The Epidemiology of Indian people with diabetes in the UK

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There has been no study of the prevalence of childhood diabetes in Indians (defined as people coming from, or whose forefathers came from, the Indian subcontinent) since the Leicester City study (1). This demonstrated a prevalence of 0.54 cases per 1000 in Indian children compared with one of 0.99 for whites. Since then there has been only studies on incidence. An initial report (2) suggested that there was a simple linear increase in incidence-starting from very low rate. By the 1980s the rate did not alter, producing a plateau at an incidence similar to that of the whites. A subsequent report (3) has emphasized the sporadic, or epidemic nature of he incidence; shown in the table 1. It is concluded that the incidence of IDDM can be as common as in white children.

GESTATIONAL DIABETES (GDM)

The definition of this which was used was diabetes or impaired glucose tolerance (WHO criteria) (4) at 28-32 weeks, which resolves in the puerperium.

Using this criterion we demonstrated an excess incidence in Indian women (5)-Table II(a). All women considered at risk of gestational diabetes for reasons such as family history to previous abnormal pregnancy had a Glucose Tolerance Test (GTT) performed. Those with normal glucose tolerance were followed up for a control group. With standard management of their diabetes (6) the white women with GDM achieved perinatal mortality and morbidity which was no different from those without glucose impairment. The Indian GDM women had results which were not so satisfactory, Table II (b). it is concluded that GDM is more common in the Indian, and it might be more significant; standard management may require modification to improve treatment results.

Table 1
Insulin dependent diabetes: cases and incidence in Indian and White Children (less than 15 years old – incidence expressed as cases/100,000/year

Year	Indian Cases	Incidence	White cases	Incidence
1974	1	7.7	32	18.8
1975	0	0	27	15.0
1976	2	13.3	32	18.5
1977	0	0	28	16.1
1978	2	13.0	27	15.2
1979	4	25.0	28	16.4
1980	2	12.5	24	14.5
1981	5	30.0	19	11.5
1982	1	5.5	26	16.25
1983	2	10.5	31	19.7
1984	0	0	27	17.2
1985	0	0	22	14.2
1986	5	24.5	26	16.6
1987	2	9.5	11	7.2
1988	2	9.2	17	11.0
1989	2	9.3	16	10.0

From: Diabetes Research Department, Leicester General Hospital, UK.

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Table II (a)
Gestational Diabetes

	Indian	White
Normal	166 (84)%	324 (91)%
Impaired tolerance*	22 (11)	27 (8.3)
Diabetes**	10 (5)	5 (0.7)

* Blood glucose > 7.7 mmol/L 2 hours after 75G glucose load
** Blood glucose > 11 mmol/L 2 hours after 75G glucose load but normal postpartum.

Table II (b)
Foetal Complications in GDM
(microsomia or macrosomia, premature delivery, foetal distress, symptomatic hypoglycaemia

	Indian	White
Normal tolerance	13%	22.5%
Impaired glucose tolerance and diabetes	38%	25%

NON-INSULIN DEPENDENT DIABETES (NIDDM): PREVALENCE

There has not been a study employing all the World Health Organisation (WHO) criteria investing the prevalence of NIDDM in Indian people in the United Kingdom. Two studies have examined the prevalence in the Midlands. In a poor part of coventry (7) one group used random glucose testing to screen the population, followed by glucose tolerance tests if the random blood glucose was elevated above > 6.0 mmol/L within 2 hours of food, N > 4.4 mmol/L 2 hour's or more after eating. No second glucose tolerance test was employed.

A further pilot study was performed in Leicester (8). It used standard epidemiological techniques to reduce the number screened, by producing a randomized list of names in two of each age and sex strate: 45-49 and 50-54, male and female: 120 in each group. All the people in the United Kingdom have to register with a General Practitioner in order to receive National Health Service. If people move they have to re-register, and the central committee, then called the Family Practitioner Committee, would de-register their name's from their original General Practitioner. This should serve as an accurate source for data, but unfortunately about a third of all people to be studied had moved. Those in the study who did not have diabetes were subjected to glucose tolerance testing but only once. The results of both these studies are shown in Table III.

Despite the difference in methodology of screening and testing there is no doubt that NIDDM is particularly common in the United Kingdom Indian, as has been found for other migrant Indians. The female is at a lower risk.

Table III
Prevalence of NIDDM in Indian people in the UK percentage (95% CI)

Age	Coventry	Age	Leicester
20-39	2.6 (1.9-4.0)		
40-59	12.6 (10.3-15.7)		
		45-49	23% (9-44%)
		50-54	31% (17-49%)
60-79	25.4 (20.1-31.8)		
20-39	1.4 (0.6-2.6)		
40-59	9.3 (7.2-12.3)		
		45-49	2.9% (06-15%)
		50-54	14.3% (3-36%)
60-79	19.9 (12.5-27.4)		

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Brief studies have been reported on differences between Indian people of different religions, but all have been of relatively small size and consequently the opportunity for error has been great. There is no data to suggest a major difference, between, for example, the mainly Gujrati folk of Leicester and the Punjabi Sikh of Coventry. There has been no study of incidence of NIDDM.

PREVALENCE OF COMPLICATIONS

a) Macrovascular

The Indian person is particularly prone to myocardial infarction, this being the commonest cause for hospital admission. The obvious risk factor, lipid abnormalities, does not explain this increased risk (9). If diabetes or glucose impairment was the cause of the increased risk of myocardial infarction in the Indian general population, then by comparing the rates of ischaemic heart disease in the two diabetic populations-white & Indian-should produce similar prevalence rates of ischaemic heart disease. For this reason a study was performed comparing 456 Indian and 451 White diabetics (10), Table V. Ischaemic heart disease was present in 24% of Indian and white males, and 25% of Indian women, compared with 20% of white. There was no significant difference. Nor was there any significant difference in the components of ischaemic heart disease, angina (pain worsened by exertion) angina with abnormal ECG, or definite myocardial infarction (two of three; classical history, evolving ECG changes with ST elevation of creatinine kinase to least twice the upper limit of the laboratory normal range).

A similar study was also reported from Southall (11), and from the diabetes drafting group comparing data from London and Delhi(12). If the same process produces ischeamic heart disease as well as peripheral vascular disease then this would be expected to have the same prevalence between the races. Oddly enough this was not found, with Asian rates of peripheral vascular disease being 0.4(0.3-0.5) for the three studies (14).

b) Microvascular

Renal disease: we had earlier reported an increased prevalence of proteinuria (13) in the Indian diabetic compared with the White. This was confirmed in the larger study (10), and an increased prevalence of microalbuminuria was also found (15) since proteinuria is a harbinger of renal failure from diabetic nephropathy, we expected that the incidence of End Stage Renal Failure (ESRF) would be more common in Indian diabetic people (14): proteinuria also increases mortality from ischaemic heart disease and this may contribute to the excess mortality in the Indian from coronary artery disease. This difference in proteinuria was not found in the diabetes drafting group study.

Retinopathy: The prevalence of retinopathy was lower in Indians (10% compared with 32% for Whites), with confidence limits of 0.37 (0.2-0.5). The retinopathy was judged by opthalmoscopic inspection through dilated eyes; and was classed into background, exudative, maculopathy and proliferative: all were more common in the White but did not achieve significance.

Cataracts were more common in the Indian: a relative risk of 6 (1.5-28).

It might be thought that there would be difficulty in viewing the retina through an eye with a dense cataract. If we assume that there would be retinopathy present in all eyes with cataracts, the difference in retinopathy would still be significant. This difference was not found in the Southall study.

Neuropathy: This has not been examined in the United Kingdom.

Incidence of complications: Prevalence studies may be subject to bias due to a survivor artefact: for example White diabetic proteinurics might die

Table IV
Incidence of ESRF treatment in people with known diabetes

Years	White		Indian	
	Cases	Population (000)	Cases	Population (000)
1974-1988	14	780	10	70
Incidence General population		1.75 (0.8-2.7)		17 (6-30)
Incidence Diabetic population		53 (28-78)		420 (240-920)

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Table V
Prevalence rates of diabetic complications in Asians and White Caucasians in three major studies

	Diabetes Drafting Group		Southall		Leicester		Cumulative risks for complications Asians: White Caucasians	
	I	W	I	W	I	W	Odds ratio	Confidence limits (95%)
M I	9%	7%	7%	7%	15%	13%	1.1	0.8-1.4
Angina	17%	16%	6%	5%	14%	12%	1.1	0.9-1.3
Retinopathy	20%	47%	16%	17%	12%	32%	0.9	0.7-1.0
Cataracts	-	-	16%	16%	10%	5%	0.9	0.7-1.3
PVD	1%	5%	16%	29%	4%	9%	0.4	0.3-0.5
Hypertension	30%	26%	29%	29%	18.5%	19.5%	0.9	0.7-1.0
Renal Involvement	7%	9%	-	-	20%	13%	1.2	0.9-1.6

at a faster rate than Indian; as a consequence there would be more Indian diabetics with proteinuria. The incidence of complications is therefore of even greater interest. We have examined this for renal disease.

Renal Disease: ESRF treatment-dialysis and transplantation-is a marker of End Stage Renal disease. The availability of this treatment was limited to diabetics in the past, but we have no evidence that there is any racial bias in the availability of treatment. The Leicester nephrology unit has also been liberal in it's use of dialysis compared with other UK units.

The numbers of diabetics: white or Indian; commencing treatment of ESRF will be a measure of the incidence of ESRF. If Glomerular Filtration Rate (GFR) fail at the same rate in both racial groups then the incidence of early renal disease would be closely related to the incidence of ESRF treatment.

It is likely then that Indian diabetics are particularly prone to develop nephropathy. No obvious explanation is available to account for this-except that the Indian is more likely to develop renal failure: diabetes acts as a further risk factor.

Conclusion

Several large questions have been produced by the present studies: is the incidence of IDDM changing; is the high risk of NIDDM due to poor nutrition in early life as has been suggested for Whites; why is there such an enormous difference in atheromatous disease of the hearts and legs; what is the reason for renal disease excess?

Comparative studies with non-migrant populations are obviously called for, particularly since the Delhi WHO report suggests a difference in renal with the Leicester series. Similarly, cohort incident studies should confirm some of the data from the prevalence studies.

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