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humans by using the DNase
distinct, novel parvoviruses in
recent publications have identified 2
members of the family
Parvoviridae
Parvoviruses are
small, nonenveloped DNA viruses
that infect both vertebrate and inverte-
brate hosts. Until recently, parvovirus
B19 and adeno-associated viruses,
which belong to the genera
Erythrovirus and Dependovirus,
respectively, were the only known
members of the family Parvoviridae
that infected humans (1). However, 2
recent publications have identified 2
distinct, novel parvoviruses in
humans by using the DNase
sequence–independent single-primer
amplification technique and a related
method (2,3). The first of these virus-
es, termed PARV4, was observed in
a patient with symptoms of acute viral
infection syndrome after high-risk
behavior for infection with HIV-1, although the patient was subsequently
confirmed as negative for HIV-1 (2).
The second parvovirus was identified
in respiratory samples from children
with lower respiratory tract infections
and termed human bocavirus (3).
Parvovirus B19 is a frequent con-
taminant of plasma pools that are used
in the manufacture of blood products,
which results in high viral loads in
pools and viral transmission in recipi-
ents of clotting factors (4). We identi-
fied PARV4 in such pools (5), albeit at
a lower frequency and titer than par-
vovirus B19, when parvovirus B19 was
not excluded by screening with
nucleic acid amplification techniques.
Sequence analysis identified a second
genotype of PARV4, which we have
termed PARV5, that shares
92% nucleotide identity with PARV4 (5).
PARV4 was originally identified
in a plasma sample from a homeless,
injection drug user with fatigue, night
sweats, pharyngitis, neck stiffness,
vomiting, diarrhea, arthralgia, and
confusion (2). This person was coin-
fected with hepatitis B virus. In this
study, we looked retrospectively for
PARV4 and PARV5 in blood samples
from a similar cohort of persons,
many of whom were known to be
infected with hepatitis C virus (HCV)
as determined by the presence of
both HCV RNA and antibodies to
HCV), and some of whom were intra-
venous drug users (IVDUs) (6).
Blood samples were collected
from 26 cadavers in London and the
surrounding area as part of a study to
investigate the inhibition of nucleic
acid amplification techniques for
bloodborne viruses in tissue samples
(6). The cohort was composed of 10
HCV RNA–positive IVDUs, 8 HCV
RNA–positive non-IVDUs, 4 HCV
RNA–negative IVDUs, and 4 HCV
RNA–negative non-IVDUs (Table). Nucleic acid was extracted as previ-
ously described (4) by using the
MagNA Pure LC instrument (Roche,
Basel, Switzerland). PCR was per-
formed with primers specific for the
second open reading frame (ORF2) in
the PARV4 genome (2), which is
homologous to the VP1 capsid of par-
vovirus B19. Primers PVORF2F (5’-
AGGAGCAGAAAACACTCA-
GAC-3’) and PVORF2R (5’TCTCTT-
CATAGCGGTGTCAACTAA-3’) amplify a 268-bp region of ORF2
(nucleotides 2710–2977, GenBank
accession no. AY622943). The PCRs
were performed and analyzed as pre-
viously described (5). The assay is
highly specific (no cross-reactivity
with parvovirus B19) and sensitive
detects 5–10 copies of PARV4 virus
DNA per reaction).
PCR products were cloned,
sequenced, and compared with the
prototype PARV4. Two blood samples
were positive for PARV4, and a third
sample was positive for PARV5, with
99%–100% nucleotide identity. These
positive samples were from HCV
RNA–positive IVDUs (Table). The
titer of PARV4 and PARV5 DNA in
the positive samples was low and did
not exceed >700 copies/mL of plas-
am, as determined by using a consen-
sus TaqMan assay (J. Fryer, unpub.
data). None of the other blood sam-
ple tested was positive for PARV4 and
PARV5, including those for per-
sons who were HCV RNA negative
and not IVDUs.
In our previous study (5) of >130
fractionation pools (composed of
thousands of units from screened
healthy donors) for PARV4, the only
positive pools were from North
America and no European pools were
positive for PARV4 or PARV5. These
viruses may be present in such pools
but diluted to undetectable levels. In

Table 1. Analysis of 26 cadavers for parvoviruses PARV4 and PARV5

<table>
<thead>
<tr>
<th>Group</th>
<th>PARV4 and PARV5 in HCV RNA–positive cadavers</th>
<th>PARV4 and PARV5 in HCV RNA–negative cadavers</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVDUs</td>
<td>3/10</td>
<td>0/4</td>
</tr>
<tr>
<td>Non-IVDUs</td>
<td>0/8</td>
<td>0/4</td>
</tr>
</tbody>
</table>

*HCV, hepatitis C virus; IVDUs, intravenous drug users.
the present study, PARV4 and PARV5 have been identified in blood samples obtained from persons from the United Kingdom. For parvovirus B19, there is evidence of persistent virus infection, at low levels, in bone marrow of previously exposed persons (7) and in plasma of immunocompromised and immunocompetent persons (8,9). There is also evidence for the lifelong persistence of parvovirus B19 (genotypes 1 and 2) in tissues such as skin and synovia (10). PARV4 and PARV5 virus genomes share only limited homology with parvovirus B19 (<30% amino acid similarity). Although they have been detected in blood and plasma, nothing is known about the role of these viruses in human disease or their ability to persist in infected persons, healthy or otherwise. Further studies will be required to determine the prevalence of PARV4 and PARV5 in healthy persons compared with its prevalence in those with chronic infections and at high risk, such as IVDUs, and to investigate the nature of persistence of these novel viruses.

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References


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Saint Louis Encephalitis Virus, Brazil

To the Editor: Saint Louis encephalitis virus (SLEV), a member of the Flaviviridae family, is widely dispersed in the Americas (1,2). In Brazil, SLEV was first isolated in the 1960s from a pool of mosquitoes at the Amazon Basin. Subsequently, the virus was repeatedly isolated from animals and arthropods in the Amazon region and São Paulo state (3). Nonetheless, isolation of SLEV from humans is rare; only 2 isolates from humans were described before 2005. Each isolate was from a patient who had jaundice and febrile illness without any neurologic symptoms (1,3). Recently in São Paulo, SLEV was isolated from a patient who had an incorrect diagnosis of dengue fever (2,4).

Despite the rare isolation of SLEV from humans, antibodies to this virus have been found in ~5% of studied populations in the north and southeast regions of Brazil. However, because of antibody cross-reactivity among different flaviviruses and the fact that this population is vaccinated against yellow fever and exposed to dengue virus (DENV), such results should be interpreted carefully. Nevertheless, in these areas, SLEV may circulate and infect humans, although most infections are undiagnosed (1,3,5).

In contrast to previous instances in which the disease was detected in only 1 patient, we describe the first community outbreak of SLEV in Brazil. The outbreak was detected in São José do Rio Preto (population 400,000), in northwest São Paulo state. This outbreak was concurrent with a large outbreak of DENV serotype 3 (DENV-3), which occurred during the first half of 2006, with >15,000 possible cases reported to public health authorities. During this time, we were involved in an epidemiologic study to monitor the disease. We tested ~250 samples for DENV, and 65% were positive. We tested for SLEV only those patients who were in our hospital or those who were referred to us for SLEV testing after an initial diagnosis of SLEV or DENV. The protocol approved by our ethical committee allowed us to test only samples from these patients (process no. 300/2004).

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