SEOV strains, 6 of which were from *R. norvegicus* rodents captured in urban areas of North Vietnam. Phylogenetic analysis showed that this SEOV belonged to the Vietnamese SEOV genotype (Figure).

We describe a clinical case of hantavirus infection and its potential rodent reservoir occurring in Vietnam. The clinical manifestations of the case-patient were compatible with SEOV infection, which is responsible for a moderate form of HFRS (10). Also, HFRS caused by SEOV occurs in urban rather than rural areas, unlike other hantavirus infections. Our epidemiologic findings were compatible with other studies indicating the source of infection was the case-patient’s home, the only place where she had a history of exposure to rodents. Although viral RNA could not be obtained from the case-patient for genotyping, the genomic comparison of the viral strains from rodents captured in the case-patient’s home and elsewhere in Vietnam suggested that the source of infection was local rodents. This report provides additional evidence that hantavirus infection is a worldwide problem and is likely underdiagnosed in Vietnam and other countries where simple standardized laboratory diagnostics are not widely available.

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**Origin of Highly Pathogenic Porcine Reproductive and Respiratory Syndrome Virus, China**

To the Editor: A highly pathogenic porcine reproductive and respiratory syndrome virus (HP-PRRSV), which affected >2 million pigs, emerged in early 2006 in the People’s Republic of China. The disease was characterized by high fever (41°C), high illness rates (50%–100%), and high death rates (20%–100%) for pigs of all ages (7). A number of HP-PRRSVs have been isolated from 2006 through 2009 from infected pigs in different provinces of China and confirmed to be the causative agent of the new outbreaks (1,2). These HP-PRRSVs have a deletion of 30 amino acids in nonstructural protein 2 (NSP-2). However, the evolutionary origin and path of the HP-PRRSV remain unknown.

We analyzed the full-length sequences of 67 PRRSVs: 35 HP-PRRSVs (HuN4 and LNSY-08-1 isolated in our laboratory and 33 viruses isolated in other laboratories), 28 classic PRRSVs (18 viruses isolated from China and 10 viruses representing other Asian countries and North
accumulation from subgroup 2 to sub-
tuations might be explained as a stepwise
for HP-PRRSVs. Deletions among subgroup 4 viruses is

Whole genome–based phylogenetic analysis showed that these
67 PRRSVs could be divided into 4
subgroups (online Appendix Figure, www.cdc.gov/EID/content/16/2/365-
appF.htm). Ten classic PRRSVs from
China, together with the North American
prototype virus VR-2332 and
modified live vaccine, were classified
into subgroup 1. The first Chinese
isolate, CH-1a, and its 3 derivatives
(CH2002, CH2003, and CH2004) were
classified into subgroup 2. All
35 HP-PRRSVs were classified
into subgroup 4, and they shared high
homology (>99%) in their genom-
ic sequences. The other 4 Chinese
PRRSVs, including HB-1(sh)/2002,
HB-2(sh)/2002, Em2007, and SHB,
belonged to subgroup 3, an interme-
diate subgroup between subgroups 2
and 4. Phylogenetically, HP-PRRSVs
had a close relationship with sub-
groups 2 and 3.
Four conserved deletions were
shown among all HP-PRRSVs, includ-
ing an adenine deletion at position 122
in the 5′-untranslated region, a guano-
sine deletion at position 15,278 in the 3′-
untranslated region, and 2 continu-
dous deletions in the NSP-2, including
a single amino acid deletion at position
482 (L482) and a second deletion of 29
amino acids between positions 533 and
561 (S533–A561). The presence of these 4
deletions among subgroup 4 viruses is
a unique phenomenon, which may be
used as a distinctive molecular marker
for HP-PRRSVs.
The occurrence of these 4 dele-
tions might be explained as a stepwise
accumulation from subgroup 2 to sub-
group 4. None of the 4 deletions were
found in subgroup 2. Among viruses
in subgroup 3, one, 2, or 3 of the 4
deletions occurred. For example, a
single deletion was present at 122 nt
in Em2007, double deletions at 122 nt
and 15,278 nt in HB-1(sh)/2002 and
SHB, and triple deletions at 122 nt,
15,278 nt, and 482 aa in GD3-2005
(this sequence was not submitted to
GenBank until now). In 2008, Ma et
al. compared GD3-2005 with several
PRRSVs and reported the homology
within them, pointing out that the 2
deletions in NSP-2 were identical to
the HP-PRRSV (5). After careful anal-
ysis, we found the GD3-2005 more
interesting than what was reported by
Ma et al.; it belongs to an intermediate
group, and shares the characteristics
of gradual evolution. Eventually, all 4
deletions occurred in subgroup 4. This
obvious pattern suggests that these
4 conserved deletions might have
evolved step by step.
The primary neutralizing epitope
(PNE), which is located on glyco-
protein 5 and composed of the resi-
dues S37H(F/L)QLIYN with F/L39 as
the binding site for the neutralizing
antibody (6,7), also displayed simi-
lar changes at the 39 position among
the 4 subgroups. The PNE residues
in subgroups 1 (SHELLQLIYN) and
2 (SFHELQLIYN) were considerably
conservative. Subgroup 3 contained
F39 or I39 (F39 in Em2007 and
SHB, and I39 in both HB-1-
(sh)/2002 and SHB); subgroup 4 con-
tained I39 only. The existence of either
F39 or I39 in subgroup 3 PNE indicates
its intermediate position between sub-
groups 2 and 4 in the evolution of HP-
PRRSVs. Pairwise comparison of subgroups
2, 3, and 4 did not find recombina-
tion or large fragment replacement,
which suggests that all HP-PRRSVs
originated from the same ancestor by
gradual evolution. Notably, the re-
cently isolated intermediate PRRSVs
mentioned above (SHB, Em2007,
and GD3-2005) were isolated in the
region of South China where the out-
break of HP-PRRS initially occurred.
Furthermore, the epidemiologic data
show that the outbreak of HP-PRRSV
emerged from 1 particular place and
then spread widely. This evidence in-
dicates that all HP-PRRSVs isolated
in China likely originated from the
same source.
In summary, our findings sug-
jest that the newly emerged HP-
PRRSVs originated from the Chinese
CH-1a-like PRRSV. Further study is
needed to determine what contributes
to the increased pathogenicity of HP-
PRRSV. Although the 4 deletions are
conserved in all HP-PRRVs, the in-
creased pathogenicity of HP-PRRSV
may not merely be caused by the de-
letions; pathogenicity is affected by
multigenic factors.

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Evidence-based Tool for Triggering School Closures during Influenza Outbreaks

To the Editor: I read with interest the recent article by Sasaki et al., “Evidence-based Tool for Triggering School Closures during Influenza Outbreaks, Japan” (1), which describes an algorithm for determining the optimal timing of school closures to control influenza outbreaks. The published information is a helpful guide for predicting influenza outbreaks in school settings. However, no data are presented to show the efficacy of school closures after the detection of such outbreaks. As such, the title “Evidence-based Tool for Predicting Influenza Outbreaks, Japan” would more accurately describe the article.

The findings presented by Sasaki et al. (1) could be used to help make a decision for school closure or dismissal in places like Japan, but no information is provided on whether this approach is effective in preventing further influenza virus transmission. This is an important distinction and should not change the current school response guidance published by the Centers for Disease Control and Prevention (CDC) (2). In general, CDC guidance suggests that during an influenza outbreak, policymakers should weigh the advantages and disadvantages of school dismissals or school closures before making a decision.

In Response: Vogt (1) correctly points out that our article (2) did not present data on the effectiveness of school closures to control influenza outbreaks. However, public health agencies continue to support school closure as a nonpharmaceutical response to the ongoing outbreak of pandemic (H1N1) 2009 (3) despite little evidence for the appropriate timing of closures, even though it is known that timely action is critical. As the title of our article reflects, our algorithm was designed as an evidence-based tool for supporting the timing of school closures.

In our article, we pointed out that evaluating the impact of school closures is a critical research question. Before April 2009, decision-making regarding school closure in Japan was left to individual schools, 98% of which are public. Since then, recommendations for public school closure have been made according to standardized rules set by the Japanese School Health and Safety Law, leaving final decision-making authority up to local education boards. Our next study will evaluate the effectiveness of this early, standardized timing of school closure in Japan.

On September 24, 2009, the Japanese Ministry of Health, Labor and Welfare presented a school closure plan for use in the different stages of an influenza outbreak; the plan is based on World Health Organization strategies.