In Britain, fat consumption is associated with high cholesterol level and increased risk of CHD. Indeed, CHD is the leading cause of death in Britain and the average serum cholesterol concentration of the population is more than the desirable level of 5.2 mmol/litre. Taking the dietary factor alone, it would appear that our subjects would have higher cholesterol levels than the British and one may extrapolate that our population is at higher risk of CHD than the British. However, it is important to interpret these results cautiously for a number of reasons. First, the study group being young and from the same institution may not represent fat consumption of the general population; second, there are factors other than diet which determine the cholesterol level (e.g., genetics) and cholesterol concentration is not the only risk factor for CHD; and third, it is not necessarily true that the consensus about the cholesterol level in Western countries is applicable in Saudi Arabia.

Nonetheless, it is worth mentioning that most of our subjects live with their parents and share the same food and there was no social class bias (A. Al-Sudairy, PhD thesis, unpublished); international studies have shown that there is a continuous association between SFA consumption and CHD; and unless there is a genetic factor there is no reason to believe that our population is different in relation to cholesterol level. Whatever the case, it is clear that a study is needed to determine the average cholesterol concentration in the Saudi population and its relation to CHD.*

Such a study should be useful in many ways: it would provide a frame of reference for the Saudi population in relation to cholesterol level which would enable doctors to justify their intervention; it would raise the awareness of the members of the population about their health; and provide sound evidence on which health service planning would be based. In the meantime, doctors (particularly general practitioners) and other health care team members should educate their patients about the principles of a healthy diet opportunistically and sensitively.


†Correspondence: Dr A. Al-Shehri, Department of General Practice, Liverpool University, The Whelan Building, PO Box 147, Liverpool L69 3BX, UK.

**Table 1**

<table>
<thead>
<tr>
<th>Fat category</th>
<th>Method of calculation</th>
<th>Usual weekly intake</th>
<th>Actual daily intake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (g)</td>
<td>SD</td>
<td>Mean (g)</td>
</tr>
<tr>
<td>Total fat</td>
<td>111</td>
<td>44.9</td>
<td>93.3</td>
</tr>
<tr>
<td>Saturated fat</td>
<td>39.7</td>
<td>21.7</td>
<td>29.8</td>
</tr>
<tr>
<td>Polyunsaturated</td>
<td>5.9</td>
<td>2.3</td>
<td>6.6</td>
</tr>
</tbody>
</table>

**References**


**Age-specific Prevalence of Antibody to Hepatitis C Virus (HCV) Among the Saudi Population**

Sir,

Dr T. M. F. Bakir carried out sero-epidemiological investigations on 2358 Saudi blood to ascertain age-specific prevalence of hepatitis C virus (HCV) in Saudi Arabia, using the first generation enzyme-immunoassay kits at the King Khalid University Hospital and the second generation kits at the Security Forces Hospital in Riyadh. There was no significant difference for prevalence for antibody to HCV among Saudis aged 20 years and above, during screening by the first and second generation kits. That would be rather unexpected and odd since the second generation kits that employ c-33-c, the non-structural protein, and c-22-3, an antigen in virus core are well known for their superiority over kits containing the c-100-c only. Those kits would detect higher HCV carriers in random samples, detect 5–40% more true-positives than the first generation kits in high risk haemophiliacs, and identify up to 10% additional chronic HCV infections. The omission of details of the kit components used at the Security Forces Hospital, would appear to be rather unfortunate, and would be essential to incriminate the reasons for the poor performance of the second generation kits in Saudi Arabia.

The results of detailed studies proposed by Bakir to investigate role of HCV in different categories in Saudi Arabia would receive a boost if antibody screening was to be undertaken by screening urine samples with second-generation detection kits. In a preliminary study on 32 urine/serum necropsy samples from an inner-city medical examiner’s office, urine samples were tested at 1/2 dilution using a modified second-generation ELISA test. There was 100% sensitivity and specificity in the group with a high frequency of HCV infection. The cost saving, ease and safety of sampling and the non-invasive nature of the assay procedure would prove ideal for screening for true incidence of HCV infection even in remote and rural areas in Saudi Arabia.

HCV transmission is now known by routes that were not completely defined earlier. HCV transmission has been documented in a 40-year-old male infected through a tattooing needle which had been passed through boiling water for 2–3 minutes before use. There are two documented cases of HCV transmission in medical staff.

Evidence of marital HCV transmission has been established during genotyping of HCV RNA in six patient–spouse pairs in Japan. The substantial resistance of HCV to drying and heat would also justify investigating the possible HCV transmission during therapeutic scarifications in Saudi Arabia. The practice has been in vogue in rural and urban settings, and the ensuing raw dermal areas on different parts of the body, might have an important role towards HCV transmission in Saudi Arabia.
Serological investigations on 42 histologically confirmed cases of hepatocellular carcinoma (HCC) at the Riyadh Armed Forces Hospital have recently identified 13 patients with antibody to HCV. Prospective serological investigations on Saudi HCC patients with second generation antibody studies could be supplemented by an in situ polymerase chain reaction on tissue sections from HCC patients. A national programme to identify HCV genetic sequences in paraffin-embedded or haematoxylin-eosin stained tissue sections would be ideal.

There should be no technical difficulties in such a programme as both RNA and DNA can be extracted from routinely fixed and stained sections using standard proteinase incubation techniques. Apart from prospective studies, even retrospective investigations would be possible, since there are prior reports of at least 75 HCC cases in the Giza Area of Saudi Arabia. The serological and molecular biological investigations on serum/urine and tissue sections for HCV should not only identify the modes of non-parenteral transmission, but would enable initiation of appropriate preventive/therapeutic measures in afflicted communities.

SUBHASH CHANDRA ARYA MBBS DCP DigiBAC PhD
Clinical Virologist,
Centre for Logistical Research and Innovation,
M-122 (of part 2), Greater Kailash-II,
New Delhi-110048,
India

References


Sirs,

I read with interest the recent article by Dr Bakir (Saud Med J 1992; 13(4): 321-324). While agreeing with the author that non-parenteral route of HCV infection is a major route of transmission, the statement that ‘early acquisition of HCV among Saudis is similar to the pattern of acquisition of HBV infection’ is questionable. In a recent survey involving 413 Saudi schoolchildren aged 5-12 years, and 2639 Saudi male blood donors under 21 years of age, no evidence of exposure to HCV infection could be demonstrated in the children whereas only 0.6% of the blood donors in this young age group were seropositive; the earliest detection of anti-HCV being at 18 years of age (Table I). On the other hand, HbsAg was detected from early age onwards, a clear indication of the differences in transmission patterns between the two virus infections. Similarly, this lack of association between markers of HBV and HCV infection has been noted in another large scale study. Only 0.9% of 4496 Saudi children aged 1-10 years were anti-HCV positive compared to 6.7% HbsAg carrier rate and 19.7% positivity for any HBV marker.

Dr Bakir’s present finding of an anti-HCV prevalence of 2.5-3.5% among Saudi blood donors, and 5.6% (28 of 500 blood donors) in a previous report from the same centre is very high when compared with the prevalence rates of 1.0% (46 of 4580 donations) reported from King Faisal Specialist Hospital, and our own results of 1.1% (107 of 9779 blood

Professor T. Bakir
Head of Virology Unit,
Department of Pathology (32),
King Khalid University Hospital and Faculty of Medicine,
PO Box 2925, Riyadh 11461,
Saudi Arabia
donors) in Riyadh Armed Forces Hospital (manuscript under preparation). Moreover our HCV data on blood donors did not show the wide regional variation ascribed to HBV in the Kingdom so this influence can be ruled out. Obvious reasons for this discrepancy in Dr Bakir's data include the relatively small sample size as well as the use of a mixed population sample of donors and patients. Therefore anti-HCV prevalence among Saudi blood donors must not be exaggerated as it is almost comparable with that reported in the USA and several European countries.

In conclusion although HCV and HBV are thought to share similar modes of transmission, serologic markers of HCV infection appear to be much less frequent than HBV markers in healthy Saudis. Due care should be taken to establish accurate estimates of the prevalence and characteristics of hepatic virus infections in Saudi communities as this is of high public health importance.

**Table 1**

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>No. tested</th>
<th>Anti-HCV (%)</th>
<th>HBsAg (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>School children</td>
<td>5–6</td>
<td>297</td>
<td>0 (0)</td>
<td>4 (1.3)</td>
</tr>
<tr>
<td></td>
<td>10–12</td>
<td>116</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td>413</td>
<td></td>
<td>4 (0.9)</td>
</tr>
<tr>
<td>Blood donors</td>
<td>17</td>
<td>109</td>
<td>0 (0)</td>
<td>7 (6.4)</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>362</td>
<td>1 (0.3)</td>
<td>12 (3.3)</td>
</tr>
<tr>
<td></td>
<td>19</td>
<td>553</td>
<td>1 (0.2)</td>
<td>12 (2.2)</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>818</td>
<td>5 (0.6)</td>
<td>31 (3.8)</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>797</td>
<td>8 (1.0)</td>
<td>21 (2.6)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td>2639</td>
<td>15 (0.6)</td>
<td>83 (3.1)</td>
</tr>
</tbody>
</table>

I would like to thank Dr Saeed for his interest in our paper. The discrepancy in the prevalence of antibody to hepatitis C virus (anti-HCV) among Saudi blood donors as reported by us (2.5–5.5%)1 and those reported by Dr Saeed (1.1%) (above) and Bernvill et al. (1%)2 is probably due to the fact that our anti-HCV-positive samples were not confirmed by RIBA or any other confirmatory test as similar conclusions were found by other investigators at different parts of the world.2-4 Confirmatory tests are currently being done on all anti-HCV-positive samples and a lower prevalence of anti-HCV-positivity is actually true. As to the influence of regional variations in the prevalence of anti-HCV among Saudis our preliminary data and those of others from Saudi Arabia7 show that such variations exist. As for the modes of transmission of HCV among Saudis, these are currently being investigated and we agree with Dr Saeed and others7 that there is a lack of association between markers of HBV and HCV infection at an early age.

**References**


Sir,

I would like to thank Dr Saeed for his interest in our paper.

The discrepancy in the prevalence of antibody to hepatitis C virus (anti-HCV) among Saudi blood donors as reported by us (2.5–5.5%) and those reported by Dr Saeed (1.1%) (above) and Bernvill et al. (1%) is probably due to the fact that our anti-HCV-positive samples were not confirmed by RIBA or any other confirmatory test as similar conclusions were found by other investigators at different parts of the world. Confirmatory tests are currently being done on all anti-HCV-positive samples and a lower prevalence of anti-HCV-positivity is actually true. As to the influence of regional variations in the prevalence of anti-HCV among Saudis our preliminary data and those of others from Saudi Arabia show that such variations exist. As for the modes of transmission of HCV among Saudis, these are currently being investigated and we agree with Dr Saeed and others that there is a lack of association between markers of HBV and HCV infection at an early age.

**References**