Pandemic Influenza School Closure Policies

To the Editor: Holmberg et al. (1) are rightly concerned that state pandemic plans in the United States represent a patchwork without central coordination or direction. These concerns are particularly relevant for school closure decisions during an influenza pandemic. The US Department of Health and Human Services’ checklist regarding school closures gives conflicting messages (2). For example, it recommends that schools stay open during a pandemic and develop school-based surveillance systems for absenteeism of students and sick-leave policies for staff and students. It also recommends developing alternate procedures to ensure the continuity of instruction in the event of district-wide school closures. These vague recommendations may reflect the paucity of data to recommend school closure.

To assess the current status of school closure decisions in the United States, I conducted an internet survey of all 50 state health commissioners during the spring of 2006. I asked the respondents 2 questions: “Who makes the school closure decisions in your state?” and “What absenteeism rate, if any, would prompt school closures during an influenza pandemic?” Of the 44 responding states, I found that school closure decisions were primarily a local-level responsibility in half. Of these 22 states, closure decisions would be made either on a school-by-school or a school district-by-school district basis. Only 6 states indicated that school closure decisions would be made at the state level, and 16 states would have decisions made jointly at the state and local levels (Table).

For a typical influenza season, only 6 states indicated that they close schools if a certain absenteeism rate due to illness were reached. For 5 of these states, the absenteeism rates ranged from 10% to 30%; the sixth state said its schools would close if the rates were anywhere from 7% to 31%. However, only 1 state reported a threshold absenteeism rate for closure during an influenza pandemic. Another state said that it was developing an absenteeism rate that would prompt closure for pandemic influenza. Forty-two states did not have threshold absenteeism rates that would prompt school closures during an influenza pandemic.

In July 2006, the Department for Education and Skills in the United Kingdom published guidelines regarding school closure (D. O’Gorman, pers. comm.). Although the final decision for school closure would lie with local school officials, the national government might advise all schools and childcare facilities to close when a pandemic reached their area to reduce the spread of infection among children (3). It is believed that all would comply with closure advice and that use of emergency powers under the UK Civil Contingency Act 2004 to require services to close would not be necessary. If all British schools in an area were advised to close during a pandemic, the situation would be reviewed after a period of time, such as 2 to 3 weeks, by local officials acting on information from the UK government, to decide whether to remain closed.

Although the United States is a nation dedicated to federalism, an uncoordinated approach for community response measures such as school closure decisions could jeopardize our efforts in containing a deadly pandemic. If schools were to remain open until a certain percentage of students and faculty became ill, as they do during typical influenza seasons, then control measures to contain the outbreaks would likely be far more difficult to achieve because a chain of transmission would be established. Some might consider it unethical for schools to stay open in the face of a pandemic with a high death rate. I therefore think a national policy, or at least specific national guidelines, should be developed jointly by the Centers for Disease Control and Prevention and the Department of Education, so that states’ school districts can develop rational, coherent, and coordinated closure plans to protect children and communities during an influenza pandemic.

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References

Table: Number of states reporting influenza pandemic school closure policies at various levels, USA*

<table>
<thead>
<tr>
<th>Region</th>
<th>Local only</th>
<th>State and local</th>
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</tr>
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<tr>
<td>South</td>
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<td>8</td>
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</tr>
<tr>
<td>Midwest</td>
<td>7</td>
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</tr>
<tr>
<td>West</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Total†</td>
<td>22</td>
<td>16</td>
<td>6</td>
</tr>
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*Northeast: CT, DC, MA, ME, NH, NJ, NY, PA, RI, VT; South: AL, AR, DE, FL, GA, KY, LA, MD, MS, NC, OK, SC, TN, TX, VA, WV; Midwest: IA, IL, IN, KS, MI, MN, MO, ND, NE, OH, SD, WI; West: AK, AZ, CA, CO, HI, ID, MT, NM, NV, OR, UT, WA, WY.
†Six states did not respond.
Symptomatic Human Hantavirus in the Americas

To the Editor: In a recent letter (1), dos Santos et al. described 3 cases of hantavirus pulmonary syndrome (HPS) from Juquitiba and stated that “the first human cases of symptomatic infection by hantaviruses were reported from Brazil in 1993.” However, we described 8 cases of symptomatic hemorrhagic fever with renal syndrome (HFRS) in Recife, Brazil, 5 months before the initial May 1993 report of Sin Nombre virus (SNV)–induced HPS in the United States (2). Our report was therefore the first published account of symptomatic hantavirus infections, not just in Brazil but anywhere in the Americas (3).

Serum samples from our Brazilian HFRS cases, collected in 1990, were screened by an immunofluorescence assay (IFA) and ELISA for immunoglobulin G as were the current Brazilian HPS cases (1). Two of our patients had an increased immunoglobulin M titer by ELISA (2). Rat-transmitted Seoul virus (SEOV) was considered most likely because this was the only hantavirus strain showing clear positive results in IFA (2,3). All the Recife cases in 1990 had reported likely rat contacts and were initially diagnosed as leptospirosis—clinical hallmarks of both HFRS and leptospirosis (3). We also subsequently found evidence of SEOV infection in 31 (15%) of 201 leptospirosis-suspected acute renal failure cases from Belém, Brazil, confirmed in 1 case with highly specific neutralization tests (4). Moreover, as we predicted (3), some of the 133 (41%) of 326 urban cases of acute renal failure from Salvador, Brazil, which appeared nonconfirmed for leptospirosis (5), were later shown to be caused by SEOV (unpub. data). Finally, of 379 schoolchildren from Salvador at high risk for frequent rat exposure, 13.2% were IFA positive for the Korean prototype Hantaan virus (HTNV) but none for the American SNV (6). Because both HTNV and its rodent reservoir are absent from the American biotope, HTNV seroreactivity should be considered a cross-reaction to another related murine antigen; that is to say, the ratborne SEOV.

Wild rats (Rattus rattus and R. norvegicus) are the only Old World rodents ubiquitous in the New World and thus a potential source of SEOV infection in the Americas (3,7). Moreover, the first hantavirus characterized in South America was SEOV, isolated as long ago as 1984 from a rat caught in Belém (7). Furthermore, the first 3 clinical cases of hantavirus infection in the United States were SEOV-induced (Baltimore rat virus) HFRS cases and not HPS (8).

The clinical syndromes of HFRS and HPS can appear identical, with pulmonary edema, shock, and renal insufficiency with marked proteinuria and thrombocytopenia (9). Moreover, worldwide ELISA testing with a single antigen such as SNV or Puumala virus (PUUV) can result in misleading cross-reactions, since both viruses are genetically related. Although this SEOV-PUUV cross-reactivity enabled the first recognition of HPS cases in the New World 14 years ago, this may now lead to the wrong clinical diagnosis and reinforces the need for specific tests such as neutralization tests or reverse transcription–PCR. Although not as lethal and probably not so frequent as HPS, SEOV-induced HFRS may still be greatly underestimated in the Americas, or misdiagnosed as leptospirosis.

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References