# Epidemiology of Diabetes in Asians of the Indian Sub-continent*

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## SUMMARY

Asians from the Indian subcontinent have received greater attention in diabetes studies because of their migration in large numbers. The prevalence of non-insulin-dependent-diabetes (NIDDM) in migrant Indians is higher than that in the population residing in the Indian-subcontinent and is also usually higher than in the other racial groups in the host country. However before drawing any conclusions with reference to the high prevalence of NIDDM in the migrant Indians, careful comparisons are required with more up-to-date information available from the Indian subcontinent itself. Recent data from India indeed indicate that the prevalence rates have either been underestimated in the past or are rising. The problem is compounded by the different diagnostic criteria used for defining diabetes. Some of the possible factors which cause variations in the rates of NIDDM in this population are discussed.

## I. INTRODUCTION

The history of diabetes mellitus in India dates back to the sixth century AD when Hindu physicians recognized it as being caused by an overindulgence in oil and sugar and were also able to identify the two separate types. It was called madhumeh (sweet urine) or bahumoothra (excess urination) and was colloquially identified as sugar disease (1).

A higher frequency of diabetes in the native population on the east coast of India compared with Eurasians, Jews, Americans, Europeans, and Americans was recognised before the turn of the century (1). Although authentic population data were not available before the 1950s, clinical reports (2-4) suggest that the prevalence of diabetes was high by that time especially in middle-aged, affluent and obese Indians (5,6).

## II. DIABETES MELLITUS SUBTYPES

Major geographic and ethnic differences exist in the prevalence and incidence of both insulin-dependent diabetes (IDDM) and non-insulin-dependent diabetes (NIDDM). There is a greater than 35-fold difference in the risk between countries having the highest and lowest incidence of IDDM (7). Similarly, there are major variations in the prevalence and incidence of micro-and macrovascular complications of diabetes between countries (8,9). IDDM (type 1 diabetes) is relatively uncommon in Indians, in the black population of sub-Saharan Africa, in the Japanese, and indeed in most non-caucasians (10). However, the question if whether IDDM is rare in developing countries still remains unanswered as there is a probability of early death without diagnosis of the diabetes; this would account for the low rates which are mostly based on hospital data (11).

### A. Insulin-Dependent Diabetes Mellitus

Although IDDM is rare in developing countries, there are differences in the relative rates between the countries, varying from 0.2 to 20% of all diabetic patients (12). This has been attributed to difference in the diagnostic criteria and bias in population sampling, especially in relation to the age on onset of diabetes (13). In India, the prevalence of IDDM varies, and no definitive reports of IDDM in migrant Indians have been forthcoming up to now (Table 1).

### B. Non-Insulin-dependent Diabetes Mellitus

This is much the commoner form of diabetes and results form a combination of genetic and environmental factors as suggested by studies in the pacific and other countries where rural vs. urban and migrant vs. non-migrant group gradients have been noted (8,14). In some developing countries and in some ethnic groups, a high prevalence and incidence of NIDDM have been found (15,16). This form of diabetes was rare while these populations lived a subsistence and traditional life. With modernization of lifestyle, either by modernization in the city or as a result of migration to an urban centre or industrialized country, the frequency of NIDDM rose. Some of the possible factors which cause variations in the rates are listed in Table II and discussed below.

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**Table I**
**Prevalence of Insulin-Dependent Diabetes Mellitus (IDDM) in Asian Indians**

| Year | Ref. | Place of Study | Population studied | Diagnostic criteria | Age group (years) | Prevalence (%) |
|---|---|---|---|---|---|---|
| 1965 | 20 | Cuttack, India (1961-1963) | Diabetes patients admitted in hospital or treated at home (urban) | Clinical records* | < 40 | 4.2 |
| 1987 | 80 | Bardoli, India | All hospital admissions (urban) | OGTT in those with post-prandial blood glucose Levels > 7.8 mmol/l | All ages | 0.7 |
| 1986 | 78 | Bhadran, India (1983) | House to house (rural) | OGTT with 75g glucose load in glycosurics | 11-20 | 0.06 |
| 1984 | 201 | Leicester U.K. | Population utilizing health services (urban) | Records | 0-15<br>0-9<br>10-15 | 0.05<br>0.03<br>0.1 |
| 1989 | 46 | Dares Salaam, Tanzania | House to house (urban) | Blood glucose WHO criteria (1985) | > 15 | 0.1 |

* Classified clinically on the basis of young age on onset (< 40 years), lean, highly susceptible to ketosis with high insulin sensitivity and no response to oral drugs.

### C. Malnutrition-Related Diabetes Mellitus (MRDM)

Diabetes related to malnutrition has received greater importance since the inception of this entity as a special type of diabetes in the World Health Organisation classification (7). MRDM, for research purposes, has been further divided into two subclasses: (1) fibrocalculous pancreatic diabetes (FCPD), and (2) protein-deficient pancreatic diabetes (PDPD). However, there has been disagreement on the classification and on the casual relationship. Both MRDM and "tropical diabetes" are clouded with confusion (17,18). Present studies in India indicate that they constitute a negligible proportion of diabetes mellitus. An analysis of 3100 case records at Madras, India showed diabetes associated with pancreatic calculous disease (FCPD) in only 0.4% of the cases (19). Others report a prevalence rate for MRDM in parts of India of 22.9% of all diabetes (20), although the picture is complicated by the common occurrence of the young-onset form of NIDDM (MODY) (Table III). Of the diabetics below the age of 30 years, 44% required insulin in south India (21). These subjects did not respond to oral hypoglycaemic agents, though they were not ketosis-prone; were not malnourished; had BMI values of more than 18; and there was no relationship to socio-economic status. Many clinical features of MRDM (such as severe emaciation, a protuberant abdomen, and signs of avitaminosis) are common to both the subtypes (FCPD and PDPD) despite difference in the geographical and racial origins of the patients (17). Twenty-five percent of the patients in Madras, India (22) were found to be underweight as opposed to 52% found in another study in India (23), 50% in Bangladesh (24) and 68% in Sri Lanka (16). The cassava/malnutrition hypothesis, although attractive has yet to be substantiated and indeed looks extremely doubtful,. Particularly in Africa (McLarty et al, unpublished data). There has also been no evidence so far of a genetic predisposition (17). The presence of islet cell antibodies is in dispute (25,26).

### D. Impaired Glucose Tolerance

Impaired glucose tolerance (IGT) replaced the confusing terms of chemical, borderline, subclinical, symptomatic, and latent diabetes in the WHO classification (27-29). Subjects in this category are not at risk microangiopathy, but are at risk of large vessel disease and coronary heart disease in association with factors such as obesity, serum lipoprotein abnormalities, and haemostatic factors (30-38). Although the risk of diabetes is greater in those with IGT than in normal subjects, a large proportion revert to normoglycaemia (28-29). The

**Table II**
**Causal Factors in Diabetes Mellitus in Asian Indians**

Geographic regions—within Indian subcontinent

Migration—internal:rural—rural
rural—urban
—external

Ethnic susceptibility?

Genetic factors —paternal influence
—inbreeding
—HLA heterogeneity

Diet —interpopulation differences
—vegetarian/non-vegeterian
—rice eating/wheat and other cereal eating

Socio-cultural factors —social class
—occupation
—ethnic culture

Physical activity —rural population
—urban population

Obesity

Stress

Age

Sex

Parity?

Insulin resistance

proportion of subjects with IGT progressing to diabetes over a 10- year period varies from 13 to 52 % with a rate of progression of 1-5% per year (27). In all studies, the baseline blood glucose concentration was the most powerful and consistent predictor of subsequent diabetes (8), with the roles of other factors such as BMI and the insulin response to a glucose load remaining controversial (40a). Much of the variability is probably due to the well-known imprecision the OGTT (40b). there are very few studies from India (41) assessing the prevalence rates of IGT using 1985 WHO criteria, (7) with most of the studies being migrant Indians (Table IV) (42-46) Figures ranged from 6% in South Africa to 25% in female Muslims in Dar es Salaam, all higher than in India itself. The seemingly low figures in Coventry (U.K.) are presumably due to initial screening values being set too high.

## III. PREVALENCE OF DIABETES ON THE INDIAN SUBCONTINENT

### A. Prevalence of Diabetes Mellitus and Geographical Differences

Some of the published studies (41,47-84) on the prevalence of diabetes mellitus in India are summarized in Table V. Ideally, the studies should be grouped on the basis of the diagnostic criteria adopted, but a critical literature search showed that no two studies had similar criteria for diagnosis (excluding the duplicate and incomplete publications) (refs. 55 and 85; 75 and 86; 65 and 87; 83 and 88). Thus, the tabulated results are the general prevalence figures from various corners of India. The few studies where the 1985 WHO Expert Committee's diagnostic criteria (7) were used (41,84) are indicated.

As anticipated, the prevalence of diabetes mellitus is higher in surveys advertised as diabetes detection camps screening the risk groups (54,61) and in hospital based studies (70). Within the same state in India, e,g, Andhra Pradesh, the prevalence varies from 2.5% (age > 10 years) to 5.3 (age > 15 years) (59,66,77). The prevalence may vary within rural villages where heterogeneous populations co-exist as known in Ganghavathi (84) where natives had a prevalence of 2.2% compared with rural-rural

**Table III**
**Classification of Ninety-Six Diabetics Seen in Cuttack, India20**

| Type | Number of patients hospitalized (n=55) | Domiciliary* (n=41) | Total* (n=96) |
|---|---|---|---|
| Juvenile | 2 (3.6%) | 2 (4.9%) | 4 (4.2%) |
| Elderly, obese | 7 (12.8%) | 38 (92.6%) | 45 (46.9%) |
| Elderly, lean | 24 (43.7%) | 2 (4.9%) | 26 (27.0%) |
| MRDM ("J" type) | 22 (39.8%) | 0 | 22 (22.9%) |

*One case with features of IDDM at onset changed over to manifest characteristic of NIDDM after a period of remission. He has been included in both, explaining the small excess above 100 in total percentage.
Reproduced with kind permission from ref.20.

**Table IVa.**
**Prevalence of Impaired Glucose Tolerance (IGT) in Asian Indians in India**

| Year | Ref. | Place of Study | Population studied | Diagnostic criteria | Sex | Age (years) | Prevalence (%) |
|---|---|---|---|---|---|---|---|
| 1984 | 77 | Tenali (1981) | House to house (urban) | 75 g OGTT-2h blood glucose > 7.2-10 mmol/l WHO criteria (1965) | M & F | > 15 | 3.3 |
| 1986 | 78 | Bhadran (1983) | House to house (rural) | 75 g OGTT on glucosurics WHO criteria (1980) | M & F | > 10 | 0.33 |
| 1987 | 80 | Bardoli (1979) | Hospital admissions (urban) | OGTT in those with post-prandial blood glucose levels > 7.8 mmol/l* | M & F | All ages | 0.37 |
| 1988 | 41 | Kudremukh (urban) | Clinic registered | WHO criteria (1985)** | M & F | > 20 | 2.3 Male<br>2.0 Female<br>2.0 Overall |

*Represents "latent diabetes" (hyperglycaemia and glycosuria associated with stress) and "chemical diabetes" (hyperglycaemia without glycosuria on oral OGTT).
** 2h post-prandial plasma glucose concentration 7.8-11.1 mmol/l.

migrants with a prevalence of 9.1%. Little information is available from the north-west desert region of Rajasthan state; the north-eastern states of Assam, Bihar, Manipur, Nagaland; and from Pakistan.

Diabetes is more prevalent in the urban than in the rural populations. In Orissa state, India (65), the prevalence of diabetes (age > 10years) adjusted for WHO criteria (7) was 2% in urban Cuttack and 0.5% in rural Badachana (89). In the Indian council of Medical Research (ICMR) study, an oral glucose tolerance test was done in urban and rural population samples of similar size in Ahmedabad, Calcutta, Cuttack, Delhi, Pune and Trivandrum in those aged 15 years and above (75). The urban prevalence rate varied from 0.9% in Delhi to 3.7% in Ahmedabad. In the rural areas, it varied from 1.1% in Pune to 1.9% in Ahmedabad. The overall prevalence rate was 2.2%, with 3.0% in urban and 13% in rural areas. For the whole of India the prevalence was 1.7%. Further analysis of the Ahmedabad data showed no correlation between caloric intake and prevalence of diabetes in the urban population, in contrast to the rural population where there was an inverse relationship (90). Khan and Ibrahim in Bangladesh have found similar results. Further studies are necessary to explain this relationship between undernutrition (in calories and protein), body mass index and the occurrence of diabetes (12).

More recently, Ramachandran et al (41), have shown a high prevalence of diabetes in an urban population in south India using WHO diagnostic criteria (7). The overall prevalence of diabetes was found to be 5% in the age group 20 years and above. The surprising outcome was that when the age of study population was adjusted to the age distribution of the Indians living in Southall, London and in Fiji, the prevalence increased to 10 and 9% respectively. Similar findings were obtained in the Daryaganj survey in New-Delhi(79), where the prevalence of known diabetes was found to be similar to that seen in a migrant Indian population in Southall, London (92). These studies show that the high prevalence of diabetes seen in migrant Indians (see Table VI) is now also seen in India, suggesting an ethnic susceptibility to diabetes which is revealed with exposure to common environmental factors.

Among the religious groups in India, parsees had the highest prevalence of diabetes followed by Christians and Hindus (93). The higher prevalence in Christians was attributed to the improvement in their socioeconomic status after converting to Christianity.

On the basis of available data, the prevalence of diabetes in Bangladesh is about 1.6% of the total population (12).

**Table IVb.**
**Prevalence of Impaired Glucose Tolerance in Migrant Asian Indians**
**(compared with other local ethnic populations where data are available)**

| Year | Ref. | Country | Population Studied | Sex | Age (years) | Prevalence (%)* in Indians | Prevalence (%)* in others |
|---|---|---|---|---|---|---|---|
| 1983 | 42 | Fiji | Urban and rural Indians and Melanesians | Male-rural | > 20 | 10.2 | 5.7 Melanesian |
| | | | | Male-urban | > 20 | 8.3 | 7.3 Melanesian |
| | | | | Female-rural | > 20 | 9.6 | 8.5 Melanesian |
| | | | | Female-urban | > 20 | 11.8 | 13.2 Melanesian |
| 1985 | 43 | South Africa | Urban Indians | Male and female | > 15 | 6.0 | |
| 1986 | 44** | Trinidad | Urban Indians, Africans, Europeans, mixed | Male | 35-69 | 10.0 | 5.8 Africans<br>4.3 Europeans<br>7.4 mixed |
| | | | | Female | 35-69 | 11.6 | 15.8 Africans<br>10.9 Europeans<br>14.3 Mixed |
| 1989 | 45 | Mauritius | Urban Hindu Indians, Muslim Indians, Creole, Chinese | Male | 25-74 | 12.6 (Hindu)<br>11.1 (Muslim) | 15.4 Creole<br>13.6 Chinese |
| | | | | Female | 25-74 | 19.7 (Hindu)<br>19.5 (Muslim) | 19.6 Creole<br>19.3 Chinese |
| 1989 | 46 | Tanzania | Urban Muslim | Male | > 15 | 18.1 | 7.8 Rural Africans |
| | | | Indians | Female | > 15 | 25.1 | 7.7 Rural Africans |
| 1989 (unpublished) | | Tanzania | Urban Hindu Indians | Male | > 15 | 15.6 | |
| | | | | Female | > 15 | 17.8 | |
| 1989 | 202*** | Coventry, U.K. | Urban Indians | Male | 20-79 | 1.1 | 1.9Europeans |
| | | | Europeans | Female | 20-79 | 1.5 | 1.6Europeans |

All studies used the 1985 WHO criteria with 75g glucose load unless otherwise indicated.
*Age-standardized prevalence rates.
**50 g glucose load was used with WHO comparable cut-off 2 h blood glucose values. IGT-2 h whole venous blood concentration of 6-8.9 mmol/l.
***Initial screening done.

## IV. PREVALENCE OF DIABETES IN MIGRANT ASIANS FROM THE INDIAN SUBCONTINENT

Asians from the Indian subcontinent have received greater attention in diabetes studies because of their migration in large numbers before and after colonization. The prevalence of diabetes in migrant Indians was initially found to be higher than in the population residing in the Indian subcontinent (68,94) and is also usually higher than in the predominant racial group or other racial groups in the host country (Table VI) (14,42,44,46,95,96). Also, wherever they are, they appear to have a higher morbidity and mortality from coronary heart disease than the indigenous population. This has been reported from countries with long-established Indian populations such as Singapore (97), Fiji (98), South Africa (99), Uganda (100), Trinidad (44) and the United Kingdom (101,103).

Migration (a move from one environment to another, be it external or internal) may lead to an increase in the prevalence of NIDDM in a number of ethnic groups going in parallel with social and cultural chances(14). The environmental factors may unmask

**Table V**
**Epidemiological Studies of the Prevalence of Diabetes Mellitus in India**

| Year | Place | Sample | Population | Blood glucose urine sugar | Age (years) | Prevalence (%) |
|---|---|---|---|---|---|---|
| 1938 | Calcutta (47) | 96 300 | Hospital records (r&u) | u.s | All | 1.0 |
| 1959 | Bombay (48) | 48 572 | Hospital records (r&u) | nm | Adults | 0.7 |
| 1960 | Multicentre (49) | 168 779 | Hospital records (r&u) | u.s | All | 0.8 |
| 1963 | Bombay (50) | 18 243 | Exhibition (r&u) | u.s | All | 1.5 |
| 1964 | Lucknow (51) | 1 446 | Hospital based (u) | bl.gl. | All | 2.3 |
| 1964 | Vellore (52) | 63 356 | Hospital records (r&u) | bl.gl. | All | 2.5 |
| 1966 | Bombay (53) | 3 200 | Textile workers | bl.gl. | nm | 2.1 |
| 1966 | Bombay (54) | 1 207 | Cancer centre (r&u) | bl.gl. | > 20 | 2.2 |
| 1966 | Chandigarh (55) | 3 846 | House to house (u) | u.s | All | 2.9 |
| 1966 | Delhi (56) | 11 216 | Hospital records | nm | nm | 2.3 |
| 1966 | Delhi (57) | 1 027 | Risk groups (u) | bl.gl. | All | 26.4 |
| 1966 | Hubli (58) | 21 232 | Hospital records (r&u) | nm | All | 2.2 |
| 1966 | Hyderabad (59) | 21 396 | Exhibition (r&u) | u.s. | > 20 | 4.1 |
| 1966 | Jabalpur (60) | 4 000 | Hospital based (r&u) | u.s. | All | 1.7 |
| 1966 | Madras (61) | 5 030 | Diab camps(u) | u.s. | All | 5.6 |
| 1966 | Pondicherry (62) | 2 694 | House to house (u) | bl.gl. | > 5 | 0.7 |
| 1966 | Trivandrum (63) | 45 267 | Hospital records (r&u) | nm | All | 8.7 |
| 1966 | Varanasi (64) | 2 572 | House to house (u) | u.s. | > 10 | 2.7 |
| 1971 | Cuttack (65) | 2 447 | House to house (r&u) | bl.gl. | > 10 | 1.2 |
| 1972 | Hyderabad (66) | 847 | House to house (r) | bl.gl. | > 10 | 2.5 |
| 1972 | Hyderabad (67) | 2 006 | House to house (r) | u.s. | > 20 | 2.4 |
| 1972 | New-Delhi (68) | 2 783 | House to house (r&u) | bl.gl. | > 15 | 2.3 |
| 1973 | Bangalore (69) | 25 273 | House to house (u) | bl.gl. | > 5 | 2.3 |
| 1973 | Calcutta (70) | 593 | Hospital based (u) | bl.gl. | > 10 | 12.7 |
| 1973 | Lucknow (71) | 2 190 | Army personnel (u) | bl.gl. | All | 1.1 |
| 1974 | New Delhi (72) | 2 291 | Army personnel (u) | bl.gl. | > 20 | 2.7 |
| 1975 | Calcutta (73) | 4 000 | Mobile clinic (r&u) | bl.gl. | > 15 | 2.3 |
| 1979 | Madurai (74) | 9 670 | House to house (u) | bl.gl. | > 4 | 0.5 |
| 1979 | Multicentre (75) | 3 516 | House to house (u) | bl.gl. | > 15 | 3.0 |
| 1979 | Multicentre (75) | 3 495 | House to house (r) | bl.gl. | > 15 | 1.3 |
| 1984 | Pondicherry (76) | 1 982 | House to house (r) | u.s. | > 15 | 1.8 |
| 1984 | Tenali (77) | 848 | House to house (u) | bl.gl. | > 15 | 4.7 |
| 1986 | Bhadlan (78) | 3 374 | House to house (r) | bl.gl. | > 10 | 3.8 |
| 1987 | Bardoli (80) | 1 348 | Hospital admissions (r&u) | bl.gl. | All | 4.4 |
| 1988 | Kudremukh (41)* | 678 | Clinic based (u) | bl.gl. | > 20 | 5.0 |
| 1988 | Rewa (82) | 15 000 | House to house (u) | bl.gl. | nm | 1.9 |
| 1989 | Gangavathi (84)* | 765 | House to house (r) | bl.gl. | > 30 | 2.2 |
| | Rural-rural migrants | 529 | House to house | bl.gl. | > 30 | 9.1 |

Nm: Not mentioned in the publication; r:rural; u:urban; u.s.:urine sugar; bl.gl.:blood glucose.
*studies done using WHO criteria (1985).

**Table VI**
**Prevalence of Diabetes Mellitus in Migrant Asian Indians**
**(compared with other local ethnic population where data are available)**

| Year | Ref. | Country | Population studied | Diagnostic criteria | Sex | Age (years) | | Prevalence(%) Male | Female | Tot |
|---|---|---|---|---|---|---|---|---|---|---|
| 1958 | 163 | Trinidad | Oil company workers Indians, Negroes, mixed | Fasting and post-meal glucose tolerance done. if glycosurics, then OGTT done | M&F | > 14 | Indians | 2.3 | 1.0 | 1.7 |
| | | | | | | | Negroes | 1.4 | 1.5 | 1.4 |
| | | | | | | | Mixed | | | 0.4 |
| 1962 | 112 | British Guiana | House to house Indians, Africans | OGTT on all with post-meal glycosuria | M&F | > 14 | Indians | 2.1 | 1.9 | 2.0 |
| | | | | | | | Africans | 1.6 | 2.8 | 2.2 |
| 1967 | 164 | Fiji | House to house Indians, Melanesians | OGTT on all with post-meal glycosuria | M&F | > 21 | Indians | | | 5.7 |
| | | | | | | | Melanesians | | | 0.6 |
| 1968 | 199 | Trinidad | House to house Indians, Negroes, mixed, others | 100 g OGTT on all glycosurics | M&F | All ages | Indians | 2.5 | 2.3 | 4.5* |
| | | | | | | | Negroes | 1.0 | 2.1 | 2.5* |
| | | | | | | | mixed | 1.2 | 1.5 | 4.4* |
| | | | | | | | others | 5.3 | 1.1 | 3.1* |
| 1969 | 130 | South Africa | House to house Indians, Malays, Africans | 50 g OGTT on all glycosurics | M&F | > 15 | Indians | | | 10.4 |
| | | | | | | | Malays | | | 6.6 |
| | | | | | | | Africans | | | 3.6 |
| 1975 | 95 106 | Singapore | House to house Indians, Chinese, Malays | OGTT in all with post-prandial glycosuria WHO 1965 Criteria | M&F | >15 | Indians | 8.1 | 3.1 | 6.1 |
| | | | | | | | Chinese | 1.7 | 1.4 | 1.6 |
| | | | | | | | Malays | 2.7 | 2.2 | 2.4 |
| 1983 | 42 | Fiji | House to house, urban and rural Indians, Melanesians | WHO 1985 criteria | M&F | >20 | Indians | 12.9 | 11.0 | Urban |
| | | | | | | | Melanesians | 3.5 | 7.1 | Urban |
| | | | | | | | Indians | 12.1 | 11.3 | Rural |
| | | | | | | | Melanesians | 1.1 | 1.2 | Rural |
| 1985 | 43 | South Africa | House to house | WHO 1985 criteria | M&F | >15 | Indians | | | 11.1 |
| 1986 | 44 | Trinidad | House to house Indians Africans Europeans Mixed | WHO 1980 criteria** | M&F | 35-69 | Indians | 19.5 | 21.6 | |
| | | | | | | | Africans | 8.2 | 14.8 | |
| | | | | | | | Europeans | 4.3 | 10.2 | |
| | | | | | | | Mixed | 8.2 | 6.7 | |

NIDDM in a genetically susceptible individual, and it appears to be a disease associated with a changing lifestyle including increased longevity, dietary changes from traditional foods, and increased stress (104). The prevalence of diabetes is also higher in other migrant groups, such as the Japanese in Hawaii (105), the Chinese and Malays in Singapore (106), the Yemenites in Israel (107), the West Indians in the U.K. (108) and the Tokelauns in New Zealand (109). Close observation made on ethnic groups in Hawaii showed a higher prevalence of diabetes (4.9%) in pure race Hawaiians than in mixed race Hawaiians (2.7%) (110).

**Table VI Cont'd**
**Prevalence of Diabetes Mellitus in Migrant Asian Indians**
**(compared with other local ethnic population where data are available)**

| Year | Ref. | Country | Population studied | Diagnostic criteria | Sex | Age (years) | | Prevalence(%) Male | Female | Tot |
|---|---|---|---|---|---|---|---|---|---|---|
| 1987 | 197 | Singapur | House to house Indians, Chinese, Malays | WHO 1985 criteria | M&F | All ages | Indians | 13.4 | 5.1 | 8.9 |
| | | | | | | | Chinese | 4.6 | 4.9 | 4.0 |
| | | | | | | | Malays | 9.5 | 7.3 | 7.6 |
| | | There were no cases of diabetes below the age of 20 in any of the groups | | | | | | | | |
| 1988 | 114 | East London U.K. | House to house Bangladeshis Europeans | WHO 1985 criteria | M&F | 35-69 | Bangladeshis | 22.0 | 23.0 | |
| | | | | | | | Eruopeans | 10.0 | 4.0 | |
| 1989 | 202 | Coventry, U.K. | House to house Indians, Europeans | WHO 1985 criteria after screening | M&F | > 20 | Indians | 11.2 | 8.9 | |
| | | | | | | | Europeans | 2.8 | 4.3 | |
| 1989 | 45 | Mauritius | House to house Hindu Indians, Muslim Indians, Creoles, Chinese | WHO 1985 criteria | M&F | 25-75 | Hindu Indians | 14.0 | 10.9 | 12.4 |
| | | | | | | | Muslim Indians | 12.7 | 13.8 | 13.3 |
| | | | | | | | Creoles | 7.7 | 13.0 | 10.4 |
| | | | | | | | Chinese | 13.5 | 9.5 | 11.5 |
| 1989 | 46 | Tanzania | House to house Muslim Indians | WHO 1985 criteria | M&F | > 15 | Muslim Indians | 7.0 | 7.6 | 7.1 |
| | | | | | | | Rural Africans | 1.1 | 0.7 | 1.1 |
| | | | | | | | Urban Africans | | | 1.9 |
| 1989 (unpublished | | Tanzania | House to house Hindu Indians | WHO 1985 criteria | M&F | >15 | Hindu Indians | 10.1 | 9.2 | |

Prevalence rates shown for all studies done from 1983 have been age-adjusted to the respective country's population. All subjects studied were from urban areas unless otherwise mentioned.
*Overall rates are age group above 20 years.
** Diabetes defined by cut-off value for 2 h whole blood glucose > 8.9 mmol/1.

West (111) summarized diabetes mellitus as high in all migrant Indian populations expect those in British Guiana (112). However, Weinstein (112) had included a larger proportion of young population in migrants than in natives (1790 migrants and 370 natives below the age of 30 years), resulting in an apparently high figure being projected for natives as compared with migrants.

Before the drawing and conclusions with reference to the high prevalence of diabetes in migrant Indians, careful comparisons are required with the information available in India. The problem is compounded by the different diagnostic criteria used for defining diabetes and age structure of the study populations. Recent data from India indeed indicate that the prevalence rates have either been underestimated in the past or are rising (41,79).Thus the pattern of known diabetes has been found to be similar in Asian inhabitants of Southall, London (92), a middle-class suburb of New-Delhi, India (79), and the rural population of Eluru, South India (83), although these were not studies of total diabetes, rather of known subjects.

**Table VII**
**Age and Sex-Specific Prevalence Rates of Diabetes Mellitus in Asian Indians as Seen in Several Studies Using the Same Diagnostic Criteria (WHO, 1985)**

| Age group (years) | Fiji[42] (%) | Mauritius[45*] Hindu Indians(%) | Mauritius[45*] Muslim Indians (%) | Tanzania Muslim Indians(46) (%) | Tanzania Hindu Indians** (%) | India[41***] (%) |
|---|---|---|---|---|---|---|
| **Male** | | | | | | |
| 15-24 | 0 (67) | -- | -- | 0 (44) | 0 (97) | 0 (46) |
| 25-34 | 1.7 (119) | 4.3 (422) | 3.6 (110) | 5.2 (96) | 5.3 (98) | 3.0 (134) |
| 35-44 | 14.3 (77) | 12.9 (356) | 4.1 (96) | 9.3 (118) | 9.4 (164) | 8.5 (118) |
| 45-54 | 36.4 (55) | 24.5 (208) | 28.1 (57) | 9.8 (112) | 10.7 (172) | 25.7 (35) |
| 55-64 | 36.4 (44) | 23.7 (152) | 34.5 (29) | 13.3 (75) | 22.4 (81) | 33.3 (9) |
| 65** | 23.8 (21) | 27.7 (83) | 15.4 (13) | 15.8 (38) | 20.4 (52) | 25.0 (4) |
| **Female** | | | | | | |
| 15-24 | 0 (82) | -- | -- | 1.4 (70) | 0 (82) | 0 (86) |
| 25-34 | 2.6 (152) | 3.4 (464) | 2.2 (138) | 0.8 (122) | 2.8 (115) | 3.3 (150) |
| 35-44 | 7.8 (90) | 7.6 (367) | 8.3 (96) | 3.7 (134) | 7.1 (162) | 8.9 (67) |
| 45-54 | 31.3 (80) | 15.2 (217) | 15.9 (63) | 12.2 (148) | 12.2 (162) | 22.2 (18) |
| 55-64 | 40.0 (40) | 25.6 (160) | 37.0 (46) | 21.7(69) | 14.3 (73) | 50.0 (8) |
| 65** | 26.3 (19) | 25.4 (114) | 47.8 (23) | 39.1 (23) | 27.9 (47) | 33.3 (3) |

The figures in parentheses are the numbers of subjects in that particular age group.
*Age group studied was 25-74 years.
**Unpublished data
***Age group studied was > 20 years.

In Fiji (42), rural and urban Indians had similar prevalence rates of diabetes (12.1 vs.12.9% for males; 11.3 vs. 11.0% for females) but these rates were much higher than those in the Melanesian population (1.1 vs. 3.5% for males;1.2 vs.7.1% for females).However, the age-standardized prevalence rates of IGT between the Indians and Melanesians, both rural and urban showed no statistical difference.

In South Africa (43), the overall prevalence in the urban Indians in Durban of IGT and diabetes mellitus was 6 and 11.1% respectively, compared with a diabetes prevalence of 3.6% in Whites and 4.1% in Africans (12), In Trinidad in the 35-69 year age group, the prevalence of diabetes mellitus by ethnic groups was the highest in Indian men and women (19.5 and 21.6%) when compared with Africans (8.2 and 14.8%), Europeans (4.3 and 10.2%) and those of mixed descent (8.2 and 6.7%) (44). The rates of IGT were similar in females of all descent, while Indian men had the highest prevalence in that gender.

The prevalence rates of diabetes mellitus have been reported to be higher in Indians compared with other ethnic groups in Singapore and Malaysia (113). Similarly, Bangladeshis residing in East London, UK. have a diabetes prevalence rate of 22% compared with 8.3% in non-Asians residing in the same area.

In Mauritius, (45) the prevalence rates of IGT and diabetes mellitus did not differ much between Indian Hindus (16.2 and 12.4%), Indian Muslims (15.3 and 13.3%), Creoles (17.5 and 10.4%), and Chinese (16.6 and 11.9%). The absence of any significant differences between the ethnic groups was attributed to the exposure of all the communities to common environmental factors.

Findings were even more striking in Tanzania, (46) where the prevalence rates of impaired glucose tolerance and diabetes mellitus in Indian Muslim were 21.5 and 7.1% respectively, compared with 16.7 and 9.6% in Indian Hindus (unpublished data). These rates were much higher than in the urban and rural indigenous Africans population (7.7% IGT and 0.9% diabetes in the rural population and 0.7% diabetes in the urban population (115, 116). The prevalence rate of IGT of 21.5% in Indian Muslims recorded in Tanzania is the highest ever recorded in this group.

All the studies done in the migrant Indian Hindu or Indian Muslim communities apart from Tanzania

**Table VIII**
**Surveys of Known Diabetes Mellitus in Asian Indians Using Questionaries**

| Year | Ref. | Place of study | Population Studied | Sex (years) | Age (%) | Prevalence |
|---|---|---|---|---|---|---|
| **In India** | | | | | | |
| 1986 | 79 | Darya Ganj, New-Delhi (urban) | House to house | M & F | All ages<br>< 30<br>> 40<br>40-64 | 3.1 Crude<br>0<br>9.1<br>8.6 |
| 1986 | 81 | New-Delhi (urban) | Hotel employees | M & F | All ages<br>< 30<br>> 40 | 1.8 Crude<br>0<br>4.5 |
| 1989 | 83 | Eluru, South India<br>1 town (urban)<br>4 villages (rural) | House to house | M & F<br><br><br>Overall (crude)<br> | All ages<br><br><br><br>> 40 | <br>1.5 urban<br>1.9 Rural<br>1.6<br>6.1 |
| **In migrant Indians** | | | | | | |
| 1985 | 92 | Southhall, U.K. (urban) | House to house | M & F | All ages<br>< 30<br>> 40<br>Overall (Age adjusted) | 2.2 Crude<br>0.1<br>9.0<br>4.6 |
| 1988 | 200* | Leicester, U.K. | Clinical records | M & F | All ages<br>< 30<br>> 45 | 2.4 Crude<br>0.1<br>16.0 |

*No questionnaire was used.

have considered them as one homogenous group, which is inappropriate. Indians differ in ethnicity, religion, place, of origin (different parts of India), and diet. In Dar es Salaam, Tanzania, the Hindu community numbers about 11800 but it made up of 20 different subcommunities, each with its own individual characteristics. Seven of these subcommunities have been investigated and we found the prevalence rates of IGT and diabetes to vary from 8.4 to 37.3% and from 6 to 16.9% respectively (unpublished data).

Hence, the differences in the prevalence rates observed in these migrant Indians in different countries and in different parts of India could well be due to differences between communities. Further studies are required to investigate these differences.

## A. Genetic Factors

Vishwanathan et al (117) have shown a high prevalence of diabetes among offspring of conjugal NIDDM parents in India. Diabetes was present in 50% of offspring and 12% had impaired glucose tolerance (IGT) according to the National Diabetes Data Group criteria (118). A large proportion of diabetic subjects in India have a family history of diabetes in first-and second-degree relatives. There is a greater paternal influence in the transmission of NIDDM. Jones et al (119), recorded family histories in 43% of older European and in 21% of Indian diabetics in Kuala Lumpur. Positive family histories were noted in 45% of migrant Indians in South Africa (120), and they were more frequent (55%) in Muslims from India. In Tanzania (46), a family history of diabetes was present in 25% of Indian Muslims above the age of 34 years, and those with a family history of diabetes had twice the prevalence of diabetes compared with those with a negative history. The Mauritius study (45) also sowed a positive correlation. Another study in India (121) has recorded positive family histories in 21% of the first-degree relatives of newly detected non-obese NIDDM subjects.

Recent studies from South Africa (122) and south India (123) have shown that NIDDM in Indian patient occurs at a younger age when compared with European populations, as an explanations for which is not clear. It may be that the genetic

**Table IX**
**Known vs Unknown Diabetes Mellitus in Asian Indians**

| Place of Study | Sex | Age (years) | Number | Prevalence (%) New | Known | Ratio new known |
|---|---|---|---|---|---|---|
| Multicentre study, India(75) | M & F | > 15 | | | | |
| Urban | | | | | | |
| Rural | | | 3516 | 1.4 | 1.6 | 0.9 |
| Bhadran, India(78)-rural | | | 3495 | 1.2 | 0.1 | 13.3 |
| Kudremukh, south India(41)- | M & F | > 10 | 3374 | 1.7 | 2.1 | 0.8 |
| Urban | M & F | > 20 | 678 | 5.0 | 1.9 | 2.6 |
| South Africa(96) | | | | | | |
| Natal-urban | M & F | > 10 | 2427 | 4.2 | 1.8 | 2.3 |
| Cape Town-urban | M & F | > 15 | 1520 | 6.6 | 4.3 | 1.5 |
| Tanzania(46)-urban | | | | | | |
| Muslim Indians | M & F | > 15 | 1049 | 3.5 | 5.2 | 0.7 |
| Hindu Indians* | M & F | > 15 | 1264 | 3.2 | 3.2 | 1.0 |
| Coventry, U.K.(202)-urban | M | 20-80 | 1087 | 2.7 | 5.8 | 0.5 |
| | F | | 1196 | 1.6 | 4.0 | 0.4 |

*Unpublished data

mechanism are stronger in Indians. Since NIDDM is the only type of diabetes in which simple autosomal inheritance has been implicated, it could be more frequent in populations where the disease is largely inherited or, alternatively, the younger age at diagnosis may be related to the younger age structure of the general population in these countries (124). It should be noted that younger onset is also associated with a family history of diabetes in Caucasian NIDDM patients. Although a weak associations between HLA antigens and NIDDM has been reported in several populations, its significance is minimal when compared with that seen with IDDM (104). Examples are Xhosas in South Africa (125), Pima Indians in North America (126), Polynesian (127) and Indian (128) diabetics in the Pacific. In Caucasoid populations, no reports of an association between HLA antigens and NIDDM have been reported (129).

Major differences in the prevalences of NIDDM have been demonstrated between populations of different ethnic origins living in the same geographical locality; e.g., Indians and Melanesians in Fiji (42); Chinese, Malays, and Indians in Singapore (106); Indians, Malays, and Bantu in South Africa (130); and Caucasoids, Pima Indians and Mexican Americans in the U.S.A (131). All these populations show a heightened genetic susceptibility to NIDDM and the disease is no doubt unmasked by environmental changes which include urbanization, dietary changes and physical inactivity (129, 132).

Inbreeding of populations is associated with an increased incidence of inborn errors (133). Discussions on a inbreeding and NIDDM would be incomplete without a note on the South Indians. Population inbreeding is a feature among the Dravidians in south India, with only a negligible percent of marriages occurring between different castes. Consanguineous marriages have been rooted in this system for centuries and they were reported in as many as 47% of the families in a population study (134). This could well be contributed to the higher prevalence of diabetes in South India (41), as well as in the Tamil Indian community in South Africa (135). Anthropological evidence suggests that India is the meeting point for three races-Caucasoid, Australoid, and Mongoloid. Admixture, however did not extend down to South India. Northern India is the easternmost, outpost of the Caucasoid racial region. Thus, heterogeneity in HLA types among the populations within India would be expected in view of the race specificity of HLA systems (136). Racial characteristics are preserved within South India to a greater extent probably because of endogamous practices. With such practices, and thus the higher degree of inbreeding coefficients (137), it is possible that there is persistence of “lethal genes” which are responsible for the higher frequency of the disease.

The absence of urban-rural differences in migrant Indians (42,138) and the higher rates observed in rural-rural settlers within south India (84) all support the underassessed issue of inbreeding. Preliminary observations (unpublished) show higher inbreeding coefficients among diabetic subjects in India. Further indirectly built corroborative evidence is the higher frequencies of diabetes mellitus in small island populations such as Fiji (138), Malta (139), Mauritius (140) and Nauru (141), where it could be attributed to the apparent effect of inbreeding. The higher prevalence of diabetes in the small inbred religious groups of Parsees in the north-west region of India also provides support (93). In the isolated tribal populations in the Ganjam district of Orissa state, India, the rates were also higher, with glycosuria being present in 4.8% of those above the age of 30 years (142).

## B.Diet

Indian lifestyle habits, especially diet, might be invoked to explain the increased susceptibility to glucose intolerance based on Neel's thrifty genotype hypothesis (143,144). Since the first WHO Expert Committee opined that the undernutrition protects populations against diabetes (145), attempts have been made to assess this in population surveys. However, none has proved an effect, partly because there are problems involved in defining leanness. Leanness also implies the efficacy of the subject to utilize calories efficiently as well as physical activity. Diet may contribute to the development of diabetes in two ways: quantitatively, by supplying calories and if activity is low by resultant obesity and qualitatively by the effects of specific foods (14). The long term effects of intermittent starvation on the course of glucose tolerance are not known. However it should be noted that some populations in India consumes very little protein on some days and an alternate starvation-excess cycle exists (146). Whether time constitutes a pathological metabolic stress is not known.

Interpopulation differences exist in both diet and the socio-cultural factors both within and outside the subcontinent. Available data suggest that diet is the main outstanding difference (75). NIDDM prevalence rates are higher on the east coast of Andhra Pradesh, India where rice has been grown and consumed for centuries. Within this state the prevalence rates in the rice-eating population of Eluru (83) and Tenali (77) were higher compared with urban (59) and rural Hyderabad (67) where wheat preparations (roti) form the basic staple food.

The dietary pattern, eating and methods of cooking vary in different parts of India (147). However, with migration, the traditional dietary pattern changes with an increase in the consumption of "modern" foods and a transition from a subsistence way of life to the "feast" situation in the urban centers in a short time (144), which in western societies has extended over many generations leading to genetic adjustments (14).

Traditionally, Hindu Indians are pure vegetarians, but with modernization the diet has become more lactovovegetarian. In addition to the use of meat, fish, and poultry, there is moderate use of eggs, dairy products, and a relatively high intake of whole grain cereals, vegetables, fruit and vegetables oils (148).

Hence they consume less saturated fat and cholesterol, and more polyunasaturated fat and vegetable fibre (149). Although the hypothesis proposing that a vegetarian diet reduces the risk of developing diabetes has been put forward (150-152) in the multicentre Indian study (75) diabetes was more prevalent in vegetarians (2.1-2.8%) than in non-vegetarians (<2%). In Tanzania, Hindu Indians also had a higher prevalence of diabetes than Muslim Indians who are non-vegetarians (unpublished data). A recent study in the United States (153) comparing Asian Indian and American vegetarians has shown that Asian Indians have higher insulin levels, higher plasma glucose levels and lower insulin binding to erythrocytes after a glucose load than Caucasoid Americans, suggesting an increased risk of developing NIDDM, The contribution of diet, therefore, to the increased prevalence of diabetes in Asian Indians is still hazy and more nutritional studies are required to known the specific contribution of diet, independent of obesity to the pathogenesis of NIDDM.

## C. Physical Activity

In 1895, Bose (2) reported diabetes as being more common among the richer class in India, who considered it a pride and honour to lead an indolent life. Exercise is probably a protective factor against the development of diabetes (111) and experimental evidence supports the value of physical exercise (14). There is increased insulin sensitivity in peripheral tissues, especially in muscle (154). Quantitative measurement of physical activity is however, difficult and existing methods are crude (104). The interaction of diet, exercise, and obesity is complex, and it is difficult to isolate exercise and to study it as a single factor in relation to diabetes prevalence and incidence (104). Migration is usually accompanied by changes in all three (155), and it is possibly the loss of these "protective" factors rather than adopted environmental risk which contributes to the higher rates in migrants (45). Recent studies in migrant Indians in Fiji (132) have shown an association of NIDDM with sedentary physical activity independent of obesity. The same was found in Mauritius

(unpublished observations). In Indian Muslims in Tanzania (46), after controlling for obesity the increased prevalence of NIDDM was attributed to physical inactivity and genetic factor. Rural-rural migrants within south India had higher prevalence for diabetes than did indigenous populations, and obesity sedentary activity, higher socio-economic status, and hypertension were more common in migrants than in the native population (84). However Zimmet et al (42) were unable to explain the lack of differences in the high prevalences of diabetes in rural and urban Indians in Fiji, suggesting that genetic factors were important than environmental factors in this ethnic group.

### D. Obesity

In 1980, the second WHO expert committee on Diabetes concluded that the most powerful risk factor for NIDDM was obesity (28). The two most important aspects are its extent and duration (111). In the western world, two-thirds or more of patients with diabetes are obese (156) as compared with NIDDM subjects in India where obesity is not common (124). Previous studies have shown both a strong (28,157,158) and a weak (41,159-162) relationship between obesity and NIDDM. A possible reason is the confounding influence of weight loss with onset of disease or with therapy (104). Ahuja (161), from comparisons between India and Indians in Malaysia and South Africa concluded that the differences in diabetes prevalence in Indians abroad could not be explained on the basis of adiposity. Zimmet et al (42) could not explain the high prevalence of diabetes in the Fiji Indian population on the basis of BMI. Indians in Cape Town, South Africa were more overweight than migrants from Malaysia (130) whereas obesity was less frequently seen in Indians in Singapore than in the Malays and Chinese (106). However it should be pointed out that the migrant Indians in a few studies were mainly from northern India (92,163), whereas in others they were from south India (95,164). Hence inter-ethnic differences have to be accounted for.

Nicholl et al (162) could not attribute the markedly higher diabetes prevalence in U.K. Asians as compared with Europeans solely to the greater degree of adiposity in the former group. In South Africa, there was no correlation between BMI and fasting or 2 h plasma glucose levels over the entire range of BMI in Indians thereby negating the role of obesity as a risk factor for glucose tolerance. Omar et al (43) therefore thought that the effects on glucose tolerance became significant only after a certain threshold of BMI, i.e. 25 or 27. In rural-urban comparisons of diabetes prevalence also, the high prevalence rates in the urban population cannot be explained on the basis of obesity as seen in Western Samoan Polynesians, Puerto Rican men (104) and more recently, indigenous Africans in Tanzania (116). Ramachandran et al (41) found that only a quarter of the diabetes were obese in an urban quarter of the south India. Studies of migrant Indians in South Africa (43), Mauritius (45) and Tanzania (46) however have shown a positive association of obesity with diabetes, especially among females.

Bjorntorp and Sjostrom (165) and Salans et al (166) have emphasized that insulin insensitivity and hyperinsulism with associated impaired tolerance of glucose and triglycerides correlate primarily with fat cell size rather than with either fat cell number or total body fat. These tend to improve with caloric restriction and disappear once weight reduction corrects the hypertrophy (167). Intra-abdominal fat cells measured by waist-hip ratio have been implicated in the pathogenesis of NIDDM (168). The waist-hip ratio correlates with glucose intolerance in Mauritius Indians independent of BMI (45). There is no evidence, however, that differences in diabetes prevalence between Indian subcontinentals and other racial groups.

Identical twin studies suggest that the genetic component in NIDDM acts independently of obesity and therefore there may be additional powerful environmental factors in the urban lifestyle operating independently of obesity which contribute to a high degree of diabetes prevalence in urban dwellers and migrants (8,14,104).

### E. Stress

No generally recognized definition exists for the term stress (111) Even more difficult is the measurement of stress. Hence, it is very difficult to assess the contribution of stress in precipitating diabetes in people with genetic susceptibility of the disease (8,14).

In migrants, the changes in diet, customs, physical activity, and socio-economic status bring potential stresses of varying degrees of magnitude. Urban stress is more marked in ethnic minorities than in local populations (169). The role of stressed and constitution as causative factors in diabetes in Indians has been discussed more in the older literature than at present (170,171). there is increasing interest in the concept of central nervous system control of insulin secretion, and with recent developments, a greater understanding of the interaction of stress, the nervous system, appetite control, and diabetes will be available (104).

### F. Age

The prevalence of diabetes increases with age in India, as elsewhere (Table VII) (111). The maximum incidence of diabetes is in the third, fourth, and fifth decades. Many previous studies underreported diabetes since they lacked age standardization. In most studies, the prevalence doubles if the results of populations below 30 years are excluded. Thus, the prevalence becomes 9.1% in Lucknow (51), 5.5% in Cuttack (87), 3.8% in Hyderabad (66) and 1.4% in Madurai (74) for populations above 30 years of age. Ramachandran et al (41) in an urban population in south India found prevalence rates of diabetes of 21% in the age group over 40 years and 41% in the age group 55-64 years. In the multicentre Study in India (75), 93% of diabetics from urban areas and 81% of diabetics from rural areas were above the age of 30. The Ahmedabad centre (90) showed a high prevalence in those above 60 years of age (16.5% in the urban and 5.3% in the rural samples) with maximum prevalence rates from the fifth decade onwards. An analysis of the data from diabetic subjects between the ages of 15 and 30 years in the age-standardized multicentre study in India (75) showed significantly higher numbers in the rural areas than in the urban areas (29 of 399 vs. 44 of 233; $p<0.0001$). Similar differences in the younger age groups were also present in the results from Orissa (65). This is an alarming situation as 70% of the population in India is rural with a large proportion under the age of 30 years (172).

NIDDM in the elderly constitutes approximately 20% of total diabetes (173) Omar et al (43) found a positive correlation between plasma glucose and age with a high prevalence of diabetes mellitus after the third or fourth decade in South African Indians. The prevalence of diabetes in Southall Asians in the U.K. aged 40-64 years was at least five times higher than in corresponding Europeans in the U.K. (92) Studies in migrant Indians in Mauritius (45) and Tanzania (46) have also shown a positive correlation between age and plasma glucose.

### G. Sex

Populations studied show that diabetes is more frequent in men than in women in India (75,84,174) and in migrant Indians in other countries (94,123,175,176). Recently, however, Campbell (120) and Omar et al (43,177) in South Africa and Ramachandran et al (41) in south India have shown a female preponderance in Indian diabetic subjects. They attribute these findings to increased frequency of obesity in females as compared with males. In Mauritius (45), even though the overall age standardized prevalence of diabetes mellitus was similar in men (12.1%) and women (11.7%) of the four ethnic groups combined, Hindu and Chinese men had a higher prevalence than their female counterparts, whilst the rates were similar in Muslims; Creole men (7.7%) had a significantly lower prevalence than women (13.0%) (Table VII).

In the Indian Muslim population in Dar es Salaam, Tanzania (46), the age-adjusted diabetes rates were similar in both sexes (7 and 7.6%, respectively). In the Mauritius and Dar es Salaam studies, females had higher BMI values than did males. These findings concur with the suggestions made by Vinke et al (178) that the predominance of the women can be explained by their greater obesity.

### H. Parity

High parity has been suggested as a causative (179) or an important provoking factor (180-182) in the development of diabetes. Most of these studies were hospital-based; population-based studies have failed to confirm the association, although a positive association was found in an English community (183). More recently, the Multicentre Study in the U.K. (175) showed no significant differences between nulliparous and parous women in relation to their age, obesity, or fasting blood glucose.

In South India, the impact of multiparity on the prevalence of diabetes is negligible (184). Although a significantly higher prevalence was noted in urban women with more than five children, such results were not reproduced in the rural populations in the same study. In countries where the fertility rates are high, parity without obesity may not be an important causal factor. (184) Parity has no independent effect on glucose tolerance, but increasing parity may be associated with being increasingly overweight, as seen in migrant Indians in Tanzania (46) and consequently with increasing glucose intolerance.

## V. KNOWN VS UNKNOWN DIABETES

In recent years, several surveys have been conducted in India to assess the prevalence of known diabetes (79,81,83). Using similar questionnaires, the results were compared with those of urban Indians in Southall, London (Table VIII) (92). These studies will, however, underestimate the magnitude of the problem, depending on the health awareness, literacy, and the availability of health care to the populations interviewed.

The ratio of known to unknown diabetes varies widely (Table IX) (41,75,78). In India, one unknown diabetic was present for each known diabetic among 3516 adults in the city; whereas in the rural areas (n=3495), there were 13 unknown diabetics (75). In

the rural-rural migrant survey in India, the ratio of known to unknown diabetics were 1:1 in the migrants and 1:3 in the local populations (84). Therefore extrapolation of the results of the questionnaire surveys to determine the prevalence is difficult. In the Mauritius study (45), for each ten known cases of diabetes there were 14 men and 13 women who were newly diagnosed during the survey. In Indian Muslims, the ratio of new to known cases was 1.1 for both men and women as compared with Indian Hindus with ratios of 1.6 and 1.3, respectively.

## VI. HYPERINSULINAEMIA AND INSULIN RESISTANCE

Bonara et al (185) have reported a wide haterogeneity in plasma insulin response to an oral glucose load in mild glucose intolerance. Lillioja et al (186) in their longitudinal and cross-sectional studies in Pima Indians have put forward a hypothesis that the development of NIDDM in obese populations is a two stage process involving insulin resistance followed by beta cell failure.

There is limited information on insulin secretion in both normal and diabetic Indian subjects (8), with most of the studies reported so far being from South Africa (187-190). According to Keller et al (187) the mean fasting and post-prandial insulin levels were significantly higher in Indians than in Blacks and Caucasians in South Africa. Jialal et al (188) reported that NIDDM subjects with age of onset under 35 years showed fasting hyperinsulinism with a delayed and attenuated insulin response to oral glucose when compared with non-diabetic controls. There were no differences in insulin and glucose responses in obese and non-obese diabetic subjects. The mean insulin response between Indian subjects with impaired glucose tolerance and matched controls showed no significant differences, except that the obese IGT subjects had significantly higher insulin levels at 120 min than did the obese controls. (189) In healthy matched male and female Indian volunteers, after 100g of oral glucose, there was no difference in the mean insulin response except that the females had a significantly greater early phase insulin release (190).

The role of insulin resistance and hyperinsulinaemia associated with raised plasma insulin levels in the aetiology of hypertension (191), cardiovascular disease, and NIDDM (192) in migrant Indians has been studied recently by McKeigue et al in London (193). They found that diabetes, fasting and 2 h immunoreactive insulin (IRI), plasma triglyceride concentrations, and systolic and diastolic blood pressures were higher in migrant Asians than in Europeans, while HDL cholesterol was lower. Although there were no differences in BMI between the groups, the mean waist-hip-ratio (fat distribution) was much higher in the Asians, and fasting and 2 h IRI correlated strongly with waist-hip ratio in both groups but much more in the Asians.

Although these findings support the hypothesis that NIDDM, dislipidaemia, raised blood pressure, and increased coronary heart disease risk occur in the setting of insulin resistance and centripetal fat distribution (Syndrome X), some caution is required in interpretation.

An excess of insulin like molecules (ILM), notably proinsulin and 32,33-split proinsulin in the circulation, contributes to insulin immunoreactivity as measured by conventional assays. Thus "hyperinsulinaemia" may not reflect an increase in true insulin. It has also been suggested that these other products may have specific atherogenic effects (194) and contribute to the increased CHD risk in addition to or instead of hyperinsulinaemia. Jarrett (195) failed to demonstrate an independent relationship between relative hyperinsulinaemia and the enhancement of CHD risk. Further information in Indian subcontinentals is required to assess the importance of these observations and, in particular, their relationship to diabetes and IGT.

## VII CONCLUSIONS

The increase in prevalence of diabetes is more a universal phenomenon of migration and Westernization rather than a manifestation confined to Indians. In Fiji, the increase in prevalence of diabetes was less over the years in migrant Indians than in natives (138). Prevalence increased 3.5 times in migrant Indians (from 5.7% in 1964 to 22.2%in 1980) but 25 times in native Fijians (from 0.6% to 15.4%) in 15 years. More Indians were sedentary than Fijians. Similar observations on physical activity and diabetes were made in migrant Indians in Surinam (South America) when compared with migrants from coastal Africa and Java (196). In Singapore, the increase in prevalence was three times higher in the Chinese and Malays than in Indians (197). These slower trends in increase pose a few questions. Is there a threshold for expression of the disease? Does prevalence fall after maximum exposure?

There remain considerable differences in the way that the new criteria of NDDG and WHO are interpreted, and these make comparison of the results from different studies a difficult task. However, there is no doubt that Asian Indians have a genetic susceptibility to develop NIDDM which appears to become exposed when they migrate and achieve improved socio-economic status.

On the other hand, recent data suggest that diabetes may be as common in Indians in India as elsewhere

when age and anthropometric factors are taken into account. Taking an arbitrary diabetes prevalence of 2%, the number of diabetic subjects in India will be 15 million (198). More population-based studies are required in India, however, using WHO criteria before any definite conclusions can be reached. Furthermore, based on the reports of the prevalence of diabetes from the various corners of India and the subcommunity differences seen in Tanzania, the question of what role does ethnicity play needs to be thoroughly scrutinized. Is the genetic susceptibility in these different ethnic groups different?

Although the inherent susceptibility of some populations is explained by the "thrifty genotype" hypothesis, current evidence from Nauru showing a decrease in the incidence of glucose intolerance indicates a decreasing pool of susceptible individuals. With the decreasing frequency of the appropriate genes over future generations, Asian populations may have already experienced such a selective process as shown by studies in Fiji and Singapore, where there is a lack of proportional increase in the prevalence of diabetes exclusively among migrants. Whether this implies that future generations are well adapted can be answered only with longitudinal cohort studies.

Present evidence suggests that insulin resistance may be a common feature in migrant Indians, leading to the high prevalence of non-insulin dependent diabetes and other metabolic disturbances that are possibly responsible for the high rates of coronary heart disease. Further studies are needed to confirm this hypothesis.

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