ETHNIC DIFFERENCES IN CIRRHOSIS OF THE LIVER IN A BRITISH CITY:  
ALCOHOLIC CIRRHOSIS IN SOUTH ASIAN MEN  

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Abstract — Aims and Methods: We studied the ethnic origin of cirrhotic patients retrospectively over the 14-year period 1987–2000 and compared the ethnic make-up of the cirrhotic patients with the ethnic make-up of the local catchment population. Results and Conclusions: Of 381 cirrhotics, 64.1% were white, 29.1% South Asian, 4.7% Afro-Caribbeans and 2.1% other races. These proportions were different from those of the local community in that South Asians were over-represented and Afro-Caribbeans were under-represented. Alcohol was the commonest cause of cirrhosis (60.9%) and South Asian non-Moslem males with alcoholic cirrhosis were over-represented and were younger at diagnosis than white alcoholic cirrhotics.

INTRODUCTION  
Young non-Moslem South Asian males with alcoholic liver cirrhosis are a relatively common clinical problem at our hospital in west Birmingham. However, there have been no studies on ethnic differences amongst cirrhotic patients in Britain. A previous paper from our hospital reported the epidemiology of liver cirrhosis in the area from 1959 to 1976, but the ethnic origin of the patients was not recorded (Saunders et al., 1981). Over the last 30 years, the population of west Birmingham has changed, becoming a mixed ethnic community, and we have therefore had the opportunity to study ethnic influences on cirrhosis and in particular alcoholic cirrhosis. We studied the ethnic origin of patients with liver cirrhosis attending our inner city hospital to determine if the ethnic make-up of the cirrhotic patients differed from the ethnic make-up of our catchment population and to determine if South Asian males are over-represented amongst those with alcoholic cirrhosis.

SUBJECTS AND METHODS  
In-patients and out-patients attending the liver service at the City hospital over a 14-year period, from 1987 to 2000, with cirrhosis were identified from a computerized database. Using the classification of ethnic origin in the 1991 census (Office of Population Censuses and Surveys, Department of Health and Social Security, 1992), the ethnic origin of each patient was recorded as white, South Asian (Indian, Pakistani or Bangladeshi), Afro-Caribbean or ‘others’. Cirrhosis was confirmed by liver histology (73% of cases) or by characteristic clinical features with portal hypertension and compatible biochemistry (low serum albumin and raised serum globulins). The demographic data of the adult population (age >20 years) of west Birmingham was obtained from the 1991 census. Data were analysed by Microsoft Excel version 4.0. Odds ratios (OR) and their 95% confidence intervals (CI) were calculated by standard methods (Morris and Gardner, 1989).

DISCUSSION  
This study indicates that there are ethnic differences in cirrhosis seen in west Birmingham. The commonest cause of

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cirrhosis was alcohol. The proportion of our cirrhotic patients with alcoholic cirrhosis was 60.9%, compared to 52% in the previous study from our hospital (Saunders et al., 1981). Another important difference between the earlier study and the present one is that 12.1% of our patients had post-viral cirrhosis and 14.9% had cryptogenic cirrhosis, compared with 6 and 35%, respectively, in the previous study. These differences can be explained by the advent of serological tests for hepatitis B and C. It is likely that some cases of cryptogenic cirrhosis could now be reclassified as cirrhosis caused by non-alcoholic fatty liver disease.

The most important finding in our study concerns alcoholic cirrhosis; the proportion of South Asian males was greater, and of Afro-Caribbean males fewer, than would be expected if the ethnic composition of the alcoholic cirrhotics reflected the local community. Virtually all the South Asian males with alcoholic cirrhosis were non-Moslems and were younger at diagnosis than their white counterparts. This latter observation could be explained by the higher proportion of younger males in the South Asian population. However, as the peak age of presentation of alcoholic cirrhosis is in the fifth decade, we believe it is more likely that the younger age at diagnosis of South Asian males is a significant finding. Our study has under-estimated the excess of alcoholic cirrhosis in non-Moslem South Asians, because the census data did not allow separation of Moslem from non-Moslem adult South Asians in the catchment population.

The explanation for the ethnic differences in alcoholic cirrhosis are not known. Possible explanations include social and cultural differences in attitudes to alcohol, the quantities of alcohol consumed by different ethnic groups and genetic differences in the metabolism and effects of alcohol.

Some studies have shown that similar or lower amounts of alcohol are consumed by Indians (South Asians) resident in Britain, compared with whites (Cochrane and Bal, 1990; McKeigue and Karmi, 1993). Although consuming no more alcohol than whites, Sikhs are more likely to consume spirits (McKeigue and Karmi, 1993) and to drink daily (Cochrane and Bal, 1991). Despite these data, the overall incidence of alcohol-related diagnoses was higher in Indian males than white males presenting to a London psychiatric hospital (Mather and Marjot, 1989) and alcoholic liver disease is more common in Indians than the general British population (McKeigue and Karmi, 1993). There is evidence that Afro-Caribbean males and females in Britain drink less than their white compatriots (McKeigue and Karmi, 1993), which is consistent with our observation that alcoholic cirrhosis was less common in Afro-Caribbeans.

As patients with severe alcoholic liver disease end up in hospital sooner or later, it seems unlikely that differences in use of primary health care by different ethnic groups or in referral patterns of different ethnic groups to hospital from primary care explain the observed differences.

Genetic factors may explain the ethnic differences in the frequency of alcoholic cirrhosis. The observation that only 15% of alcoholics develop liver cirrhosis has led to the suggestion that genetic factors may be responsible for the individual susceptibility to develop cirrhosis (Sorensen et al., 1984). This is supported by data from twin studies, which have conclusively shown that there is increased concordance of alcoholic cirrhosis in monozygotic twins, compared with dizygotes (Hrubec and Omenn, 1981). Acetaldehyde, a metabolite of alcohol, is thought to be important in the development of alcoholic cirrhosis and its formation and degradation are dependent on the activity of a number of alcohol-metabolizing liver enzymes (Leiber, 1995). Genetic polymorphisms of these enzymes in different ethnic groups may explain variability in susceptibility to the development of alcoholic cirrhosis. Polymorphisms of alcohol/alddehyde dehydrogenase and cytochrome P-450 have been studied in British whites (Day et al., 1991; Ball et al., 1995), but not in other ethnic groups. There is evidence that liver enzyme activities are higher (Clarke et al., 1990; Wickramasinghe et al., 1995) and acetaldehyde-mediated haemoglobin modification is more marked (Wickramasinghe et al., 1995) in Indian alcohol misusers, compared with comparable white alcohol misusers, although the whites had misused alcohol for longer periods. These data suggest increased susceptibility of the Indians to the hepatic effects of alcohol. The younger age at diagnosis of our Indian males with alcoholic cirrhosis also points to the possibility of genetic influences, particularly as there is evidence that young Indian males in Britain drink less than older individuals from their community (Cochrane and Bal, 1990).

More recently, polymorphisms of genes encoding for components of the immune response, e.g. TNFα (Grove et al.,

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Whites</th>
<th>South Asians</th>
<th>Afro-Caribbeans</th>
<th>Others</th>
</tr>
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<tr>
<td></td>
<td>Male</td>
<td>Female</td>
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<td>Female</td>
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<td>Alcohol</td>
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<td>52</td>
<td>58</td>
<td>1</td>
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<td>Cryptogenic</td>
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<tr>
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<tr>
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<td>67</td>
<td>18.5</td>
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Table 1. Aetiology of cirrhosis at City Hospital, Birmingham, 1987–2000

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1997) and interleukin-10 (Grove et al., 2000), have been associated with the development of advanced alcoholic liver disease, but nothing is known about ethnic differences of these genetic polymorphisms.

There is some evidence to suggest that there are ethnic differences in the pattern of organ damage seen in alcoholics. African-Americans are much less likely than whites to be hospitalized with cirrhosis, but are more likely to be admitted with chronic pancreatitis (Lowenfels et al., 1999). These differences could be genetically or environmentally determined.

Our study has an important public health message about the dangers of alcohol for our inner city population. The risks of alcohol to young non-Moslem South Asian males needs vigorous emphasis. Resources should be spent on a health campaign targeting at-risk groups in the community. Individuals with suspected alcoholic liver disease need early referral for assessment and counselling. We can only make these recommendations on the basis of the findings in our own community, but they are likely to be relevant to other areas with a similar ethnic population mix.

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REFERENCES


