (> 19.72 kg/m^2) and for a similar increase in WHR (> 0.82) was 73.5 %. In women, PARF was 68 % for an increase in BMI above the first tertile and 63 % for increasing WHR.

Table 3 shows the fasting and 2 h plasma insulin values in normal, IGT and diabetic subjects. The

Table 3. Plasma insulin response and insulin : glucose ratios in relation to glucose tolerance

	Normal $n = 725$	IGT $n = 136$	Diabetes $n = 159$
Insulin (μU/ml)			
Fasting	15.0, 10.4	19.3, 10.9[a]	21.1, 13.2[b]
2 h	48.0, 43.4	106.8, 71.0	73.3, 59.8[b,c]
Insulin/glucose ratio (pmol/mmol)			
Fasting	23.4, 14.9	25.0, 14.5	10.4, 12.9[b,c]
2 h	70.6, 54.1	91.3, 61.8	35.7, 37.3[b,c]

Mean ± SD BMI corrected; Difference between group means tested by 1 way ANOVA: $p < 0.05$; [a] normal vs IGT, [b] Normal vs diabetes, [c] IGT vs diabetes

Table 4. Results of multiple logistic regression analysis dependent variable – diabetes compared to normoglycaemic subjects

Independent variables	Coefficient	SEB	Significance	Odds Ratio
Age	0.47	0.14	0.0008[a]	1.60
Sex (M = 0, F = 1)	0.61	0.24	0.0104[a]	1.84
BMI	0.48	0.14	0.0008[a]	1.62
WHR	1.197	0.29	0.0000[a]	3.31
Family history of diabetes	0.36	0.56	0.52	1.43
Physical activity	0.07	0.33	0.834	1.07
Sex by family history	0.41	0.30	0.17	1.51
Age by family history	0.37	0.15	0.01[a]	1.45
Age by physical activity	0.11	0.07	0.123	1.11
BMI by waist : hip ratio	0.11	0.05	0.041[a]	0.89
BMI by family history	–0.21	0.08	0.011[a]	0.81
WHR by physical activity	0.10	0.10	0.303	0.90

[a] Significant; Age in 10-year categories, BMI = units of 2, waist : hip ratio = units of 0.2, family history and sex dichotomised, Physical activity – light, moderate, heavy; SEB, standard error of regression coefficient

values have been corrected for BMI using linear regression. Insulin (pmol/l) to glucose (mmol/l) ratios are also shown. Fasting insulin values showed an increased trend with higher glucose values. Post-glucose 2-h insulin was significantly higher in IGT, compared to normal ($p < 0.05$ ANOVA).

The value was lower in diabetes compared to IGT, but similar to the normal value. Fasting insulin : glucose ratios were similar in normal and IGT while they were lower in diabetes ($p < 0.05$) compared to both groups. In IGT 2-h insulin : glucose ratio was higher, but in diabetes there was a significantly lower value compared to IGT and normal subjects ($p < 0.05$).

Multiple regression analysis showed that the parameters influencing diabetes were age, WHR, BMI and female sex in that order of significance (Table 3). Level of physical activity was not correlated with diabetes. A positive family history of diabetes was strongly related with age and BMI. BMI and WHR were also strongly correlated with each other.

A positive family history of diabetes produced a higher risk of diabetes in older individuals. The age

Table 5. Results of multiple logistic regression analysis dependent variable – IGT compared to normoglycaemic subjects

Independent variables	Coefficient	SEB	Significance	Odds Ratio
Age	0.28	0.14	0.044[a]	1.32
Sex (M = 0, F = 1)	0.24	0.23	0.289	1.28
BMI	0.50	0.14	0.0003[a]	1.65
WHR	0.82	0.30	0.007[a]	2.27
Family history of diabetes	–0.68	0.61	0.26	0.51
Physical activity	0.17	0.31	0.58	1.19
Sex by family history	0.32	0.36	0.37	1.37
Age by family history	0.39	0.16	0.017[a]	1.48
Age by physical activity	0.07	0.07	0.34	1.07
BMI by waist : hip ratio	–0.11	0.06	0.037[a]	0.89
BMI by family history	–0.17	0.09	0.066	0.84
WHR by physical activity	–0.12	0.106	0.25	0.89

[a] Significant; Age in 10-year categories, BMI = units of 2, waist : hip ratio = units of 0.2, family history and sex dichotomised. Physical activity – light, moderate, heavy; SEB, Standard error of coefficient

groups 45 years and older and under 45 years were compared. Odds ratio (OR) for diabetes was higher in older subjects with a positive family history (OR 3.5, 95 % CI 2.32–5.26 vs OR 2.31, CI 1.55–3.42). Positive family history had a greater influence on diabetes in lean subjects (BMI ≤ 25 kg/m^2) compared to overweight subjects (OR 2.6, CI 1.89–3.60 vs OR 1.81, CI 1.13–2.9).

Prevalence of IGT was dependent on the age, BMI and WHR (Table 4). Strong interactions of age and family history and BMI and WHR were evident. The odds of developing IGT were also higher in older subjects, in the presence of positive family history of diabetes (OR 0.76, CI 0.49–1.19) in under 45 years and 1.76, CI (1.03–3.00) in over 45 years. As the results of the analyses, performed separately for men and women, were very similar, they are not shown separately.

Discussion

Serial cross-sectional or prospective studies [1, 2, 13, 15–17] have shown that the prevalence of NIDDM has been rising in almost all populations. However, a striking feature has been the significant and substantial increase in NIDDM in certain minority communities and migratory populations, such as Pima Indians [21], Nauruans [1], Japanese Americans [17], Mexican Americans [22], and migrant Asian Indians [1]. Recent studies showed that urbanization and economic development are causing high prevalence of NIDDM even in the developing countries [10, 13, 15].

In this study in southern India, a 40 % increase in prevalence of NIDDM has been noted over a period of 6 years, from 8.2 % in 1988–1989 to 11.6 % in 1994–1995. No attempt has been made to calculate the incidence of NIDDM as the same individuals

were not tested in these surveys and mortality data was not available. There was an increase in the diabetes prevalence in all age groups significant in the three decades from 35 to 64 years. The age structure of the population had not shown any major change from the 1981 to 1991 census. The data obtained in 1989 gave similar values for prevalence of diabetes and IGT when age-corrected for either of the population census indicating a true increase in the prevalence of NIDDM in the present survey.

Risk factors for NIDDM, regarding age, family history of diabetes, BMI, and WHR did not show any difference from the previous survey. There was a higher prevalence of diabetes in women in this survey. The male : female ratio was 0.82 in this survey while it was 1.06 in the previous survey. Newly diagnosed diabetes was more prevalent among women and this was probably related to the fact that fewer women report for regular medical check-ups than men. Mean BMI and WHR were similar in both surveys. The cause of the rising prevalence of diabetes is unknown. The strong association of NIDDM with BMI and central adiposity was found in men and women, both in the multiple logistic regression and the analysis of prevalence in the tertiles of BMI and WHR in the present survey. As changes in obesity or central adiposity were not seen in the present study it suggested the presence of unidentified, powerful environmental factors, against a background of strong genetic predisposition causing the rising prevalence of diabetes in urban Indians.

Fasting and 2-h plasma insulin values in normal subjects were higher than the values reported in a white population [5]. This was in confirmation with our earlier observation during the 1989 survey suggesting high insulin resistance in an Indian population [23]. As expected insulin responses were higher in IGT but the insulin : glucose ratios were lower. With the expression of clinical diabetes beta-cell function decreased further, as shown by a quantitative reduction in the 2-h insulin value. No comparison of the insulin data was made with the data from the 1989 survey, as the tests were not performed in the same individuals.

A follow-up survey of NIDDM in the modernising Pacific Island population of Western Samoa by Collins et al. [24] conducted 13 years after a first survey in 1978, showed that the prevalence of NIDDM in urban Apia increased from 8.1 to 9.5 % in men and 8.2 to 13.4 % in women. A marked increase in prevalence of NIDDM was found in rural areas also, especially in men. The increase in BMI seen in men and women in the second survey was a strong contributory factor for the increased prevalence of diabetes, although differences in BMI, WHR, physical activity, family history of diabetes or sex did not fully explain the increase in prevalence of diabetes in the Western Samoan population [24].

Asian Indians have been identified as one of the ethnic groups with a high prevalence of NIDDM [1, 13, 15, 16] and high familial aggregation of NIDDM [25, 26]. In other ethnic groups with a high prevalence of NIDDM, such as the Naruans, the prevalence seems to have reached its peak and a further increase has not been seen [27]. On the other hand, populations such as Asian Indians seem to be currently experiencing increasing prevalence of NIDDM. This has been well illustrated in the migrant Indians in Singapore [15]. In Singapore, Cheah and Thai [15] noted a rising prevalence of NIDDM in all ethnic groups between 1984 and 1992. In Chinese it doubled from 4 to 8 %, in Malays it increased from 7.6 to 9.3 %, while in Indians it rose from 8.9 to 12.8 %. The highest prevalence was among the Indians [15].

The ratio between diabetes and IGT is considered an index of the 'epidemic stage' of the population. This has been suggested by King and Rewers [16] in their analysis of a large number of epidemiological studies from different populations. In the present study, the prevalence of IGT showed a simultaneous increase (although not significant) along with an increase in NIDDM. This suggests that along with conversion of IGT to NIDDM there is replenishment of the IGT pool by possible conversion of 'normal susceptibles' to IGT [16, 28]. Therefore, the persistent high prevalence of IGT may be a possible predictor of further increase in NIDDM in this population in the years to come.

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