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Prevalence of chronic viral hepatitis in people of south Asian ethnicity living in England: the prevalence cannot necessarily be predicted from the prevalence in the country of origin

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SUMMARY. The prevalence of hepatitis B and hepatitis C in immigrant communities is unknown. Immigrants from South Asia (SA) are common in England and elsewhere, and the burden of viral hepatitis in these communities is unknown. We aimed to determine the prevalence of viral hepatitis in immigrants from SA living in England, and we therefore undertook a community-based testing project in such people at five sites in England. A total of 4998 people attending community centres were screened for viral hepatitis using oral fluid testing. The overall prevalence of anti-hepatitis C virus (HCV) in people of south Asian origin was 1.6% but varied by country of birth being 0.4%, 0.2%, 0.6% and 2.7% in people of this ethnic group born in the UK, India, Bangladesh and Pakistan, respectively. The prevalence of hepatitis B surface antigen was 1.2%–0.2%, 0.1%, 1.5% and 1.8% in people of this ethnic group born in the UK, India, Bangladesh and Pakistan, respectively. Analysis of risk factors for HCV infection shows that people from the Pakistani Punjab and those who have immigrated recently are at increased risk of infection. Our study suggests that migrants from Pakistan are at highest risk of viral hepatitis, with those from India at low risk. As prevalence varies both by country and region of origin and over time, the prevalence in migrant communities living in western countries cannot be easily predicted from studies in the country of origin.

Keywords: hepatitis B, hepatitis C, immigrants, prevalence.

INTRODUCTION

Chronic hepatitis because of either the hepatitis C virus (HCV) or the hepatitis B virus (HBV) is common with an estimated 500 million people infected worldwide [1]. Infection with either virus causes slowly progressive liver damage that, without therapy, may lead to cirrhosis and/or liver cancer after many decades. Effective therapy, either with oral antiviral agents (HBV infection) or with pegylated interferon and ribavirin (HCV infection), is available and can reduce mortality [2,3]. In the developed world, acquisition of infection is associated with particular 'high risk' behaviours including injecting drug use (HCV and HBV) and multiple sexual partners (HBV), although other routes of infection (for example from contaminated blood or blood products) were more important in the past. In the developing world, chronic HBV is most often transmitted from infected mothers to their offspring or by early childhood exposure to infected children [4]. The main routes of transmission for HCV in the developing world are not clear but probably include iatrogenic infection (via contaminated medical equipment) and use of other contaminated items, such as razors [5]. In the Indian sub-continent, the prevalence of viral hepatitis has been assessed by a number of studies, and for chronic HBV infection, the WHO describes the Indian sub-continent as 'intermediate endemicity', with an estimated prevalence between 2% and 8% [4]. In 1999, the WHO estimated that in India, Bangladesh and Pakistan, antibody to hepatitis C was found in 1.8%, 2.4% and 2.4% of the population, respectively [6].

Abbreviations: HBc, hepatitis B core; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; HCV, hepatitis C virus; SA, South Asia; HBeAg, hepatitis B e antigen; GP, general practitioner.

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An outstanding question relates to the prevalence of viral hepatitis in communities living in industrialized countries but with origins in other parts of the world. In many countries, including the UK, immigrants are not screened at entry for viral hepatitis and therefore the prevalence in immigrant communities is unknown. Studies on unselected populations in Europe (such as pregnant women) show that the prevalence of HBV is higher in those born abroad, approaching the prevalence in their country of origin [7,8]. However, it is also possible that the prevalence in migrants is lower than those who remain in the country of origin, as those who chose to migrate often tend to be healthier than those who remain [9]. It is also unknown whether second generation immigrants born in low prevalence countries, such as the UK, remain at higher risk of infection – for example through regular travel to their 'home' country or close contact with other chronically infected individuals. To date, no studies have addressed the issue of chronic viral hepatitis in immigrants from the Indian sub-continent, and here we report the results from a large-scale, multisite study of the prevalence of viral hepatitis in individuals of south Asian ethnicity living in England.

MATERIALS AND METHODS

Study sites and testing technique

The study was conducted in five regions – East London, West London, Walsall, Sandwell and Bradford. They were selected because of their large south Asian communities who have migrated from India, Pakistan and Bangladesh coupled with local public health physicians and gastroenterologists/hepatologists who were willing to participate. At each site, local religious leaders and community representatives were contacted, and the nature and purpose of the study outlined. Based upon this dialogue, potential testing sites were identified, and the site management committee asked to consider participation, with all agreeing to do so. Public meetings were held at the sites, and attendees were provided with details of the study and invited to participate. Testing sessions were arranged at convenient times advertized internally. The number of testing sessions at each site varied according to local interest and requests – typically three to six sessions.

At each session, a team of volunteers, trained in obtaining oral mucosal fluid samples, were used to recruit and consent study participants. Volunteers included public health physicians and nurses, the study team and medical students in addition to well-informed members of the local community who, usually, had previous medical or nursing training. Adults (older than 16 years) attending the sessions were provided with written information (in appropriate languages) about chronic viral hepatitis and the nature and purpose of the study. They were invited to provide an oral fluid sample for testing and to complete a simple survey form with information on age, sex, place of birth, date of moving to the UK, other significant medical conditions and contact details. Following a review of the first 500 patients tested in East London, we added an additional question relating to city of birth, in addition to country of origin. When the results of the oral fluid test were available (usually within 1 week), those who had tested positive were asked to attend the local hospital for a confirmatory blood test. Those confirmed as hepatitis B surface antigen (HBsAg) or anti-HCV-positive on serum were offered an appointment with the local treating physician and managed according to local guidelines. For patients who were unwilling to re-attend for confirmatory blood tests, their general practitioner (GP) was informed of the result. We also tested a small cohort of patients attending a general practice in East London. Patients from appropriate ethnic groups who were waiting to see their GP for an unrelated condition were approached and asked to participate. Those who agreed were screened for viral hepatitis with the oral mucosal fluid sample and managed as above.

Virological assays

Oral fluids were assayed for anti-HCV and for HBsAg, as previously described [10,11]. After the first 1000 subjects, it became apparent that a number of HBsAg-'positive' tests were not confirmed on blood testing. To minimize this, the oral fluid was tested for antibodies against hepatitis B core (HBc), and only samples that were HBsAg-positive and anti-HBc-positive were reported as positive. Where available, this correlated well with the results of parallel blood samples. Patients who re-attended for confirmatory blood tests were tested for anti-HCV using commercial, validated assays and were tested for chronic HBV infection [HBsAg and hepatitis B e antigen (HBeAg)/anti-HBe] using standard assays in accredited laboratories. Patients who had anti-HCV were tested for HCV RNA using commercial polymerase chain reaction assays, and patients with detectable HBsAg were tested for HBV DNA by standard assays.

At the conclusion of the study, patients who had declined to re-attend for confirmatory blood tests were re-assessed. For patients with HBsAg in oral fluid who did not have a blood test, the original oral fluid was retested for anti-HBc (in patients in whom this had not been tested), and the diagnosis of chronic HBV infection made if both HBsAg and anti-HBc were present. For patients who had anti-HCV detected in oral fluid but did not re-attend, the samples were re-assessed based on the results obtained from patients who did undergo confirmatory testing, and a decision regarding infectivity made. (Supporting Information contains the details of the testing regime and outlines the procedures used to assign infection status).

Designation of origin

The aim of the study was to test people of south Asian ethnicity (ancestors who originated from India, Pakistan or

Bangladesh), and ethnicity was defined by ancestral origin. Thus, people attending sessions in Hindu temples who were of Indian descent but who were born to Indian parents in Africa (in Uganda, Tanzania or Kenya) were designated 'Indian', although their African origin was noted. For people of Pakistani origin, we assigned people to one of five regions and included the separate administrative area of Islamabad within the Punjab.

Statistical analysis and ethical approval

The study was powered to detect a higher prevalence of viral hepatitis in the south Asian population that was of sufficient magnitude to justify case finding. After discussion, we assumed that a prevalence five times higher than the UK average (assumed to be 0.4% for HCV and 0.1% prevalence for HBsAg) would justify case finding in ethnic minorities. We therefore estimated that a minimum of 1000 patients from the three different communities from the Indian sub-continent (Indian, Pakistani and Bangladeshi) would provide a power of 80% with 95% confidence intervals. A secondary objective was to determine whether the prevalence of hepatitis C varied between those born abroad and those born in the UK, so we aimed to test a total of 1000 people of south Asian ethnicity who were born in the UK. Based on interim analyses that suggest a particularly high prevalence in those of Pakistani origin, the number of individuals in this group was increased to over 2000 so that additional risk factors (such as region of origin) could be studied.

Statistical analysis was performed by spss version 14 and to further define groups that may be at particular risk of chronic HCV infection, tests for interactions were performed, collapsing categories where necessary because of low numbers. The study was approved by the appropriate local research ethics committees.

RESULTS

We tested volunteers in a total of 52 different sites from the five different regions of England. A total of 4381 first generation immigrants who originated in the Indian sub-continent (India, Bangladesh or Pakistan) were screened in the community along with 446 people whose parents were born in the Indian sub-continent and six people whose grandparents were born there (three with family origins in Pakistan and three with family origins in Bangladesh).

Table 1 summarizes the results. In 1197 people born in India, or who originated in India and came to the UK via Africa (Kenya $N = 131$, Tanzania $N = 23$ or Uganda $N = 69$), the prevalence of viral hepatitis was low in two patients with hepatitis C (both were viraemic), and one patient with hepatitis B. In 726 people born in Bangladesh (the vast majority of whom originated from Sylhet), chronic HCV exposure was uncommon (four patients – 0.6%), but chronic HBV was prevalent (1.5%). An additional 171 people of Bangladeshi origin were tested in a general practice in East London. As there was no infections detected in this group and the prevalence did not differ from that seen in those who attended sessions in community centres, these results were pooled for the analysis of risk factors for exposure. In the 2458 individuals who were born in Pakistan, the prevalence of anti-HCV was 2.7% and that of chronic hepatitis B infection (HBsAg-positive) 1.8%.

In the 57 patients who had anti-HCV and who re-attended for confirmatory blood tests, viraemia by PCR testing was detected in 55 (96%). In 45 patients in whom a viral genotype was determined, 41 were infected with genotype 3. For chronic HBV infection, 48 of 58 patients re-attended – four patients were HBeAg-positive, and of the 44 patients who were HBeAg-negative, 10 had viral loads of >10 000 IU/mL indicating active disease, whilst 12 patients had very low level of HBV DNA (<100 IU/mL). Four patients (all from Pakistan) were anti-HCV-positive and HBsAg-positive. These data indicate that nearly one in twenty people born in Pakistan and living in England has chronic viral hepatitis.

We studied 441 individuals who were born in the UK to parents of south Asian origin. A total of 1/353 (0.3%), 1/83 (1.2%) and 1/21 (4.8%) were positive for HBsAg, and 1/353 (0.3%), 0/81 (0%) and 0/21 (0%) were anti-HCV-positive amongst those of Pakistani, Indian and Bangladeshi origin, respectively, giving an overall prevalence of 0.5% for HCV and 0.2% for HBV. Of note was the prevalence in the 353 individuals who were born in the UK to parents from Pakistan which was low (0.3% for both HBV and HCV).

Logistic regression showed a higher odds of hepatitis C infection in people from Pakistan (Table 2), particularly those from the Punjab. People who came to the UK recently (within the last 20 years) were also more likely to be infected than those who had lived in the UK for a longer period of time – prevalence rates were 3.8% and 1.5% in people coming to the UK within the last 10 or 20 years, respectively, compared to a prevalence rate of around 0.8% in those arriving in the UK in the more distant past. People who were tested in East London were more likely to be infected than people tested in other areas, and this difference remained significant by multivariate analysis.

There was a strong interaction between centre and sex, with both East London and Bradford showing an excess prevalence mainly in males ($P = 0.0021$). All other tests for interactions were not significant.

For chronic HBV infection, odds of infection were higher in those born in Pakistan and Bangladesh than those born in India or the UK (Table 3). Unlike chronic HCV infection, there was no association with region of origin within Pakistan, and length of stay in the UK did not influence the prevalence.

Table 1 Prevalence of viral hepatitis in people of south Asian origin. Prevalence of anti-HCV and hepatitis B surface antigen in oral fluid by ethnicity/place of birth in people tested in community centres

	Bangladesh			India			Pakistan			UK			Total		
	N	HCV	HBV	*N*	HCV	HBV	*N*	HCV	HBV	*N*	HCV	HBV	*N*	HCV	HBV
Age, females															
16–29	32	0 (0.0%)	0 (0.0%)	20	1 (5.0%)	0 (0.0%)	113	3 (2.7%)	0 (0.0%)	116	0 (0.0%)	0 (0.0%)	281	4 (1.4%)	0 (0.0%)
30–39	62	0 (0.0%)	1 (1.6%)	54	0 (0.0%)	0 (0.0%)	147	5 (3.4%)	2 (1.4%)	54	1 (1.9%)	0 (0.0%)	317	6 (1.9%)	3 (0.9%)
40–49	47	0 (0.0%)	0 (0.0%)	84	0 (0.0%)	0 (0.0%)	206	7 (3.4%)	6 (2.9%)	19	0 (0.0%)	0 (0.0%)	356	7 (2.0%)	6 (1.7%)
50–59	54	1 (1.9%)	0 (0.0%)	184	0 (0.0%)	0 (0.0%)	177	5 (2.8%)	3 (1.7%)	1	0 (0.0%)	0 (0.0%)	416	6 (1.4%)	3 (0.7%)
60–69	33	0 (0.0%)	0 (0.0%)	182	0 (0.0%)	0 (0.0%)	97	1 (1.0%)	2 (2.1%)	2	0 (0.0%)	0 (0.0%)	314	1 (0.3%)	2 (0.6%)
≥70	9	0 (0.0%)	0 (0.0%)	114	0 (0.0%)	0 (0.0%)	56	3 (5.4%)	0 (0.0%)	0	0 (NA)	0 (NA)	179	3 (1.7%)	0 (0.0%)
Age, males															
0–29	59	0 (0.0%)	1 (1.7%)	30	0 (0.0%)	0 (0.0%)	249	8 (3.2%)	2 (0.8%)	143	0 (0.0%)	1 (0.7%)	481	8 (1.7%)	4 (0.8%)
30–39	125	0 (0.0%)	2 (1.6%)	45	1 (2.2%)	0 (0.0%)	352	13 (3.7%)	8 (2.3%)	77	1 (1.3%)	0 (0.0%)	599	15 (2.5%)	10 (1.7%)
40–49	113	0 (0.0%)	3 (2.7%)	98	0 (0.0%)	0 (0.0%)	300	6 (2.0%)	8 (2.7%)	24	0 (0.0%)	0 (0.0%)	535	6 (1.1%)	11 (2.1%)
50–59	57	0 (0.0%)	2 (3.5%)	146	0 (0.0%)	0 (0.0%)	361	7 (1.9%)	7 (1.9%)	7	0 (0.0%)	0 (0.0%)	571	7 (1.2%)	9 (1.6%)
60–69	63	1 (1.6%)	0 (0.0%)	123	0 (0.0%)	0 (0.0%)	191	4 (2.1%)	3 (1.6%)	5	0 (0.0%)	0 (0.0%)	382	5 (1.3%)	3 (0.8%)
≥70	72	2 (2.8%)	2 (2.8%)	117	0 (0.0%)	1 (0.9%)	209	5 (2.4%)	3 (1.4%)	4	0 (0.0%)	0 (0.0%)	402	7 (1.7%)	6 (1.5%)
Total by sex															
Females	237	1 (0.4%)	1 (0.4%)	638	1 (0.2%)	0 (0.0%)	796	24 (3.0%)	13 (1.6%)	192	1 (0.5%)	0 (0.0%)	1863	27 (1.4%)	14 (0.8%)
Males	489	3 (0.6%)	10 (2.0%)	559	1 (0.2%)	1 (0.2%)	1662	43 (2.6%)	31 (1.9%)	260	1 (0.4%)	1 (0.4%)	2970	48 (1.6%)	43 (1.4%)
Length of UK stay															
0–9	137	1 (0.7%)	2 (1.5%)	131	2 (1.5%)	1 (0.8%)	610	30 (4.9%)	12 (2.0%)	0	0 (NA)	0 (NA)	878	33 (3.8%)	15 (1.7%)
10–19	159	0 (0.0%)	4 (2.5%)	92	0 (0.0%)	0 (0.0%)	332	10 (3.0%)	4 (1.2%)	67	0 (0.0%)	0 (0.0%)	650	10 (1.5%)	8 (1.2%)
20–29	177	1 (0.6%)	3 (1.7%)	142	0 (0.0%)	0 (0.0%)	314	5 (1.6%)	8 (2.5%)	192	0 (0.0%)	1 (0.5%)	825	6 (0.7%)	12 (1.5%)
30–39	100	1 (1.0%)	1 (1.0%)	345	0 (0.0%)	0 (0.0%)	430	5 (1.2%)	8 (1.9%)	131	2 (1.5%)	0 (0.0%)	1006	8 (0.8%)	9 (0.9%)
≥40	127	1 (0.8%)	1 (0.8%)	348	0 (0.0%)	0 (0.0%)	531	8 (1.5%)	9 (1.7%)	62	0 (0.0%)	0 (0.0%)	1068	9 (0.8%)	10 (0.9%)
Missing	26	0 (0.0%)	0 (0.0%)	139	0 (0.0%)	0 (0.0%)	241	9 (3.7%)	3 (1.2%)	0	0 (NA)	0 (NA)	406	9 (2.2%)	3 (0.7%)
Testing centre															
Bradford	112	0 (0.0%)	2 (1.8%)	65	1 (1.5%)	0 (0.0%)	950	16 (1.7%)	20 (2.1%)	260	2 (0.8%)	0 (0.0%)	1387	19 (1.4%)	22 (1.6%)
East London	598	4 (0.7%)	9 (1.5%)	220	1 (0.5%)	1 (0.5%)	1076	44 (4.1%)	18 (1.7%)	81	0 (0.0%)	0 (0.0%)	1975	49 (2.5%)	28 (1.4%)
Sandwell	0	0 (NA)	0 (NA)	229	0 (0.0%)	0 (0.0%)	204	3 (1.5%)	1 (0.5%)	15	0 (0.0%)	0 (0.0%)	448	3 (0.7%)	1 (0.2%)
Walsall	16	0 (0.0%)	0 (0.0%)	321	0 (0.0%)	0 (0.0%)	228	4 (1.8%)	5 (2.2%)	79	0 (0.0%)	1 (1.3%)	644	4 (0.6%)	6 (0.9%)
West London	0	0 (NA)	0 (NA)	362	0 (0.0%)	0 (0.0%)	0	0 (NA)	0 (NA)	17	0 (0.0%)	0 (0.0%)	379	0 (0.0%)	0 (0.0%)
Total (row %)															
Total	726	4 (0.6%)	11 (1.5%)	1197	2 (0.2%)	1 (0.1%)	2458	67 (2.7%)	44 (1.8%)	452	2 (0.4%)	1 (0.2%)	4833	75 (1.6%)	57 (1.2%)

HCV, hepatitis C virus; HBV, hepatitis B virus.

Table 2 Multivariable analysis of factors that may influence the prevalence of chronic hepatitis C. The variables noted were assessed in people tested in the community using oral mucosal fluid testing

			Univariate		Multivariate	
	N	Hepatitis C virus (%)	OR (95% CI)	*P*	OR (95% CI)	*P*
Age						
16–29	762	12 (1.6%)	1 (ref)		1 (ref)	
30–39	916	21 (2.3%)	1.47 (0.72, 3.00)	0.295	1.75 (0.83, 3.68)	0.141
40–49	891	13 (1.5%)	0.93 (0.42, 2.04)	0.848	1.58 (0.68, 3.69)	0.285
50–59	987	13 (1.3%)	0.83 (0.38, 1.84)	0.653	1.89 (0.78, 4.61)	0.161
60–69	696	6 (0.9%)	0.54 (0.20, 1.46)	0.225	1.65 (0.55, 4.90)	0.370
≥70	581	10 (1.7%)	1.09 (0.47, 2.55)	0.834	3.53 (1.30, 9.63)	0.014
				(0.320)		(0.263)
Sex						
Female	1863	27 (1.4%)	1 (ref)		1 (ref)	
Male	2970	48 (1.6%)	0.90 (0.56, 1.44)	0.648	1.03 (0.62, 1.71)	0.921
Country/region						
UK	452	2 (0.4%)	1 (ref)		1 (ref)	
India	1197	2 (0.2%)	0.38 (0.05, 2.68)	0.329	0.24 (0.03, 1.94)	0.181
Bangladesh	726	4 (0.6%)	1.25 (0.23, 6.83)	0.800	0.36 (0.06, 2.15)	0.260
Pakistani: Punjab	1049	39 (3.7%)	8.69 (2.09, 36.14)	0.003	2.63 (0.57, 12.26)	0.217
Pakistani: Kashmir	570	3 (0.5%)	1.19 (0.20, 7.16)	0.849	0.56 (0.09, 3.62)	0.542
Pakistani: Other	239	2 (0.8%)	1.90 (0.27, 13.56)	0.523	0.45 (0.06, 3.55)	0.450
Pakistani: Unknown	600	23 (3.8%)	8.97 (2.10, 38.24)	0.003	3.55 (0.75, 16.85)	0.111
				(0.000)		(0.000)
Length of UK stay						
0–9	878	33 (3.8%)	1 (ref)		1 (ref)	
10–19	650	10 (1.5%)	0.40 (0.20, 0.82)	0.012	0.47 (0.22, 0.98)	0.045
20–29	825	6 (0.7%)	0.19 (0.08, 0.45)	0.000	0.28 (0.11, 0.73)	0.009
30–39	1006	8 (0.8%)	0.21 (0.09, 0.45)	0.000	0.29 (0.12, 0.69)	0.005
≥40	1068	9 (0.8%)	0.22 (0.10, 0.46)	0.000	0.25 (0.10, 0.63)	0.003
Missing	406	9 (2.2%)	0.58 (0.28, 1.22)	0.153	1.06 (0.43, 2.65)	0.895
				(0.000)		(0.002)
Testing centre						
Other	1471	7 (0.5%)	1 (ref)		1 (ref)	
East London	1975	49 (2.5%)	5.32 (2.40, 11.78)	0.000	4.55 (1.80, 11.53)	0.001
Bradford	1387	19 (1.4%)	2.90 (1.22, 6.93)	0.016	2.37 (0.94, 5.99)	0.069
				(0.000)		(0.004)

DISCUSSION

This is the first large-scale study to address the prevalence of viral hepatitis in those of south Asian ethnic origin in England and one of the largest studies of viral hepatitis in migrants. Previous studies in the UK have suggested higher rates of hepatitis B infection in south Asian antenatal women [12] and blood donors [13]. However, no previous studies have targeted people of south Asian origin who had emigrated from their country of origin, and worldwide, there is very limited information on the impact of viral hepatitis on immigrant communities from this region. Overall, our study shows that the prevalence of hepatitis B and hepatitis C was higher than would be expected in the indigenous UK population, and the prevalence of viral hepatitis was particularly high in people originating in Pakistan. In England, the overall prevalence of anti-HCV infection in adults has been estimated at around 0.53% [14], similar to the prevalence we observed in those born in Bangladesh, India and the UK, but substantially lower than the prevalence in those born in Pakistan. Estimates of overall HBV prevalence in England are less well defined, although one study of residual samples found a prevalence of 0.3% [15], similar to the level we observed in those born in India and the UK, but lower than those born in Bangladesh and Pakistan. Although the numbers of individuals of south Asian origin born in the UK were relatively small, the low prevalence in this population suggests that most chronic hepatitis infection was acquired prior to arrival in England.

Table 3 Multivariable analysis of factors that may influence the prevalence of chronic hepatitis B. The variables noted were assessed in people tested in the community using oral mucosal fluid testing

			Univariate		Multivariate	
	N	Hepatitis B virus (%)	OR (95% CI)	*P*	OR (95% CI)	*P*
Age						
16–29	762	4 (0.5%)	1 (ref)		1 (ref)	
30–39	916	13 (1.4%)	2.73 (0.89, 8.40)	0.080	2.74 (0.86, 8.69)	0.088
40–49	891	17 (1.9%)	3.69 (1.23, 11.00)	0.019	4.38 (1.34, 14.27)	0.014
50–59	987	12 (1.2%)	2.33 (0.75, 7.26)	0.144	3.51 (0.99, 12.42)	0.051
60–69	696	5 (0.7%)	1.37 (0.37, 5.13)	0.639	2.71 (0.62, 11.83)	0.184
≥70	581	6 (1.0%)	1.98 (0.56, 7.04)	0.293	3.54 (0.83, 15.16)	0.088
				(0.151)		(0.271)
Sex						
Female	1863	14 (0.8%)	1 (ref)		1 (ref)	
Male	2970	43 (1.4%)	0.52 (0.28, 0.94)	0.032	0.61 (0.32, 1.17)	0.137
Country/region						
UK	452	1 (0.2%)	1 (ref)		1 (ref)	
India	1197	1 (0.1%)	0.38 (0.02, 6.04)	0.491	0.26 (0.01, 4.61)	0.356
Bangladesh	726	11 (1.5%)	6.94 (0.89, 53.92)	0.064	3.73 (0.43, 32.79)	0.235
Pakistani: Punjab	1049	16 (1.5%)	11.36 (1.49, 86.69)	0.019	6.33 (0.76, 52.59)	0.088
Pakistani: Kashmir	570	14 (2.5%)	6.99 (0.92, 52.83)	0.060	3.76 (0.44, 31.77)	0.224
Pakistani: Other	239	7 (2.9%)	13.61 (1.66, 111.26)	0.015	7.61 (0.82, 70.66)	0.074
Pakistani: Unknown	600	7 (1.2%)	5.32 (0.65, 43.43)	0.118	3.23 (0.36, 28.87)	0.295
				(0.005)		(0.028)
Length of UK stay						
0–9	878	15 (1.7%)	1 (ref)		1 (ref)	
10–19	650	8 (1.2%)	0.72 (0.30, 1.70)	0.450	0.64 (0.26, 1.58)	0.167
20–29	825	12 (1.5%)	0.85 (0.40, 1.83)	0.675	0.95 (0.40, 2.26)	0.120
30–39	1006	9 (0.9%)	0.52 (0.23, 1.19)	0.122	0.51 (0.20, 1.32)	0.280
≥40	1068	10 (0.9%)	0.54 (0.24, 1.22)	0.138	0.44 (0.15, 1.24)	0.903
Missing	406	3 (0.7%)	0.43 (0.12, 1.49)	0.182	0.48 (0.12, 1.83)	0.653
				(0.487)		(0.489)
Testing centre						
Other	1471	7 (0.5%)	1 (ref)		1 (ref)	
East London	1975	28 (1.4%)	3.01 (1.31, 6.90)	0.005	1.06 (0.41, 2.75)	0.903
Bradford	1387	22 (1.6%)	3.37 (1.44, 7.92)	0	1.23 (0.49, 3.10)	0.653
				(0.016)		(0.853)

All prevalence studies are hampered by selection bias, and our study is potentially biased by the selection of testing sites and by 'self selection' in those coming forward for testing. We chose to screen people in community and religious centres because this represents a convenient, nonadversarial way of accessing a 'healthy' population. We considered random selection of individuals within the community, but we rejected this on the advice of community advocates. Community centre testing proved popular but may access a self selected group. An alternative approach using a local general practice was piloted but proved labour intensive. Multiple visits to the practice were required to test a relatively small number of subjects, and this approach was not pursued further. We postulated that our community sample or recruitment method may have attracted only those at low risk but that those attending GP surgeries may be more likely to have health problems including viral hepatitis. This is of particular relevance as chronic HCV infection can cause a variety of nonspecific symptoms including malaise, fatigue and arthralgia [16]. We found a similar or lower prevalence of viral hepatitis in patients attending the surgery, suggesting that our community study is representative.

Community testing requires a rapid method that avoids the risk of disseminating bloodborne viruses. We rejected blood testing because of the practical difficulties of testing large numbers of individuals and avoiding exposure to sharps and potential viral transmission. The oral fluid testing system permitted rapid, safe testing in a community setting using a range of staff. Oral fluid assays have been widely tested in patients with chronic HCV infection where anti-

HCV was detected with a sensitivity of 92% [10]. The major disadvantage is that if the patient does not re-attend for a confirmatory blood test, it is impossible to assess viraemia and false-positives may be difficult to detect. Because not all the patients who had positive oral swab tests re-attended, we were unable to confirm all of the oral swab results by blood testing. To ensure the validity of the oral swab results, we re-examined and recalibrated all of our positive HCV oral fluid tests based upon the results obtained from those patients who had re-attended for confirmatory blood tests. Scrutiny of the optical density values from the ELISA assay allowed us to assign a probable diagnosis in nearly all patients, although we accept that some patients may have been incorrectly assigned. However, re-analysis of the data excluding these 'dubious' samples did not materially alter the conclusions. The failure of some people who were detected by oral fluid testing in the community to re-attend for further evaluation is an area of concern. We made great efforts to persuade all those who tested positive in our oral fluid assay to re-attend (including visiting them in their homes and offering them a taxi service to and from the local hospital), but a proportion of patients were adamant that they did not wish to access services for their infection. It will be important to determine why a substantial proportion of patients decline to access support for chronic viral hepatitis to facilitate future case finding in these communities.

We were disappointed that our approach failed to identify and test a large cohort of people of south Asian ethnicity who were born in the UK. It is unclear why this is the case and may be related either to our choice of testing sites or to a decision on the part of 'second generation' immigrants that they are not 'at risk' and therefore did not present for testing. Because many 'second generation' immigrants attended the Mosques and centres where we tested for viral hepatitis (and played an important part in the study by acting as interpreters), we favour the latter explanation. Although the numbers of people of south Asian origin born in the UK were small, we did not find evidence of a high prevalence of viral hepatitis in more than 300 'second generation' immigrants from Pakistan, suggesting that these individuals are at lower risk of infection than people who were born in Pakistan. We were unable to determine whether or not people born in the UK to parents of south Asian origin have an increased rate of infection when compared to the indigenous population, and further studies will be required to address this issue.

The pattern of infection differed markedly for hepatitis B and hepatitis C and between the different countries of origin. For chronic HBV infection, the prevalence was similar in migrants from Pakistan and Bangladesh, where the prevalence was in line with WHO estimates, but the prevalence was lower in people from India. The pattern of infection for chronic HCV was more complex. Again immigrants from India were unlikely to be infected, but those from Bangladesh were found to have a low prevalence of chronic HCV infection despite a high prevalence of chronic HBV infection and reports of high rates of HCV infection in Bangladesh [17]. One plausible explanation is that studies of HCV prevalence in Bangladesh have been performed in urban areas, and most UK immigrants from Bangladesh originate from the rural area of Sylhet where the prevalence has not been studied. For people originating from Pakistan, the prevalence of chronic HCV infection was similar to WHO estimates, but we found marked regional variation. Immigrants from the Punjab had high rates of infection, whereas immigrants from other areas were much less likely to be infected. Surprisingly, recent immigrants from Pakistan were much more likely to be infected than immigrants who had come to the UK many years ago. It is not clear whether these regional and temporal differences reflect changes in the prevalence within Pakistan itself or are related to different migration patterns from different regions over time. Whatever the explanation for these differences, it is clear that simple extrapolation from the current overall prevalence in the country of origin is unlikely to give an accurate picture of the immigrant community in the UK.

Studies in other western countries, including France and in the USA, have shown high rates of hepatitis in migrants, similar to those in the country of origin [7,8]. In contrast, one small study of immigrants from the former Soviet Union living in New York found a higher prevalence of chronic HCV infection than would be expected [18]. No previous studies agree with our observations in the Indian-born population, which show substantially lower rates of infection than would be expected in the country of origin. The reasons for the lower prevalence of hepatitis in people from India may relate to selective migration of younger, healthier and more affluent migrants (the 'healthy migrant' effect) [19]. Alternatively, our selection and recruitment methods may be less suitable for identifying a representative sample of the Indian population.

The impact of chronic viral hepatitis on the communities we have studied cannot be determined from this prevalence study. Nearly all of the people who had detectable anti-HCV were viraemic. This is in contrast to other studies where a proportion of patients have evidence of exposure but not infection. It is not clear whether this represents a tendency for genotype 3 HCV infection, which predominated in this community, to either progress to chronic infection or whether antibodies have waned, leading to under-detection of exposed and not infected individuals in our oral swab test. Studies in East London suggest that a high proportion of elderly patients from Pakistan have cirrhosis [20], and population-based studies indicate that there is an increase in end-stage liver disease from HCV infection from people born in Pakistan and Bangladesh [21], strongly suggesting that the high prevalence of chronic HCV infection in people from Pakistan is likely to lead to significant liver disease in this population in the near future. The impact of chronic HBV infection is more difficult to assess. A proportion of patients detected in this study had evidence of on-going liver disease,

but many were in a quiescent phase of infection (HBsAg-positive with undetectable HBV DNA and normal liver function tests). In view of the tendency for patients with chronic HBV to develop liver disease after many decades and the risks of infecting others, it is likely that this infection will impact upon the immigrant communities in due course.

The high prevalence in certain groups of the south Asian community, in particular those born in Pakistan, would lend support to efforts to identify and treat infected individuals before they develop significant disease. Our study has suggested one mechanism for case finding, but other approaches including that in general practice need further evaluation.

This is the first detailed study of viral hepatitis in immigrants from countries of high prevalence living in England. Although we have no data on the prevalence of viral hepatitis in immigrants in other countries, we suspect that the prevalence will be similar. Our study shows that applying current WHO estimates of prevalence in the country of origin are likely to be too crude for health care planning. Geographical variation in prevalence and 'selective' migration, which may differ over time, may confound attempts to estimate prevalence based upon available data. The suggestion that up to one in 20 migrants from Pakistan may be chronically infected with harmful but treatable viruses that can be transmitted to others is worrying, given that over 300 000 individuals were living in the UK at the time of the 2001 census. We believe that these data provide strong justification for a policy of case finding in this population.

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CONTRIBUTIONS

GRF designed the study, raised the funds, helped to collect and analyse the data. He is the guarantor for the study. GU, DS, SS and RM helped design and run the study. MR and RH helped design the study and completed the statistical analyses. I U-L, WCF and SC performed the virological and serological analyses. HCT, SK, SM, SA, BW, NP, RJ, AH and SS set up and managed the external sites as well as helped to develop the study, analyse the data and write the paper.

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CONFLICTS OF INTEREST

Professor Foster has received grant funding and consultancy fees from companies who market drugs for the treatment of viral hepatitis, including Roche, Gilead, Novartis and BMS.

Professor Thomas's department has received funds for conducting studies on therapy of viral hepatitis from Schering Plough, Vertex, Gilead, GSK, Novartis and BMS and has been supported by a NIHR Biomedical Research Centre Award to Imperial College.

Dr Singhal has received educational funding support from companies who market drugs for the treatment of viral hepatitis, including Roche, Schering-Plough and Gilead.

Professor Carman and Dr Cameron act as consultants to Altrix who market the oral fluid testing device used in this study.

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