

## SHORT COMMUNICATIONS

**Hepatitis B virus markers in blood donors  
in the west of Scotland\***A. BARR, S. R. HOUSTON, I. P. MACVARISH, B. C. DOW, R. MITCHELL  
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Lanarkshire, Scotland**(Received 10 March 1981)**Key words:* hepatitis B antigens; blood banks; blood donors; blood transfusion.

The Glasgow and West of Scotland Blood Transfusion Service collects in the region of 140 000 donations annually from an area with a mixed rural and industrial population with no major ethnic variations.

The incidence of hepatitis B surface antigen (HBsAg) in our blood donors is shown in Table 1. In over 10 years of total screening we have tested in excess of 1 000 000 blood donations. Despite the high incidence of HBsAg in male prisoners (1 in 145) viral hepatitis is not a serious clinical problem in the institution surveyed, and the positive donors are not drug addicts. This high incidence is probably related to social habits and hygiene.

TABLE 1  
HBsAg screening of 1 302 234 donors 1970-1980

Donors tested for the first time	Number	Incidence of HBsAg positive
Male	211 506	1 in 624
Institutionalised	6 234	1 in 145
Non-institutionalised	205 272	1 in 693
Female	134 638	1 in 1795
All donors	346 144	1 in 836

In almost 4 years of total screening by radioimmunoassay (RIA) only two cases of confirmed post-transfusion hepatitis B have come to our attention, and in neither case were we able to demonstrate HBsAg in the donor's blood.

The first case involved a lady with ulcerative colitis who was transfused with 3 units of red cells. Four months later she was readmitted to hospital. Her liver function tests were elevated, HBsAg was detected in her serum, and acute hepatitis was confirmed by liver biopsy. Antibody against hepatitis B core antigen (anti-HBc) was also present, and in due course HBsAg disappeared and anti-HBs appeared. There can be little doubt that her acute attack of hepatitis was of type B. The stored sera of the three blood donors were re-examined and neither HBsAg nor anti-HBs were detected. One of the donors, however, had a high-

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TABLE 2

	Patient (RM)	Husband	Son	Donor
HBsAg	+	-	-	-
Anti-HBs	-	+	-	-
Anti-HBc	+	+	+	+

titre of anti-HBc. The donor's health record showed no history of illness to support the idea of a recent hepatitis infection. No history of other exposures to the virus were noted and he had been a conscientious donor for many years. Recent serum samples from the donor now reveal a weak anti-HBs in addition to the high titre anti-HBc.

It would appear that this case of post-transfusion hepatitis B implicated a donor with anti-HBc as the sole marker. Being cautious about reporting our results, we decided to look at the close contacts of the patient. From Table 2 it may be seen that anti-HBs was detected in her husband, and high titre anti-HBc in both her husband and son. Neither showed evidence of recent or past illness suggestive of clinical hepatitis. Three questions still remain unanswered. (i) Did the blood donation cause hepatitis in the recipient? (ii) Did the recipient then expose her family to the virus? (iii) Was exposure from family contacts the source of the illness? The donor has at present been suspended from our active donor panel.

The second case involved a patient (WL) undergoing treatment in a renal dialysis unit. Such patients are routinely monitored for the presence of HBsAg by the regional virus laboratory and in July 1980 this patient was found to be HBsAg positive. Other samples available from this patient from earlier months were immediately re-examined for HBsAg and other markers (Fig. 1).

1980: Month	January	February	March	April	May	June	July	August	September	October	November	December
HBsAg	-	-	-	-	-	-	+	+	-	-	-	-
HBeAg	-	-	-	-	-	-	(+)	+	-	-	-	-
Anti-HBc	-	-	-	+	(+)	-	-	-	+	+	+	+
Anti-HBe	-	-	-	-	-	-	-	-	+	+	+	+
Anti-HBs	-	-	-	-	-	-	-	-	-	-	+	(+)
Blood given	1	1	1	1	1	1	1	-	-	-	-	-

Fig. 1. Results of testing WL for all HBV markers.

During the period January to March, no hepatitis B virus (HBV) markers were detected in the patient. Anti-HBc was detected with no evidence of other markers during April and May. By June there were no apparent hepatitis markers, but by July HBsAg and weak HBeAg were present. Further monitoring revealed the loss of HBsAg and hepatitis Be antigen (HBeAg), with sero-conversion to anti-HBe, anti-HBc, and eventually anti-HBs. The patient had no clinical symptoms of hepatitis. These results suggested that the patient had suffered from acute sub-clinical hepatitis B, possibly transmitted by one of the nine donations of blood transfused during the period February to July. Stored samples from each of the nine donations were re-examined for HBV markers. The negative results for HBsAg were confirmed but a high level of anti-HBc, accompanied by a weak anti-HBs, was found in one of the donors. A repeat sample from this donor (NM) confirmed these results. The donations from NM had been transfused to the patient on 24 April, five days before the initial finding of anti-HBc in the patient.

Collectively, this information suggests that the donor was probably the source of the initial weak anti-HBc detected in the patient and - more importantly - the likely cause of the post-transfusion hepatitis in the patient.

These two cases raise the question of whether donations should be screened for anti-HBc as well as HBsAg. We have not looked at this problem in depth, but Table 3 records the results of a small study, carried out in a selected group from one of Her Majesty's prisons, and a control group matched for sex and age.

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TABLE 3  
Incidence of Anti-HBs and Anti-HBe in donors tested by  
RIA

	Institutional	Non-institutional
Number tested	395	395
Anti-HBs only	4	5
Anti-HBs Anti-HBe	18	3
Anti-HBe only	6	1

The incidence of HBsAg in male prisoners is significantly higher than that in the random donor population, but the results indicate the problem blood transfusion centres could have in sorting out anti-HBs positive donors from the anti-HBe positive donors. It could also be argued that blood destined for specialised (e.g. renal dialysis) units, and blood taken from known risk groups, should be simultaneously screened for the presence of HBsAg and anti-HBe.

Most workers now agree the relatively high infectivity associated with the persistence of HBeAg. Conversely, sero-conversion of anti-HBe is thought to be indicative of recovery from the viral attack and is associated with low infectivity. Our results of testing 246 HBsAg positive donors in the West of Scotland have shown that approximately 1 in every 5 HBsAg positive donors is HBeAg positive.<sup>1</sup> The percentage of carriers with HBeAg declined with increasing age. With a few exceptions, the mean HBsAg concentration is four times greater in donors with HBeAg than in those with anti-HBe. No significant associations were found with HBsAg subtypes, sex or blood groups.

Recently the anti-HBe status of 246 HBsAg positive donors was investigated (Table 4). Only nine donors were negative for anti-HBe, two of which could not be classified as HBeAg or anti-HBe and also had low HBsAg levels. Seven were from donors who were HBeAg positive. All anti-HBe positive donors were found to have anti-HBe. So far nine of our HBsAg positive donors have been followed up. Five remained HBeAg and HBsAg positive, one had sero-converted to anti-HBe whilst remaining HBsAg positive, and three had sero-converted to anti-HBe and anti-HBs.

TABLE 4  
Relationship of Anti-HBe to HBeAg/Anti-HBe status of 246 HBsAg positive donors

	Anti-HBe positive	Anti-HBe negative
HBeAg	38 (84.4%)	7 (15.6%)
Anti-HBe	197 (100%)	0
Neither	2 (50%)	2 (50%)
Total	237 (96.3%)	9 (3.7%)

In summary, all the Hepatitis B Virus markers offer practical advantages to blood transfusion services. Firstly, a sensitive method of screening for HBsAg should prevent most cases of post-transfusion hepatitis B. Secondly, selective screening for anti-HBe may be carried out to prevent introduction of Hepatitis B virus into 'at risk' areas such as renal dialysis units. Finally, determination of the HBe status of HBsAg carriers may be helpful in reassuring donors and in offering rational advice to nursing, medical and dental personnel concerned with their care.

## Reference

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### BLOOD-DONORS WITH HISTORY OF JAUNDICE

SIR,—Both the Department of Health and Social Security (1975) and the World Health Organisation (1977) now advise that, provided sera are tested for HB<sub>s</sub>Ag, there is no need to turn away blood-donors who have a history of jaundice. Renton et al.<sup>1</sup> explored the likely effect of this policy change and examined the relation between jaundice history and hepatitis B surface antigen (HB<sub>s</sub>Ag) carriage among blood-donors. The history was recorded at the time of donation.

447 HB<sub>s</sub>Ag-positive donors have been found in the West of Scotland since testing was introduced in 1970. 13 (2.9%) of these donors admitted to a history of jaundice. This proportion is very similar to the proportion (2.8%) of those with a history of jaundice among 228 631 donors in our active donor file and to the proportion (2.6%) found in a sample of 7460 new donors who first gave blood in 1978–79. If a jaundice history was an important determinant of HB<sub>s</sub>Ag carriage it should have been present in a higher proportion of these HB<sub>s</sub>Ag positive donors.

Our active donor file records all donors who have given blood since January, 1975, and the prevalence of HB<sub>s</sub>Ag among jaundice-history donors is as follows:

History	Total	Hb <sub>s</sub> Ag positive
No jaundice	222 249	193 (0.087%)
jaundice	6 382	9 (0.141%)

$\chi^2=1.495$ ; 1 d.f.;  $0.25 > p > 0.20$ .

The record includes new and previously screened donors but the comparison seems to be valid. If there is a real difference it is likely to be small. The Manchester workers<sup>1</sup> found more new donors with a history of jaundice (6.3%) than we did (2.6%) and the prevalence of HB<sub>s</sub>Ag in their new donors was lower (0.078% compared with 0.129%). Methods of testing have varied but almost all our donors have been tested by sensitive radioimmunoassay methods. Wallace<sup>2</sup> suggested that radioimmunoassay methods might give a different pattern of results from those of Renton et al.

In studies to be reported separately we have tested the sera of donors with a history of jaundice for other markers of prior exposure to both hepatitis B and hepatitis A viruses by radioimmunoassay methods. Two markers for hepatitis B were used—antibody to hepatitis B surface (anti-HB<sub>s</sub>) and to hepatitis B core (anti-HB<sub>c</sub>). As with HB<sub>s</sub>Ag there was no evidence that the prevalence of these markers in donors with a jaundice history was different from that in the random population. On the other hand the marker for hepatitis A virus, antibody to hepatitis A (anti-HAV) was significantly more common in the jaundice group than in the controls. Carriage of HB<sub>s</sub>Ag tends to follow subclinical infection and most of our HB<sub>s</sub>Ag donors have never had clinical hepatitis. Of those HB<sub>s</sub>Ag carriers who have had jaundice most will have antibody to HAV. We have tested 8 and found anti-HAV in 6.

We conclude from these results that a history of jaundice does not materially increase the prevalence of HB<sub>s</sub>Ag among blood-donors and is likely to imply previous infection with HAV rather than with HBV.

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### BOTTLE-FEEDING AND THE LAW IN PAPUA NEW GUINEA

SIR,—Bottle-feeding of babies is spreading in the urban areas of many developing countries where, because of ignorance and poor living conditions, it often leads to infection and malnutrition.<sup>1</sup> Babies' bottles used to be widely available in shops in Port Moresby, Papua New Guinea, and a survey of children in five typical settlements in December, 1975, and January, 1976, showed that of 127 children under two years of age, 45 (35%) were artificially fed.<sup>2</sup> 52 of the children were below 80% of the Harvard 50th centile of weight for age, and of the underweight children a significantly greater proportion were artificially fed.

The Baby Food Supplies (Control) Act, restricting the sale of baby bottles and teats to registered pharmacists, was passed in 1977. Each sale now has to be authorised by a health worker, who must ensure that the mother is fully taught about bottle-feeding. We repeated the survey in four of the five original areas in March, 1979, to evaluate the effect of this Act.

In the three larger settlements a 1-in-3 sample of houses was obtained using random-number tables, and in the smallest settlement all houses were visited. The survey was done during the day, and the selected households were visited and asked if there were any children under two years of age. Mothers were interviewed using a standard questionnaire, and the child was weighed on a pretested Salter scale. The ages of nearly all children were known accurately to within one month.

5 children were excluded from the 1979 survey results in table 1 because they were no longer receiving breast or arti-

TABLE I—FEEDING METHODS 1975/76 AND 1979

Date	Breast-fed	Artificially fed	Total
1975/76	82 (65%)	45 (35%)	127
1979	127 (88%)	17 (12%)	144

Chi square with Yates' correction 20.02,  $p < 0.0005$ .

TABLE II—WEIGHT FOR AGE IN RELATION TO HARVARD MEDIAN

Date	80% or more	79–60%	59% or less	Total
1975/76	75	38	14	127
1979	103	40	6	149

Chi square 5.94, not significant.

cial milk. Of the 17 children who were artificially fed, 11 used feeding-bottles, and 10 of these bottles had been obtained on prescription the other being obtained illegally. 6 babies were fed by cup and spoon. We found no evidence of bottles other than baby bottles being used for baby feeding.

Although there was a trend for higher weight-for-age in the 1979 survey this trend did not quite reach statistical significance (table II).

We believe that the introduction of a law to make bottle-feeding a little more difficult for those with no medical reason to bottle-feed has resulted in a reversal of a dangerous drift towards bottle-feeding among urban mothers. The contribution of health education by the Health Department and by lay groups must be acknowledged.

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