Clinical Forum

Prevalence of non-insulin dependent diabetes mellitus in Indian migrants in Melbourne, Australia, using fasting plasma glucose

Ibiebele TI[1], Wattanapenpaiboon N[2], Hsu-Hage BH-H[3], Wahlqvist ML[1]

[1]International Health & Development Unit, Faculty of Medicine, Monash University, Melbourne, Australia., [2]Monash Asia Institute, Monash University, Melbourne, Australia., [3]Department of Rural Health, The University of Melbourne, Melbourne, Australia

Abstract

Background: This paper reports the prevalence of NIDDM and its associated risk factors in migrants of Indian ethnic background, who resided in Melbourne, Australia, and were recruited for an Indian Health Study from July 1993 to October 1995. The relationships between fasting plasma glucose and other NIDDM associated factors were investigated, and a comparison of the NIDDM prevalence of this population was made with that of Anglo-Celtic Melbournians.

Methods: A representative group of 297 men and 255 women (aged 25-78 years) was recruited using a method of identification of characteristic surnames in the telephone directory. Participants were considered to be diabetic either if they had been diagnosed by a medical practitioner, regardless whether they were receiving medication for it, or if they had a fasting plasma glucose ≥ 7.8 mmol/L.

Results: The prevalence of NIDDM in the Indian Australians was 10.1% for men and 12.2% for women. No significant gender difference was observed in the study population (χ^2 test $P = 0.44$). The high prevalence of NIDDM of the study population is consistent with the findings of studies of South Asian migrants in other countries. Compared to the Anglo-Celtic population, the prevalence of NIDDM was higher for the Indians. It was also found that fasting plasma glucose was positively associated with age, body fat mass, abdominal circumference, abdomen-to-hip ratio, and fasting insulin, both in men and women.

Conclusions: The Indian migrants in Melbourne, Australia, had a 11.1% prevalence of NIDDM, which was significantly higher than that of the Anglo-Celtic Australians. These findings are in agreement with other studies of South Asian migrants. It is proposed that the mechanism(s) underlying the pathophysiology of the metabolic disturbances in those of Indian ancestry may differ from other ethnic groups, and this may involve the abdominal fat distribution.

Keywords : *Non-insulin dependent diabetes mellitus, Indians, South Asians, fasting plasma glucose, cross-sectional study, abdominal obesity*

Introduction

Epidemiological studies carried out in different countries including Fiji, South Africa, Mauritius and the United Kingdom have shown that people from South Asia who are of Indian ancestry have been observed to have a higher prevalence of non-insulin dependent diabetes mellitus (NIDDM) than other local ethnic groups.[1-8]

Received on: 19th October 1999
Accepted on: 22nd June 2000

Correspondence to : Prof Mark L Wahlqvist, International Health & Development Unit, PO Box 11A, Monash, University VIC 3800, Australia, Telephone:(+61 3) 9905 8145 Facsimile:(+61 3) 9905 8146, E-mail:mark.wahlqvist@med.monash.edu.au

The progressive removal of immigration restrictions in Australia based on country of origin, race or colour, together with the extension of assisted migration schemes to non-British groups and refugees, have ensured a greater diversity among the Australian population.[9] As a result, the numbers of people from South Asia (India, Pakistan, Sri Lanka, Bangladesh) increased by 93%, from 70, 430 in 1984 to 135, 960 in 1994[9]. Indian-born Australians alone, increased from 1,730 in 1984 to 3,128 in 1994 accounting for 49% of arrivals from South Asia[9].

Since the national census data in Australia do not contain morbidity data on NIDDM for migrant populations, little is known about the prevalence of NIDDM in Australians who migrated from South Asia. This paper reports the prevalence of NIDDM and associated risk factors in migrants of Indian ethnic background, residing in Melbourne, Australia. A comparison with a population of Anglo-Celtic Australians in Melbourne, which is considered to be a reference population, is also made.

Subjects and Methods

The Melbourne Indian Health Study (MIHS) was a cross-sectional study investigating the association between food intake, lifestyle and health status, especially obesity and NIDDM, in Australians of Indian ethnic background. This study was part of a cross-cultural project conducted by the Monash Nutrition Research Group, which is currently located in the International Health & Development Unit, Faculty of Medicine, Monash University. In this report, the NIDDM prevalence of migrants of Indian ethnic background were compared with that of 488 Australians of Anglo-Celtic origin, as reported in the Anglo-Celtic Study which was also part of the same project. As these two studies followed a similar protocol including the use of the telephone directory for subject sampling, it is possible for a comparison between populations with different ethnicity to be made. Ethics approval was granted by Monash University Standing Committee for Research in Humans.

The term '*Indian*' or '*South Asian*' is used to denote origin from India, Sri Lanka, Pakistan and Bangladesh or have Indian ancestry, while the term '*Anglo-Celtic*' denotes origin from England, Ireland, Scotland and Wales.

Subject recruitment

Representative samples of the Indian population were recruited from July 1993 to October 1995 by the method of identification of distinctive or characteristic surnames in the telephone directory, as described by Hage *et al.*[10] Lists of common presumptive distinctive Indian surnames were sought from active Indian community leaders, and from telephone directory listings of countries in the Indian sub-continent such as India. The list of presumptive surnames was then used to search for matching surnames in the Melbourne Telephone Electronic White Pages Databases. A total of about 6,000 telephone connections were identified, then one name out of every five names on the list, along with the address and matching telephone number was randomly selected. A sampling list of 1, 200 households was formed for the study.

The study eligibility criteria were: a) aged 25 years or over, b) being of Indian ancestry as judged by grandparental origin and country of birth, and c) being permanent resident or citizen of Australia and residing in the Melbourne Statistical Division at the time of contact. Since ethnicity is a

characteristic willingly taken on by an individual, subjects with one parent or grandparent with Indian ethnicity were considered to be eligible for this study. The study also included all immediate family members living in the same household who met the eligibility criteria.

A letter of invitation to participate in the study, together with background information on the study and a summary of the study procedure were sent to the head of each of the 1,200 households in the sampling list. A telephone call was then made to each of the households to establish eligibility and willingness to participate in the study. After the first phone call, subjects were classified as: a) not eligible, b) eligible and willing, c) eligible but not willing, and d) unable to be contacted.

Information on demography, lifestyle and health status

Information regarding demography, existing health conditions such as already diagnosed diabetes, family history of diabetes, and lifestyle factors such as physical activity levels, was obtained through the use of a self-administered questionnaire which was written in English and designed for the purpose. The questionnaire was mailed to each subject after he/she received background information on the study, and the summary of the study procedure, and the study participation was confirmed over the phone. Subjects were asked to return their completed questionnaire on the day of their appointment for blood tests and body composition measurements.

Physical activity was assessed by modifying the method employed by Dowse *et al*.[11] The physical activity level score was derived by combining leisure activity scores and occupational activity scores. Both leisure and physical activity were categorised on a three-level scale. For example, leisure activities that were house-bound with no regular outside activity such as reading or watching television were given low scores, while those involving active sports such as jogging, weight-lifting or cycling were assigned high activity scores. Occupational activities that require sitting all day such as clerical or shop assistants and general house work were categorised as low level and assigned low scores, while occupations that require standing and moving about such as building labourers or mining were assigned high scores.

Body composition measurements

All anthropometric measurements were carried out at the Clinical Nutrition and Metabolism Unit, Monash Medical Centre, Melbourne, Australia by trained personnel, using the method outlined by Lohman *et al*.[12] Standing height and weight were measured with the subjects in light clothing without shoes. Height was measured to the nearest centimetre using a fixed stadiometer (Medizintechnik Ka, West Germany). Weight was measured to the nearest tenth of a kilogram, using a digital scale of 130 kg capacity (Soehle Digitals). Weight and height were measured three times, and the average of three readings was recorded. Body mass index (BMI) was calculated from height and weight measurements as weight in kilograms divided by height in meters squared. Overweight was defined as BMI greater than 25 and less than or equal to 30, while obesity was defined as BMI greater than 30.[13]

The measurements of circumferences were made using a non-stretchable fibre glass tape to the nearest centimetre at the levels of abdomen (at mid-point between the iliac crest and the lower margin of the ribs with the subject standing) and

hip (at the level of the maximum gluteal extension). Abdominal-to-hip ratio (AHR) was calculated as abdominal circumference divided by hip circumference in centimetres. AHR was used as an indicator of abdominal fatness. Abdominal obesity was defined as AHR greater than 0.95 in men and 0.85 in women.[14,15]

Four skinfold thickness measurements were made to the nearest 0.2 mm on the right side of the body at the biceps, triceps, subscapular and suprailiac areas using the Harpenden Caliper (British Indicators, Luton, UK). The average of two readings was recorded for each site. Skinfold thicknesses at these four sites were used to predict body density and hence body fat, according to sex- and age-specific regression equations.[16]

Biochemical measurements

Subjects were asked to fast for at least 8-12 hours prior to blood sampling, during which time they were allowed to drink water only. Venous blood samples were collected in plain tubes for assays requiring serum, or in tubes containing ethylene diaminetetraacetic acid (EDTA) as an anticoagulant for assays requiring whole blood or plasma. Blood samples were allowed to clot at room temperature for at least two hours and the serum or plasma separated by centrifugation before analysis. Samples not analysed after centrifuging were stored at or below −70°C.

Fasting plasma glucose (FPG) was analysed using an adaptation of the hexokinase- glucose-6-phosphate dehydrogenase method with the Dimension AR Clinical Chemistry Analyser (EI duPont de Nemours, Wilmington, DE 19898 USA). Glycated haemoglobin (HbA1c), an index of glycaemic control, was analysed using the Bio-Rad Variant with a high performance liquid chromatography (HPLC) system. Serum insulin was analysed in one batch using a two-site immuno-enzymometric method with a commercial assay kit AIA-PACK IRI and the TOSOH 1200/600 system (TOSOH Corporation, Tokyo, Japan). All the biochemical analyses were performed by the Department of Clinical Biochemistry, Monash Medical Centre.

NIDDM classification

The recommendations of the World Health Organisation Study Group in 1985[17] were used to classify NIDDM. Subjects were considered to be diabetic if they: *a)* had been diagnosed with diabetes by a medical practitioner but were not receiving medication for it (medically *'untreated'*); *b)* had been diagnosed with diabetes and were taking oral hypoglycaemic agents or insulin (medically '*treated*'); or *c)* had a fasting plasma glucose ≥ 7.8 mmol/L. The crude prevalence rate of NIDDM in this population was age adjusted and standardised to Segi's world population using the direct method,[18] as recommended by King and Rewers.[19]

According to the Department of Clinical Biochemistry at Monash Medical Centre, the HbA1c values between 6.0 and 7.5% were regarded as acceptable values for good blood glucose control, while values greater than 7.5% were indicative of poor glycaemic control.[20] A value of fasting insulin ≥ 20 μmol/L was used to define hyperinsulinaemia, as suggested by both Lind and Chu and their colleagues.[21,22] Following the homeostasis model developed by Matthews *et al*,[23] an insulin-glucose ratio for individual subjects was calculated from FPG and serum insulin levels using the following formulae:[23] insulin-glucose ratio = [insulin/(22.5 e^{-}

$^{\ln(glucose)}$)] or [insulin/(22.5/glucose)]. Because there are no recommended cut-off points to be used as an indicator of insulin resistance, subjects in the present study were categorised as insulin resistant if their insulin-glucose ratio was equal to or greater than the 80th percentile of the study population (5.8 for men and 4.4 for women).

Statistical analyses

Data analyses were performed using the Statistical Analysis System (SAS) software version 6.12 (SAS Institute, Cary, NC, USA). Spearman correlation coefficients (r_s) were used to indicate the relationship between FPG and the risk factors related to NIDDM. Comparisons between diabetic and non-diabetic subjects, and between the Indian and Anglo-Celtic Australians, after adjustment for confounding factors, were made using an analysis of covariance. The significance level was set at 5%.

Results

Study population

None of the participants were Australian-born, and 85% of the study subjects were born in South Asian countries, namely India, Sri Lanka, Pakistan, Bangladesh and Burma. Of 1,200 letters mailed out to all households in the sampling list, 100 were returned undelivered because the addressees had moved. Three hundred households could not be contacted by telephone after five attempts. Of 800 households successfully contacted, only 472 households were eligible. Singhalese-speaking Sri Lankans were not recruited in this study because they did not claim Indian ancestry, resulting in the bulk of the ineligible subjects.

Among those 472 eligible households, 314 were willing to take part in the study. Therefore, the household response or participation rate was 66.5%. It was found that English was the main language spoken at home by more than 50% of the participants, and therefore it was possible to use a self-administered questionnaire written in English for the study. From the 314 households, 552 individuals agreed to have their blood taken for biochemical measurements, 547 had body composition measurements, and only 513 completed the health status and lifestyle questionnaires. The characteristics of the study population are listed in Table 1.

The overall prevalence of NIDDM was 11.1%, and no gender difference was observed (10.1% for men and 12.2% for women, χ^2 test $P = 0.44$). The prevalence of NIDDM was observed to increase with age, particularly in men, as shown in Figure 1. Subjects aged between 55 and 64 years had the highest prevalence compared to other age groups, both in men and women. Compared to the Anglo-Celtic Australians in Melbourne, the Indians had a higher prevalence of NIDDM in every age group, especially in the age groups of 55-64 years, and 65 years and over (Figure 1).

It was found in the present study that about 9% of the male and 7% of the female study population had poor blood glucose control. In addition, 24% of the men and 18% of the women were hyperinsulinaemic and about 20% of the

Table 1: Selected characteristics of study subjects

	Men				Women			
Parameters	n	Mean ± SD	Median	Min – Max	n	Mean ± SD	Median	Min – Max
Age (years)	297	43.7 ± 10.8	41.0	25.0 – 78.0	255	40.4 ± 9.4	39.0	25.0 – 70.0
Anthropometry								
Weight (kg)	264	72.2 ± 10.4	71.6	43.7 – 107.4	231	61.3 ± 10.4	59.9	34.6 – 91.5
Height (cm)	264	170.2 ± 6.4	170.2	152.3 – 188.0	231	156.9 ± 5.8	156.5	143.5 – 173.3
Body mass index (kg/m^2)	264	24.9 ± 2.9	24.7	17.9 – 32.4	231	24.9 ± 4.0	24.3	14.1 – 38.1
% Body fat[a]	264	24.1 ± 3.8	24.0	11.3 – 34.2	231	32.9 ± 3.8	33.1	15.5 – 41.8
Body fat mass (kg)[b]	264	17.6 ± 4.4	17.4	7.3 – 31.3	231	20.4 ± 5.1	20.2	5.4 – 35.7
Abdominal circumference (cm)	264	90.8 ± 8.2	90.5	68 – 116	231	85.4 ± 9.8	85.0	55.0 – 114.0
Abdominal-hip-ratio[c]	264	0.94 ± 0.05	0.94	0.76 – 1.08	231	0.87 ± 0.07	0.87	0.73 – 1.07
Biochemical parameters								
Fasting plasma glucose (mmol/L)	297	5.5 ± 2.0	5.0	2.8 – 22.8	254	5.2 ± 2.2	4.7	3.2 – 26.0
Glycated hemoglobin (%)	296	6.3 ± 1.4	6.0	3.9 – 17.3	254	6.1 ± 1.0	5.9	3.2 – 13.1
Fasting insulin (mU/L)	292	18.7 ± 19.9	12.3	2.0 – 124.9	251	19.2 ± 32.7	12.0	2.1 – 33.4
Insulin-glucose ratio[d]	292	3.5 ± 3.6	2.4	0.3 – 27.3	250	3.4 ± 3.8	2.5	0.5 – 27.1

[a] % Body fat was estimated from the sum of the four skinfold thicknesses.[16]
[b] The body fat mass in kilograms was calculated as [(% body fat/100) x body weight].
[c] Abdominal-hip ratio = abdominal circumference/hip circumference.
[d] Insulin-glucose ratio was calculated as [insulin/(22.5/glucose)].

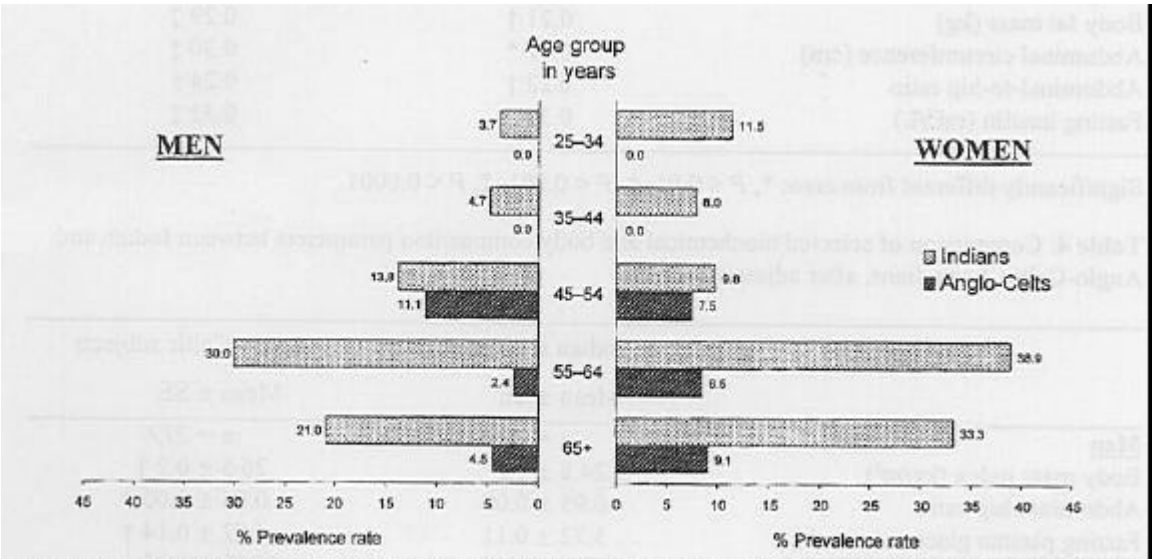

Fig 1: Prevalence rate of non-insulin dependent diabetes mellitus by age group in Indian (297 men, 255 women) and Anglo-Celtic (210 men, 261 women) Australians in Melbourne, Australia

Fig 1: Prevalence rate of non-insulin dependent diabetes mellitus by age group in Indian (297 men, 255 women) and Anglo-Celtic (210 men, 261 women) Australians in Melbourne, Australia

Table 2: Comparison of selected characteristics between diabetic and non-diabetic subjects

	Diabetics	Non-diabetics
	Mean ± SE	Mean ± SE
Men	*n = 30*	*n = 262*
Age (years)	52.8 ± 1.9	42.7 ± 0.6 †
Fasting plasma glucose (mmol/L)	10.1 ± 0.2	5.0 ± 0.1 †
Glycated hemoglobin (%)	9.4 ± 0.2	5.9 ± 0.0 †
Fasting insulin (mU/L)	22.2 ± 3.6	18.3 ± 1.2
Body mass index (kg/m^2)	25.2 ± 0.6	24.8 ± 0.2
Body fat mass (kg)	18.7 ± 0.9	17.5 ± 0.3
Abdominal circumference (cm)	93.7 ± 1.7	90.5 ± 0.5
Abdominal-hip-ratio	0.98 ± 0.01	0.94 ± 0.00 †
Women	*n = 30*	*n = 225*
Age (years)	44.4 ± 1.7	39.9 ± 0.6 *
Fasting plasma glucose (mmol/L)	8.1 ± 0.3	4.7 ± 0.1 †
Glycated hemoglobin (%)	7.6 ± 0.2	5.9 ± 0.1 †
Fasting insulin (mU/L)	43.3 ± 5.9	16.0 ± 2.1 †
Body mass index (kg/m^2)	25.8 ± 0.7	24.7 ± 0.3
Body fat mass (kg)	20.8 ± 0.9	20.3 ± 0.4
Abdominal circumference (cm)	87.4 ± 1.8	85.1 ± 0.7
Abdominal-hip-ratio	0.89 ± 0.01	0.87 ± 0.00

Significant different from the diabetic group: *, $P < 0.05$; †, $P < 0.0001$.

Table 3: Spearman correlation coefficients (r_s) describing the relationships between fasting plasma glucose (mmol/L) and factors associated with NIDDM

Parameters	Men (n = 297)	Women (n = 255)
Age (years)	0.29 ‡	0.32 ‡
Body mass index (kg/m^2)	0.10	0.29 ‡
Body fat mass (kg)	0.21 †	0.29 ‡
Abdominal circumference (cm)	0.17 *	0.30 ‡
Abdominal-to-hip ratio	0.22 †	0.24 †
Fasting insulin (mU/L)	0.30 ‡	0.32 ‡

Significantly different from zero: *, $P < 0.01$; †, $P < 0.001$; ‡, $P < 0.0001$.

Table 4: Comparison of selected biochemical and body composition parameters between Indian and Anglo-Celtic Australians, after adjusting for age

	Indian subjects	Anglo-Celtic subjects
	Mean ± SE	Mean ± SE
Men	*n = 297*	*n = 219*
Body mass index (kg/m^2)	24.8 ± 0.2	26.5 ± 0.2 †
Abdominal-hip-ratio	0.95 ± 0.00	0.94 ± 0.00 *
Fasting plasma glucose (mmol/L)	5.72 ± 0.11	4.82 ± 0.14 †
Glycated hemoglobin (%)	6.41 ± 0.08	5.49 ± 0.10 †
Women	*n = 255*	*n = 269*
Body mass index (kg/m^2)	25.3 ± 0.27	24.95 ± 0.26
Abdominal-hip-ratio	0.89 ± 0.00	0.89 ± 0.00
Fasting plasma glucose (mmol/L)	5.43 ± 0.12	4.68 ± 0.11 †
Glycated hemoglobin (%)	6.23 ± 0.08	5.44 ± 0.08 †

Significant different from the Indian counterparts: *, $P < 0.05$; †, $P < 0.0001$.

Table 5: Comparison of standardized prevalence (%) of NIDDM in Melbourne South Asians to South Asians elsewhere[a]

Country where studied	Men	Women
South Africa	14.4	20.8
Mauritius	17.8	13.2
Singapore	22.7	10.4
Fiji	23.0	20.3
Australia (the present study)	12.3	15.8

[a] Data on standardized prevalence rate of NIDDM for Indians or South Asians studied elsewhere taken from King and Rewers.[19]

male study subjects were insulin resistant. This prevalence may be higher since diabetics on medication would possibly show good blood glucose control.

Fasting plasma glucose and other NIDDM associated factors

Subjects with diabetes were observed to be older, and, as expected to have, a higher FPG and HbA1c, compared to those without diabetes (Table 2). No significant differences were observed between the diabetic and non-diabetic groups in abdominal circumference, BMI and body fat mass. Diabetic men, however, had a higher AHR, and diabetic women had higher fasting insulin, compared to their non-diabetic counterparts.

As shown in Table 3 population FPG was positively associated with age, abdominal circumference, body fat mass, BMI (in women only) and AHR.

Comparisons between Indian and Anglo-Celtic Australians

After age was adjusted for, the Indians were found to have a higher FPG and HbA1c, compared to their Anglo-Celtic counterparts (Table 4). A higher AHR, but lower BMI was observed in Indian men. However, there was no significant difference in AHR or BMI for the women between these two populations.

Discussion

The study population of Indian migrants had the age-standardised prevalence rate of NIDDM of 12.3% for men and 15.8% for women, within the high prevalence category (11-20%) according to King and Rewers.[19] This prevalence rate was more or less the same as those observed in South Asians in Fiji[1,2], South Africa[3,4] and Mauritius[5] (Table 5).

Epidemiological studies of NIDDM in migrant Indian or South Asian populations have consistently shown that they have a much higher prevalence of diabetes compared to other ethnic groups living in the same community.[1-8] In Fiji, the crude prevalence rate of NIDDM in urban South Asians (13% for men and 11% for women) were much higher than the urban Melanesian population (4% for men and 7% for men).[1, 2] Likewise, studies in South Africa showed that the crude prevalence rate of NIDDM was 10% in South Asians compared to 4% in both whites and Africans.[3,4] Again, other studies conducted in the United Kingdom found similar results.[6-8] The prevalence of known NIDDM in South Asians was reported to be 3.8 times higher than that of the Europeans.[6,7] These observations were supported by the finding in the present study of a higher NIDDM prevalence in migrants of Indian ethnic background, compared to an Anglo-Celtic Australian population at every age group. As shown in the Figure 1, NIDDM was common in the Indian population, both in men and women, across the five age groups, while in the Anglo-Celtic population, NIDDM was uncommon before age 45.

Several epidemiological studies have reported an increase in the prevalence and incidence of NIDDM with age.[24, 25] In the present study, it was observed, that in men, the prevalence of NIDDM increased with advancing age, and reached a peak between 35 and 64 years. However, a different pattern was observed in women. The highest prevalence was observed in those between the ages of 25 and 34 years. The prevalence decreased in the remaining reproductive years of women. The presence of gestational diabetes in women in this age group should not be ruled out, as it would have resulted in the high prevalence of diabetes observed.

Many risk factors have been described for NIDDM, including a family history of NIDDM, physical inactivity, obesity and abdominal fat distribution. In the present study, it was however observed that those with or without a family history of diabetes had a similar FPG, both in men and women. One of possible explanations for this is that subjects with a family history of diabetes could have been more cautious, and able to maintain a low blood glucose level. In terms of physical activity, since over 50% of men had a moderate physical activity level, and over 55% of women had a low physical activity level, an effect of physical activity level on FPG could be not be demonstrated, using

statistical means, in this study population.

The finding that FPG was strongly associated with AHR, but not with BMI, in the Indian men, is consistent with other studies reporting that individuals with abdominal obesity have a greater risk of developing diabetes compared to those with general or overall obesity.[26, 27] It has been known that obesity, particularly abdominal obesity, has a causative effect in the development of NIDDM,[28] and in addition, women with this 'android' type of fat distribution have been shown to have a higher prevalence of insulin resistance, hyperlipidaemia and hypertension.[29] It is likely that the distribution of fat in Indian men is mostly intra-abdominal, as there is convincing evidence that individuals with central obesity are prone to develop insulin insensitivity, glucose intolerance, hypertriglyceridaemia and related disorders of hypertension and atherosclerosis.[30] In women, hyperinsulinaemia and insulin resistance, in addition to general and central obesity, appeared to be important in the expression of NIDDM. It is proposed that the mechanism underlying the pathophysiology of the metabolic disturbances in Indian men and women may differ from other ethnic groups.

Identification of naming patterns of ethnic minorities with distinctive surnames has been considered to be a method reliable enough to facilitate random sampling of relevant minority groups.[31, 32] This method along with the use of the telephone directory has been successfully employed to obtain representative samples from Chinese and Greek migrant populations in Australia.[10,33] Use of population registers was not possible for this study as they do not exist in Australia, and electoral rolls would introduce bias as they contain names of only those who have Australian citizenship. Like most developed countries, Australia has a high telephone user service, it is therefore suitable to use the identification of distinctive or characteristic surnames with the use of the telephone directory in the present study. However, there may be an unintentional exclusion of individuals with non-Indian surnames, or females with non-Indian surnames as a result of marriage to non-Indian males. Furthermore, it is worth mentioning that those with unlisted telephone numbers were missed out, and the number of this group of people was unknown.

Although it was found that 50% of the study population spoke English at home, and the use of English in the information sheets provided to the participants and in the self-administered questionnaire, may be appropriate, it was not possible to identify those who refused to participate in the study due to the language problem. Such a problem may cause bias in subject recruitment.

While the 1985 WHO classification criteria[17] are important in that they provide a standardised method for the diagnosis of diabetes, many studies have reported that the oral glucose tolerance test has a higher specificity and a greater sensitivity than FPG.[19,34,35] Nevertheless, the use of the oral glucose tolerance test would have posed difficulties for the study subjects, including multiple venipuncture, and prolonged visits to the survey site, and which could have resulted in a low response or participation rate. It is important to note that this study aimed to estimate the prevalence of NIDDM in Indian Australians in comparison with other populations in Melbourne. It was required that a similar protocol was followed for the cross-sectional studies,

thereby allowing comparison of data across populations of different ethnicity. It is very likely, we believe, that with a more sensitive test, the prevalence of NIDDM in the study population would be much higher.

From the point of view of public health, the high prevalence of NIDDM observed in the present study could suggest a higher cost of health care services in Australia with time. It is important to note that since the present age distribution of the Indian population in Australia skews towards the young,[9] a high prevalence of NIDDM in the young age group would lead to a large number of diabetics, when they get older. Demographic shifts will also increase the public health impact of the high prevalence of NIDDM and its complications. Therefore, there is the need for preventive and treatment programs to reduce NIDDM and its complications in the population of Indian ethnic background.

In conclusion, the present study has found that Indian migrants in Melbourne, Australia had a high prevalence of NIDDM, and also confirms the observations previously reported of a higher NIDDM prevalence in the Indian population, compared to other ethnic groups in the same community wherever they are studied.

Acknowledgments

The Melbourne Indian Health Study is part of a research project partially funded by the Victorian Health Promotion Foundation, Australia. The authors are grateful to the investigators of the Anglo-Celtic Study for allowing the use of their data, and to the Monash University Post-graduate Publication Awards Committee for providing Dr TI Ibiebele with financial support during the preparation of the manuscript.

References

1. Zimmet PZ, Taylor R, Ram P, King H. Prevalence of diabetes and impaired glucose tolerance in the (Melanesian and Indian) population of Fiji: a rural-urban comparison. Am J Epidemiol 1983;118:673-688.

2. Tuomilehto J, Ram P, Eseroma R, Taylor R, Zimmet P. Cardiovascular diseases and diabetes mellitus in Fiji: analysis of mortality and morbidity and risk factors. Bulletin of the World Health Organisation 1984;62(1):133-143.

3. Omar MAK, Seedat MA, Dyer RB, Rajput MC, et al. The prevalence of diabetes mellitus in a group of South Asian Indians. S Afr Med J 1985;67:924-926.

4. Omar MAK, Seedat MA, Dyer RB, Motala AA, et al. South Asian Indians show a prevalence of NIDDM and bimodality in plasma glucose distribution patterns. Diabetes Care 1994;17(1):70-73.

5. Dowse DG, Gareeboo H, Zimmet PZ, Alberti KG, et al. High prevalence of NIDDM and impaired glucose tolerance in Indians, Creole and Chinese Mauritians. Diabetes 1990;39:390-396.

6. Mather HM, Keen H. The Southall diabetic survey: Prevalence of known diabetes in Asians and Europeans. BMJ 1985;291:1081-1084.

7. Mather HM, Keen H. Prevalence of known diabetes in Asians and Europeans. BMJ 1986;292:621-622.

8. Simmons D, Williams DRR, Powell MJ. Prevalence of diabetes in a predominantly Asian community: preliminary findings of the Coventry diabetes study. BMJ 1989;298:18-21.

9. Australia Bureau of Statistics Catalogue No.1301.0. Year Book of Australia 1996.

10. Hage B H-H, Graeme Oliver R, Powles JW, Wahlqvist ML. Telephone Directory Listings of Presumptive Chinese Surnames. An Appropriate sampling frame for a Dispersed Population with Characteristic

Surnames. Epidemiology 1990;1(5):405-408.

11. Dowse GK, Zimmet PZ, Gareeboo H, Alberti KGMM, et al. Abdominal obesity and physical inactivity as risk factors for NIDDM and impaired glucose tolerance in Indians, Creole, and Chinese Mauritius. Diabetes Care 1991;14(4):271-282.

12. Lohman TG, Roche AF, Martorell R. (eds.) Anthropometric Standardization Reference Manual (Abridged). Human Kinetic Publishers, Illinois, 1988.

13. National Health and Medical Research Council. Report on the one hundredth session. Canberra: AGPS, 1985.

14. Depres JP. Assessing obesity. Beyond BMI. NIN Review. 1992;7:1-4.

15. Shera AS, Rafique G, Khwaja IA, Ara J, et al. Pakistan National Diabetes Survey: Prevalence of glucose intolerance and associated factors in Shikapur, Sindh Province. Diabetes Medicine 1995;12:1116-1121.

16. Durnin JVGA, Womersley J. Body fat assessed from total body density and its estimation from skinfold thicknesses: measurement on 481 men and women aged from 16-72 years. Br J Nutr 1974;32:77-97.

17. WHO Study group: Diabetes Mellitus. Technical Report Series No. 727. Geneva, World Health Organisation, 1985.

18. Armitage P, Berry G. Statistical methods in medical research. New York, Wiley. 1981.

19. King H, Rewers M. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. Diabetes Care 1993;16(1):157-177.

20. Martinez-Triguero MI, Salvador A, Samper MJ, Almela M, et al. Lipoprotein (a) and other risk factors in patients with non-insulin dependent diabetes mellitus. Coronary Artery Dis 1994;(9):755-760.

21. Lind H, Nilsson P, Holthuist N, Lindholm L. Non obese men with high lipoprotein (a) values: a cardiovascular risk group different from those with the metabolic syndrome? Scan J Clin Lab Invest 1994;54:177-183.

22. Chu NF, Lee MMS, Wang DJ, Chen LM, et al. The interrelationship between impaired glucose tolerance and other risk factors for cardiovascular disease: Is it a predictor for cardiovascular disease? J Clin Epidemiol 1994;47(5):485-493.

23. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, et al. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentration in man. Diabetologia 1985;28:412-419.

24. Verma NPS, Mehta SP, Madhu S, Mather HM, Keen H. Prevalence of known diabetes in an urban Indian environment: the Darya Ganj diabetes study. BMJ 1986;293:423-424.

25. Horton ES. NIDDM - the devastating disease. Diab Res Clin Pract 1995;28(Suppl):S3-S11.

26. McKeigue PM, Pierpoint T, Ferrie JE, Marmot MG. Relationship of glucose intolerance and hyperinsulinemia to body fat pattern in South Asians and Europeans. Diabetologia 1992;35:785-791.

27. Haffner SM, Mitchell BD, Hazuda HP, Stern MP. Greater influence of central distribution of adipose tissue on incidence of non-insulin dependent diabetes in women than men. Am J Clin Nutr 1991;53:1213-1317.

28. Felber JP. From obesity to diabetes. Pathophysiological considerations. Int J Obes Rel Dis 1992;16:937-52.

29. Schmidt MI, Duncan BB, Canani LH, Karohl C, Chambless L. Association of waist-to-hip ratio with Diabetes Mellitus. Strength and possible modifiers. Diabetes Care 1992;15:912-14.

30. Robertson DA, Singh BM, Nattrass M. Impaired glucose intolerance in obesity is associated with insensitivity to insulin in multiple aspects of metabolism as assessed by a low dose incremental insulin infusion technique. Diabetic Med 1991;8:718-725.

31. Ecob R, Williams R. Sampling Asian minorities to assess health and welfare. J Epidemiol Community Health 1991;45:93-101.

32. Chatuverdi N, McKeigue PM. Methods for epidemiological surveys of ethnic minority groups. J Epidemiol Community Health1994;48:107-111.

33. Rutishauser IE, Wahlqvist ML. Food intake pattern of Greek migrants to Melbourne in relation to duration of stay. Proc Nutr Soc Austr 1983;8:49-55.

34. Haffner SM, Rosenthal M, Hazuda HP, Stern MP, Franco LJ. Evaluation of three potential screening tests for diabetes mellitus in a biethnic population. Diabetes Care 1984;7:347-353.

35. Finch CF, Zimmet PZ. Determining diabetes prevalence: a rational basis for the use of fasting plasma glucose concentration. Diabetic Med 1990;7(7):603-610.