the current understanding of norovirus-host interaction. In addition, several points should perhaps be reconsidered with respect to data analysis and interpretation of the results.

The ability of noroviral virus-like particles to recognize human HBGAs is highly strain-specific [2, 3], and this fact is the basis of current exploration of norovirus host range. Different HBGA binding patterns have been found in both GI and GII noroviruses [4]. Recent data even suggest variations in HBGA recognition within the GII-4 genotype [5]. Thus, it is critical for an outbreak study to have a single norovirus strain involved in each outbreak. Unfortunately, this does not appear to be the case for the study described in the Halperin et al. article [1]. The 2003 outbreak was caused by at least 2 strains that phylogenetically clustered with 2002B and 2003A of GII-4 [1], respectively. This could result in a misleading outcome if the 2 strains have different HBGA binding patterns. Similarly, data for 2 outbreaks that occurred in 2 different years and were caused by genetically distinct strains should not be pooled for analysis.

Other concerns about this study include the following. First, the low reverse transcriptase polymerase chain reaction detection rates of norovirus in the 2 outbreaks (33% and 28%, respectively) [1] raises the possibility that additional causes, such as other noroviruses and/or non-norovirus pathogens, were involved, which could further dilute the results. Second, it is known that in addition to the ABO family, the Lewis and secretor families are also involved in norovirus-host interaction. The Halperin et al. study [1] focused only on the ABO family, and another reason for the negative results could be that the important information of the secretor family was missed, as shown by several previous studies [6–9]. Third, GII is composed of at least 17 genotypes and many of them have not yet been studied in terms of virus-host interaction. Therefore, the conclusion that there is no association between HBGA and susceptibility to clinical infection with GII norovirus—based on only 2 GII-4 outbreaks—is overstated. Fourth, it would be informative if the HBGA binding patterns of the causative strains involved in the outbreaks could be determined, which would address the key question about the strain-specific host range.

Our understanding of the interaction between the polymorphic human HBGAs and the broadly diverse human noroviruses remains preliminary. Recent studies also suggested that host factor(s) may be strongly associated with the evolution and epidemiology of GII-4 noroviruses, in which both host immunity and HBGAs could play an important role [5]. Because human noroviruses remain difficult to cultivate and an effective animal model is still lacking, population studies that use epidemiological approaches remain an important tool for norovirus research.

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References


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Intital Descriptive and Analytical Data on an Outbreak of Norovirus Infection at Marine Corps Recruit Depot Parris Island, South Carolina

To the Editor—We read with interest the brief report by Halperin et al. in which the authors found no association between histo-blood group antigens (HBGAs) and susceptibility to clinical genogroup II
norovirus (NoV) infection [1]. We report similar results for an outbreak of NoV infection that occurred in a United States military training facility. In 2007, an outbreak of genogroup II NoV infection was confirmed by reverse-transcriptase polymerase chain reaction (PCR) of stool samples from clinically infected recruits at the Marine Corps Recruit Depot (MCRD) Parris Island, South Carolina. A case-control study to evaluate the risk factors for symptomatic infection was approved by the institutional review board at the Naval Medical Center, Portsmouth, Virginia. Case patients were defined as those who reported gastrointestinal illness in a questionnaire. ABO blood type was abstracted from electronic medical records. Clinical characteristics of infection were collected from the electronic medical records and the questionnaire.

One hundred eighteen recruits completed risk-factor questionnaires. Four subjects were excluded because of incomplete answers. The percentages of blood types A, B, AB, and O in the study population were 45%, 6%, 3%, and 46%, respectively. Of the 114 case patients, 77% reported diarrhea, 27% reported fever, 27% reported vomiting, and 69% reported nausea and vomiting. The modes for the length of illness and the number of training days missed were 2 days and 1 day, respectively. Eighty-three percent of case patients missed an average of one day of training. When patients in the A, B, and AB blood groups were compared with patients in the O blood group, there was no association between HBGA and susceptibility to clinical infection with genogroup II NoV (table 1).

We provide data that further support the assertion of Halperin et al. [1] that that genogroup II NoV is capable of infecting persons regardless of their ABO blood type. Furthermore, we highlight the fact that outbreaks occur frequently and are significantly costly to the military, with regard to both decreased productivity and military readiness.

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Reference

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Table 1. Association between histo–blood group antigen (HBGA) and susceptibility to symptomatic genogroup II norovirus infection.

<table>
<thead>
<tr>
<th>HBGA type</th>
<th>Case patients, no. (%)</th>
<th>Control patients, no. (%)</th>
<th>OR (95 % CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>25 (47)</td>
<td>26 (43)</td>
<td>1.16 (0.53–2.51)</td>
</tr>
<tr>
<td>AB</td>
<td>1 (2)</td>
<td>2 (3)</td>
<td>0.91 (0.18–4.45)</td>
</tr>
<tr>
<td>B</td>
<td>3 (6)</td>
<td>4 (6)</td>
<td>0.60 (0.06–7.08)</td>
</tr>
<tr>
<td>O</td>
<td>24 (45)</td>
<td>29 (48)</td>
<td>1.00 (reference)</td>
</tr>
</tbody>
</table>

NOTE. CI, confidence interval; OR, odds ratio.