Diabetes and related variables among the five main racial groups in South Africa: Comparisons from population studies

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Summary
We have investigated the total prevalence of diabetes and related factors among representative, randomly chosen samples of the five ethnic groups living in Cape Town, and (East) Indians in Durban. Comparisons are hindered by differences in age distribution of the populations, while small, isolated groups were found to be unrepresentative. The variability of a single individual's blood sugar levels led us to require at least three abnormal values on 2 different days for a positive diagnosis. The use of different criteria for the diagnosis of diabetes varying from 'lax' to 'stringent' alters the discovered prevalence in our groups by the factor of approximately 2.

Mean blood glucose levels rose with middle age but never between childhood and early adulthood. Afternoon screening tests appeared valid, despite the agreed diurnal difference in glucose tolerance figures.

Both high screening blood glucose levels and diabetes itself were most common among Indians and coloured people and least among Whites and Bantu, each of the latter having a total diabetes prevalence of approximately 3-5% over age 15. It is noted that the Cape Coloured have more diabetes than any of the constituent races from which they originated.

The reasons for such racial differences are unclear—obesity cannot be the explanation here, since, to take one example, the fattest group of all, the Bantu women, have the lowest prevalence of diabetes. We found mild diabetes not uncommon among young people under 20 in the Indian, Malay and coloured population but none among White or Bantu. There was little difference between the sexes, and if anything the poorer people had more diabetes than the better-off.

My colleagues and I have investigated the five major racial groups in the Cape Town area of South Africa in an attempt to determine the prevalence of diabetes and glycosuria and to correlate blood sugar levels with certain variables such as age, sex, weight, race, religion, diet, income, time of day and fecundity (Jackson, Marine & Vinik, 1968; Jackson et al., 1968; Goldberg et al., 1969; Jackson et al., 1969; Marine et al., 1968; Jackson et al., 1970a; Michael et al., 1971). In addition we also studied an 'urban village' near Durban comprising South African Indians (Goldberg et al., 1968; Goldberg, 1968).

This paper attempts to present the most outstanding features arising from these population studies, especially in relation to comparisons between the different racial groups.

Subjects studied (Total numbers selected, with percentage actually screened, in parentheses)

South African Indians (Cape Town 1520, 75%; Durban 2427, 90%)

Very briefly, these comprise:
(1) Tamil-speaking Hindu who were brought over mainly from the Madras area to work on the sugar plantations in Natal around 1860.
(2) Moslems from around Bombay and
(3) Gujarati-speaking Hindu; both these groups paid their own passage.

The Durban (Natal) Indians are overwhelmingly Hindu, mostly arising from the indentured labourers, whereas the Cape Indians are at least 65% Moslem.

Cape Malay (1254, 88%)
These people came originally from the Malay-Indonesia area, are all Muslims and have largely maintained their individual identity.

Bantu (African) (1029, 86%)
The Bantu differ considerably from West African negroes, belong to the southern Nguni group and migrated from central Africa some 300 years ago, reaching Cape Town only in recent years.

Cape Coloured (1534, 63%)
This community is a mixed group owing its origin to four basic elements; aboriginal Hottentot, slaves from West Africa and the Dutch East Indies, early
white settlers and a small contribution from Bushmen and more recently the Bantu. Over the past 100 years this community has consolidated its identity and has intermarried but little with other ethnic groups. Despite the low 'recovery rate' in this group we have reasons for believing that the screened population was reasonably represented.

Whites (1650, 72%)
The white people are primarily English or Afrikaans-speaking, descended from early British or Dutch settlers, or later immigrants from Europe.

In each survey area were chosen as far as possible representative of the total population of the relevant ethnic group, and randomly selected entire households over the age of 10 were interviewed and invited to attend for screening.

Methods
These have been described in detail elsewhere (Marine et al., 1969; Jackson et al., 1970a). Briefly, screening was performed at a set time (2 hr when practicable) after 50 g of oral glucose by urine testing and capillary blood sampling. For reasons of convenience, screening in most of the studies was done in the late afternoon or evening, at least 4 hr after the preceding meal. All subjects whose screening blood sugar values exceeded an agreed level (159 mg/100 ml at 1 hr, 119 mg/100 ml at 2 hr) or who showed glycosuria were invited to undergo full oral glucose tolerance tests (GTT), together with matched negative-screening controls. The final diagnosis of newly discovered diabetes was made if two or more GTT values were abnormal (venous plasma, Auto-Analyzer, Hoffman method*), i.e.

- Fasting level > 120 mg/100 ml
- Maximum level > 185 mg/100 ml
- 2-hr level > 140 mg/100 ml

In all groups approximately 90% of positive screeners attended for GTT—among those who did not attend 'presumed diabetes' was diagnosed when the screen 1 hr blood sugar exceeded 250 mg (at 1 hr) or 200 mg (at 2 hr), together with glycosuria.

The term 'lag (storage) curve' is applied to a GTT in which a peak value over 185 mg/100 ml occurred at 30 min or earlier, with other values normal. 'Borderline' is applied to a GTT with one of these three values abnormal (excluding lag curve). Glycosuria is considered 'renal' if no blood glucose value was over 159 mg/100 ml.

The presence of already 'known diabetes' was checked from records or by blood sugar estimations.

Weight standards were obtained from Documenta Geigy tables, 'obesity' being defined as greater than 15% above standard weight.

Results
Age distribution (Table 1 shows the example of the Cape Coloured racial group compared to Whites)
(1) There were minor differences between the age distribution of each of the various surveyed communities and the corresponding general population of each race. Age correction for this made some but little difference (e.g. total diabetes prevalence for Whites could be age-corrected from 3.2 to 3.6%).
(2) There were major differences between non-white and white races in that a much greater proportion of the population were young in the former and old in the latter. Age correction of non-white groups to match white distribution always led to a considerable increase in prevalence rate (8.7-10.7% in the coloured example). Figures mentioned below, however, are not age-corrected unless so stated.

Table 1. Age distribution of screened sample compared with whole coloured and white populations of South Africa over 10 years of age (1960 census), expressed as percentage of total

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Coloured survey population</th>
<th>% of whole country</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>10-14</td>
<td>213</td>
<td>22.0</td>
</tr>
<tr>
<td>15-19</td>
<td>179</td>
<td>18.5</td>
</tr>
<tr>
<td>20-24</td>
<td>119</td>
<td>12.3</td>
</tr>
<tr>
<td>25-29</td>
<td>48</td>
<td>5.0</td>
</tr>
<tr>
<td>30-34</td>
<td>31</td>
<td>3.2</td>
</tr>
<tr>
<td>35-39</td>
<td>55</td>
<td>5.7</td>
</tr>
<tr>
<td>40-44</td>
<td>58</td>
<td>6.0</td>
</tr>
<tr>
<td>45-49</td>
<td>70</td>
<td>7.2</td>
</tr>
<tr>
<td>50-54</td>
<td>59</td>
<td>6.1</td>
</tr>
<tr>
<td>55-59</td>
<td>60</td>
<td>6.2</td>
</tr>
<tr>
<td>60-64</td>
<td>36</td>
<td>3.7</td>
</tr>
<tr>
<td>65-69</td>
<td>27</td>
<td>2.8</td>
</tr>
<tr>
<td>Over 70</td>
<td>13</td>
<td>1.3</td>
</tr>
<tr>
<td>Total</td>
<td>968</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Screen blood sugar (Tables 2 and 3)
In both sexes in all races the mean blood sugar levels rose with age, though in white people the rise was seen only over the age of 60. In no race was there a significant difference between the first two age groups—i.e. no rise in blood sugar from childhood to young adulthood. There was no significant difference between the sexes in any group.

The white and African people clearly had the lowest mean blood sugar levels of all groups, but these two cannot be directly compared with each other because of differences in timing of the samples. The coloured community showed the highest mean values of all races, especially if due allowance is made for their estimations being performed by Auto-
**Diabetes in different races**

TABLE 2. Mean 1-hr post-glucose blood sugar screening values (mg/100 ml) with standard errors

<table>
<thead>
<tr>
<th>Age group</th>
<th>Indian</th>
<th>Male</th>
<th>Female</th>
<th>African</th>
<th>Male</th>
<th>Female</th>
<th>Malay</th>
<th>Male</th>
<th>Female</th>
<th>Coloured*</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-14</td>
<td>120(2-3)</td>
<td>120(2-3)</td>
<td>105(1-5)</td>
<td>107(1-5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15-34</td>
<td>122(2-9)</td>
<td>120(2-0)</td>
<td>113(2-5)</td>
<td>106(3-2)</td>
<td>124(1-3)</td>
<td>132(0-6)</td>
<td>127(2-9)</td>
<td>128(2-8)</td>
<td>141(3-6)</td>
<td>144(3-3)</td>
<td>148(6-3)</td>
<td>145(4-7)</td>
</tr>
<tr>
<td>35-54</td>
<td>132(5-6)</td>
<td>138(4-3)</td>
<td>127(2-9)</td>
<td>128(2-8)</td>
<td>141(3-6)</td>
<td>144(3-3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55+</td>
<td>163(7-9)</td>
<td>144(16-6)</td>
<td>128(4-7)</td>
<td>130(7-4)</td>
<td>156(5-6)</td>
<td>174(8-7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* AutoAnalyzer Hoffman method, others Hagedorn-Jensen.

Figures in brackets are standard errors.

Analyzer as against a Hagedorn-Jensen method for the others (a difference of ±10 mg/100 ml).

Frequency distribution curves of blood sugar levels were unimodal in all cases, but showed varying differences with age. In the white community all curves were identical in shape and position except for an increased tailing to the right with age; in all others there was some degree of shift to the right of the whole curve with age (Jackson et al., 1969; Marine et al., 1969; Michael et al., 1971).

No significant correlation between blood sugar levels and body weight could be found in any racial group or sex, nor between blood levels and parity in women over 40 years of age.

**Glycosuria (Table 4)**

The frequency of glycosuria, both diabetic and non-diabetic, rose with age in all races and was in general more common among males. It was commonest among the coloured people. Glycosuria of all types was rare in children—even renal glycosuria we found only once under the age of 15 out of a total of seventy-three diagnosed cases in the Cape surveys. Renal glycosuria occurred equally in both sexes.

TABLE 4. Glycosuria and diabetes over age 15 in different races in Cape Town

<table>
<thead>
<tr>
<th>Glycosuria</th>
<th>Known diabetes</th>
<th>Total diabetes*</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>7-9</td>
<td>0-9</td>
</tr>
<tr>
<td>Bantu</td>
<td>6-9</td>
<td>0-9</td>
</tr>
<tr>
<td>Indian</td>
<td>7-9</td>
<td>4-3</td>
</tr>
<tr>
<td>Malay</td>
<td>9-5</td>
<td>1-4</td>
</tr>
<tr>
<td>Coloured</td>
<td>13-5</td>
<td>1-3</td>
</tr>
</tbody>
</table>

* Not corrected for age-distribution.

Glycosuria was more frequently non-diabetic than diabetic; 155 diabetic out of a total of 400 glycosuric (39%). This proportion varied enormously with age and sex; 4% of young glycosuric men being diabetic as against 77% of elderly women.

Glycosuria after a glucose load was inconstant even under reasonably standardized conditions in approximately one-third of some 350 patients who received two loads and showed glycosuria at least once.

Out of 158 newly discovered diabetics, sixty-six had no glycosuria at post-glucose screening (42%), and twenty-one out of forty-one (50%) had no glycosuria post-prandially.

**Diabetes etc.**

Basic results of the Cape studies are shown in Table 4 and Figs. 1 and 2.
Age correction to compare with the white group raises the coloured over-15 prevalence from 8.7% to 10-7%, the Cape Indian prevalence from 10.2% to 10.1%, and the African from 3.6% to 4.2%. Prevalence of known plus discovered diabetes in Natal Indians was 7.9% (uncorrected).

Young diabetics, under the age of 20, were found among coloured, Indian and Malay people but not among white or African. Diabetes, both known and newly discovered, occurred equally in males and females, except that it appeared to be more common among Malay women (fourtyn-four to nineteen) and elderly Indian women than in their menfolk.

Symptoms of diabetic type were present in only approximately 5% of newly diagnosed diabetics, and absent in some subjects whose post-glucose blood sugar levels exceeded 400 mg/100 ml. 'Lag storage' glucose tolerance curves (strictly defined) were encountered in seventeen subjects—sixteen of them male.

A small community of 365 highly inbred Tamil Indians in Cape Town were also examined, and 37% over the age of 25 were found to be diabetic (Marine & Jackson, 1966). Detailed studies of this community are being reported separately; none of their results are included in the Cape Indian figures given here.

Body weight (Tables 5, 6, 7)

Among the surveyed populations the relative percentages of each sex classified as obese are shown in Table 5. The influence of obesity on diabetes diagnosis is shown in Table 6.

Table 7 compares the frequency of obesity among 'old' and 'young' female diabetics discovered at survey with that among non-diabetics. (Only Indian and coloured figures are presented because of insufficient 'young' discovered diabetics in other races—non-diabetics in all races showed a similar pattern, i.e. more obese over 40 than under 40.)

Reproducibility of blood glucose levels and time of day

The blood glucose levels attained in positive screenees and controls at screening could be compared with the 1 hr values during full GTT, performed later. In all surveys, there was considerable scatter from the regression line, with a positive correlation of around 0.7.

Intra-individual variations were frequently encountered—positive screenees with very high blood levels having normal GTTs and vice versa. It may be noted that 'discovered diabetes' was diagnosed only when the blood glucose levels were abnormally high.
The relative frequency of diabetes found in the different races was well mirrored in the corresponding mean blood glucose levels obtained at screening. It has recently been established that blood glucose levels on glucose tolerance testing are usually higher in the afternoon than first thing in the morning, though the difference is less in hyperglycaemics (Jarrett & Keen, 1970). We have also observed this when testing individuals, yet on mass screening we found no real difference in mean values between morning and afternoon as shown in Table 8. This phenomenon has also been found and commented upon by Jarrett & Keen (1970), while Hayner and co-workers (1965) found no difference between morning and afternoon screening levels, provided no food had been taken for at least 4 hr. It does at least suggest that afternoon or evening screening of populations is a valid procedure.

Glycosuria. Glycosuria is clearly inefficient, inconsistent and insensitive as a sole screening test, even after a glucose load (Jackson et al., 1968a). Nevertheless, the great majority (approximately 90%) of all newly discovered diabetics showed glycosuria at some time during their testing—we had initially expected to find far more non-glycosuric hyperglycaemia. The marked increase in renal glycosuria with age suggests that in some people the renal threshold for glucose falls as they get older, though it is common knowledge that it rises in others.

Diabetes

Comparisons. The total prevalence of diabetes between a rapidly breeding (young) and slowly breeding (old) population cannot be validly be directly compared, and either age correction should be applied or individual age-groups be considered. Thus, the already high prevalence rate of the Cape Indians is almost doubled by age correction to compare with the white population. Where we have not age-corrected we have therefore tended to underestimate the diabetes prevalence among the non-white group as compared with the white.

Another hazard of direct comparison lies in the use of small isolated groups. Thus, our inbred Tamil Indians in Cape Town had the remarkable diabetes frequency of 37% over age 25—nearly three times as high as the other groups of Cape Indians who were tested. This sort of prevalence is very similar to that

Discussion

Blood glucose levels

The mean blood sugar levels rose at middle age. We had hoped that the blood glucose distribution at different ages might give a clue concerning the normal range in older people. Thus, if the whole distribution curve shifted to the right with age it might be argued that relative hyperglycaemia was a natural effect of ageing, whereas if the main curve stayed the same with only an increased tail to the right, then it might be argued that hyperglycaemia was equally abnormal at any age. Unfortunately the curves obtained from different surveys varied. In one respect we found general agreement—that the blood glucose levels did not rise between childhood and young adulthood.

Table 5. Obesity prevalence in screened populations (\%)

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>16</td>
<td>23</td>
</tr>
<tr>
<td>African</td>
<td>7</td>
<td>52</td>
</tr>
<tr>
<td>Indian</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>Coloured</td>
<td>7</td>
<td>28</td>
</tr>
</tbody>
</table>

Table 6. Diabetes prevalence in obese and non-obese subjects

<table>
<thead>
<tr>
<th></th>
<th>Obese</th>
<th>Non-obese</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% in population</td>
<td>% diabetic</td>
</tr>
<tr>
<td>Africans</td>
<td>37</td>
<td>2·3</td>
</tr>
<tr>
<td>Indians</td>
<td>11</td>
<td>14·5</td>
</tr>
</tbody>
</table>

Table 7. Obesity in 'young' and 'old' discovered diabetic women

<table>
<thead>
<tr>
<th>Age group</th>
<th>Discovered diabetics</th>
<th>Non-diabetics</th>
<th>(negative screeners)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. % obese</td>
<td>No. % obese</td>
<td></td>
</tr>
<tr>
<td>Under 40</td>
<td>50</td>
<td>46</td>
<td>720</td>
</tr>
<tr>
<td>Over 40</td>
<td>110</td>
<td>23</td>
<td>130</td>
</tr>
</tbody>
</table>

on at least three occasions—once at screening and twice during GTT.

No consistent difference was found between mean screening (afternoon) blood levels and GTT (morning) levels (Table 8).

Table 8. Blood sugar a.m. and p.m. correlations: repeated tests on positive screeners and controls 1 hr after glucose (mean values, mg/100 ml)

<table>
<thead>
<tr>
<th></th>
<th>Africans (95)</th>
<th>Cape Indians (79)</th>
<th>Natal Indians (363)</th>
<th>Whites (90)†</th>
<th>Coloured (314)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening (p.m.)</td>
<td>180</td>
<td>193·5</td>
<td>151</td>
<td>100†</td>
<td>181†</td>
</tr>
<tr>
<td>GTT (a.m.)</td>
<td>170</td>
<td>206</td>
<td>166</td>
<td>118†</td>
<td>161†</td>
</tr>
</tbody>
</table>

* Capillary blood after at least 4 hr fast.
† Venous plasma.
‡ 2 hr after glucose.
of the Pima (American) Indians (Bennett, Burch & Miller, 1971). The Pimas are a considerably larger group, but otherwise similar to our Tamil in being highly inbred, very fat and in a low income bracket. The Pima Indians are of mongoloid origin, the Tamil Indians are Caucasoid with Australoid admixture (Coon, 1966). These two ‘Indian’ groups of entirely different ethnic origin have the highest prevalence of diabetes so far described.

Diagnosis. Because of the known variability of blood glucose levels from day-to-day in some individuals (with which our own experience is in full agreement) diabetes was diagnosed only when subjects ‘screened positive’ and had a GTT judged to be diabetic on at least two blood glucose levels. It seems clearly incorrect to diagnose diabetes on a single abnormal GTT in an asymptomatic person. From a statistical rather than individual point of view, however, this may be less important, since those diagnosed falsely positive on single blood glucose levels will probably counterbalance those diagnosed false negative.

Criteria for diagnosis differ with different authorities, but re-analysis of our data according to ‘lax’ criteria and to ‘stringent’ criteria (Marine et al., 1969) has indicated a mean difference in prevalence between these two extremes of only twofold*. Among the Cape Indians the difference was very little, 5:1% by strict criteria and 6:7% on lax criteria; among white people the figures were 2:0% and 4:9% respectively. This is far less than found by O’Sullivan & Williams (1966) in the Sudbury survey where, using similar criteria for comparison, the difference between stringent and lax was 1:2% and 13:4%.

We have argued before whether our hyperglycaemics are really diabetic, and while in many individual cases we cannot be certain, yet in general I believe we can be vindicated. In the few who had clear-cut diabetic symptoms, the two with retinal micro-anurysms (some others had retinopathy that was probably diabetic), and those with all blood sugar levels over 200 mg/100 ml there can be little doubt. Pointers to the validity of the diagnosis were the presence of glycosuria in the great majority and the fasting blood glucose levels being over 120 mg/100 ml in more than 60%. In the Natal Indian and Cape Coloured surveys where the best clinical assessments were made, ischaemic heart disease was considerably more common among the hyperglycaemics than in normoglycaemics (Michael et al., 1971; Jackson et al., 1970b).

Prevalences. The prevalence of already known diabetes among the Cape white people of 0:8% was similar to that found in white communities in several countries including Britain, the U.S.A., Canada, Norway and Sweden (Jackson, 1970) and was the same as found among the Cape Bantu, but considerably less than among Cape Indians.

The prevalence of total diabetes over the age of 15 among the white people was 3-2% (3:7% age corrected); this compared closely with the 3-6% from Cape Bantu. These semi-urbanized Africans thus have as much diabetes as their white neighbours, despite their low income and their recent, incomplete separation from tribal life, in which diabetes is uncommon.

The Indians (and Malays also) had more diabetes than either Whites or Africans and certainly more than their forebears and counterparts in India (and Malaysia) itself. The coloured people are particularly interesting; they had higher blood sugar levels and more diabetes than any of their progenitors, despite the absence of any obvious and particular environmental diabetogenic factor. Evidently hyperglycaemia has evolved from racial mixing.

Closer consideration of the total diabetes prevalence of 3-2% found in the white population indicates that this is low compared with other studies of white populations that have been based on post-glucose blood sugar estimations, from which a very approximate figure of 10% might be quoted (Birmingham, Bedford, Berlin, nation-wide U.S.A., Tecumseh, Pennsylvania) (Jackson, 1970). Other reports of expatriate white populations have also indicated similar comparatively low diabetes rates (Australia, Hawaii—Jackson, 1970). There is thus some evidence that certain comparatively recently established white populations have less hyperglycaemia than their home-staying cousins.

Racial differences. It is difficult to explain these racial differences on an environmental basis. White South Africans have an incidence of myocardial infarction amongst the highest in the world, a very large mean per capita consumption of sugar (110 lb/head/year according to the International Sugar Council 1968) and are considerably obese (Table 9). Their intake of total and saturated fat is higher than that of the Cape Coloured and Bantu. Yet comparatively speaking their diabetes prevalence is not great. In their rural state the Bantu diet is very largely carbohydrate (maize is their staple foodstuff); the town dwellers in general eat more animal fat (still little compared with the white population), more protein, more calories and considerably more sugar. The Indians were not particularly fat—considerably less obese than the Bantu and although they may eat more calories and certainly more sugar than Indians in India itself, yet their consumption is lower than among white people. As with diabetes, so with ischaemic heart disease, we found Indians to suffer

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*‘Lax’ criteria diagnose diabetes at GTT on single value > 130 mg/100 ml at 2 hr. ‘Stringent’ criteria diagnose diabetes at GTT on two values; > 130 mg fasting; 200 mg (peak); 150 mg/100 ml (2 hr).
even more than white people (Jackson et al., 1970b), but Bantu, even when diabetic, were completely free of the clinical effects of coronary disease.

West & Kalbfleisch (1971) have provided evidence that overweight may be the most important single environmental factor in the emergence of diabetes in different races. Our South African populations do not support this thesis. Thus, the two ‘most obese’ races (White and Bantu) had the lowest frequency of diabetes. Bantu women are fat by tradition and by their husbands’ preference yet we found marginally more diabetes among the men. The importance of overweight was certainly shown within the Indian population, however, in that diabetes was over three times more common in obese than in the non-obese. On the other hand, the frequency of diabetes showed little relationship to weight among the Bantu (Table 6).

To our surprise, we found obesity more common in diabetic women under the age of 40 than above this age (Table 7)—in the general population in all races obesity was more common in the over 40s. We therefore wonder whether obesity may be a more important diabetogenic factor in young than in older women (discussed in more detail elsewhere—Jackson et al., 1972).

True juvenile, ketosis-prone diabetes is comparatively rare among our non-white racial groups (almost non-existent among Indians), but mild, ‘maturity-onset’ type diabetes was discovered in several young people under 20 among Indian, Malay and coloured people but was not seen among Bantu or White.

For a long time diabetes has been said to be much more common among white women and Indian men than among white men and Indian women. This belief has probably been erroneous, and properly conducted population studies have not supported it. In Cape Town, where we also have a large preponderance of white women attending our diabetes clinic, we actually found rather more white male than female diabetics, and virtually no sex difference among the Indians.

Diabetes is usually considered to be more common among affluent societies, but among white people we found no relation between income group and hyperglycaemia (Jackson et al., 1969), while to our surprise diabetes appeared more common in the lower income group Indians, both in Cape Town (Marine et al., 1969) and Durban (Goldberg, 1968). In Durban an assessment was made which placed the lower income group Indians below the poverty datum line.

Acknowledgments

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References


