Impact of Epidemic Influenza A–Like Acute Respiratory Illness in a Remote Jungle Highland Population in Irian Jaya, Indonesia


From the U.S. Naval Medical Research Unit No. 2 and the Ministry of Health (National Institute of Health Research and Development and Directorate General of Communicable Diseases Control and Environmental Health), Jakarta, Indonesia; Provincial Health Service (Jayapura) and Regency Health Service (Wamena), Irian Jaya, Indonesia; Naval Medical Research Institute, Bethesda, Maryland, USA; and Centers for Disease Control and Prevention, Atlanta, Georgia, USA

A suspected epidemic of unknown etiology was investigated in April/May 1996 in the remote jungle highlands of easternmost Indonesia. Trend analysis demonstrates the area-wide occurrence of a major respiratory infection outbreak in November 1995 through February 1996. The monthly mean rate of respiratory infection episodes for the peak outbreak months (2,477 episodes/100,000 persons) was significantly higher ($P < .0001$) than for the 34 months leading up to the outbreak (109 episodes/100,000 persons). Notable were the high attack rates, particularly among adults: 202 episodes/1,000 persons aged 20–50 years in one community. Excess morbidity attributed to the outbreak was an estimated 4,338 episodes. The overall case-fatality rate was 15.1% of outbreak cases. Laboratory evidence confirmed the circulation of influenza A/Taiwan/1/86–like viruses in the study population, and high hemagglutination inhibition titer responses were indicative of recent infections. Historical documents from neighboring Papua New Guinea highlight the role of influenza A virus in repeated area outbreaks.

Outbreaks of influenza-like illness are reported from temperate and tropical areas worldwide. Influenza outbreaks generally have the most severe impact on immunologically naive and/or immunocompromised populations, principally the very young, elderly, and infirm. In fact, most accounts of epidemic influenza A and B transmission reported from Western countries involve schools, day-care centers, and nursing homes. Epidemic influenza spread, however, also occurs in adult occupational-type environments that include workplace, correctional, and military settings [1–5]. In more developing regions like India (Delhi), attack rates associated with influenza A outbreaks are often similar among nominally healthy young adults and young children [6, 7].

The spread of the influenza virus is principally via direct exposure to airborne droplets, although the virus may remain infectious in dried mucus for many hours. Notable is the rapidity with which epidemic influenza, particularly influenza A, can overwhelm an affected population; attack rates generally range from <15% to 25% in large communities and ≥40% in closed populations. Consequently, the outbreak potential of influenza A, relative to the proportion of the population exposed and area involved, can be significant [1, 8]. Recognition of epidemic influenza is usually based on the epidemiological picture, in the absence of or in conjunction with supportive laboratory evidence [1]. Excess mortality of diagnosed pneumonia and/or influenza is the predominant surveillance measure used in monitoring the severity of epidemic influenza. In addition, trend information pertaining to hospitalizations and age-specific attack rates of severe respiratory disease can provide alternative indices in early influenza outbreak detection [1, 9].

This report describes an investigation of a possible respiratory infection outbreak involving suspected influenza A viruses, the aim of which was threefold: (1) to determine if disease occurrence was of epidemic proportion; (2) to identify the cause; and (3) to highlight the event from an epidemiological perspective.

The Outbreak

First reported by local (Jakarta) newspapers in February 1996, an outbreak of “wabah babi” (translated as “pig epidemic” from the Indonesian language), involving >300 human deaths, was described from Irian Jaya, Indonesia [10]. Early accounts suggested the outbreak was also temporally associated with an excess of pig deaths reported from the same area. Signs and symptoms were initially purported to be febrile/hemorrhagic in nature [11].

The remoteness of the outbreak area precluded a timely or complete assessment of epidemic conditions: persons, place, time, and probable cause. In response to reports of a large outbreak, national, provincial, and local health authorities, in cooperation with the U.S. Naval Medical Research Unit No. 2 (NAMRU-2), organized an investigation.
Anecdotal information obtained from mission nursing staff in Kabupaten Jayawijaya, via two-way radio communications, yielded clues that proved invaluable to the outbreak investigation. An epidemic was indeed evident, starting in early November 1995 and lasting through February 1996. Significant mortality was reported in association with a large number of respiratory infection cases observed throughout the region. Mission nursing staff described an immediate and dramatic response to antibiotic and antipyretic therapy, suggestive of a contributing bacterial etiology, particularly in severe cases.

Methods

Area/subjects. The outbreak area of Kabupaten Jayawijaya is located in Irian Jaya, the easternmost regency of Indonesia, bordering with Papua New Guinea (figure 1). This highland region, covering >47,000 km², is characterized by rugged, mountainous terrain that supports a lushly forested landscape.

Figure 1. A map of the area affected by the outbreak of influenza A–like acute respiratory illness (*landing-strip elevations, provided by Mission Aviation Fellowship; ‡capital city of subdistricts; †communities).
Table 1. Hierarchy of outbreak investigation and data collection strategies.

<table>
<thead>
<tr>
<th>Data</th>
<th>Reporting source/mechanism</th>
<th>Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory illness</td>
<td>Government health center (Puskesmas) records</td>
<td>Regency of Kabupaten Jayawijaya</td>
</tr>
<tr>
<td></td>
<td>Village headmen, tribal records</td>
<td>Regency of Kabupaten Jayawijaya</td>
</tr>
<tr>
<td></td>
<td>Mission health centers</td>
<td>Cohort of five villages</td>
</tr>
<tr>
<td>Serology + respiratory illness history</td>
<td>Case-control field study</td>
<td>Kurima (including Ninia and Soba)</td>
</tr>
<tr>
<td>Deaths</td>
<td>Close family member</td>
<td>Wamena and Soba</td>
</tr>
<tr>
<td>Climate and environmental conditions</td>
<td>Government and NAMRU Staff</td>
<td>Regency of Kabupaten Jayawijaya</td>
</tr>
</tbody>
</table>

NOTE. NAMRU = U.S. Naval Medical Research Unit.

was obtained from the records of Puskesmas (government health centers) throughout Kabupaten Jayawijaya. This information provides the only standardized collection of health statistics that can be translated for trend analysis. Data pertaining to age, sex, and clinical diagnoses (based on signs/symptoms) are nominally recorded for all clinic visits by government health centers and are summarized annually for reporting purposes.

Unfortunately, there are significant concerns about the reliability of such data in the absence of standardized case definitions, and there are no organized surveillance efforts targeting influenza (or respiratory infections in general). In addition, the validity of routinely collected data is poor, given that government and/or mission health-related services reach only a small proportion of the principally rural population of Irian Jaya. Reports of deaths (attributed to the outbreak) from area village headmen were obtained for review; five village headmen provided area-representative information of outbreak-attributed deaths, by age, in their respective communities.

Verbal input by mission nursing staff at mission health centers in the regency led investigators to focus on the villages of Ninia and Soba (figure 1) for field data and specimen-collection efforts. Significant disease occurrence and area accessibility served as criteria in the selection of these two communities for study purposes. Health information obtained from records maintained by the mission health centers in Ninia and Soba, along with accurate demographic data pertaining to the local population, allowed estimation of area-specific attack rates.

Case-control study. A case-control field study was carried out in two areas of Kabupaten Jayawijaya: the Wamena-Kelila area, accessible by vehicle and foot from the regency capital of Wamena, and Kurima (includes the communities of Ninia and Soba; figure 1). Families (all members) were randomly selected and served as the principal sampling unit.

Cases were differentiated from controls for comparative analytical examination on the basis of a respiratory disease experience during the suspected outbreak period of November 1995 through February 1996. After informed voluntary consent was obtained, a standardized interview of each participating family member was conducted by trained health workers in the appropriate (local) dialect(s). In addition, information on absent family members was obtained, including detailed accounts of outbreak-related deaths.

A 10-mL blood sample was collected from each study participant with use of a Vacutainer (Becton-Dickinson, Franklin Lakes, NJ) collection system. Clotted specimens were centrifuged and sera were transferred in cryovials (Nunc Inc., Illinois) and stored in portable liquid nitrogen containers at −196°C.

Serological analysis. Hemagglutination inhibition (HI) tests were carried out at the Centers for Disease Control and Prevention. Single serum samples collected from outbreak cases, controls, and historical controls within the population were tested. Three of the antigens used were representative of the three predominant groups of influenza viruses circulating worldwide at the time, i.e., A/Taiwan/1/86 (H1N1), A/Johannesburg/33/94 (H3N2), and B/Harbin/7/94 ET (ether-treated). In addition, several viral antigens representing influenza A subtypes that have circulated previously in humans or have so far circulated only in birds were tested, including A/Japan/305/57 (H2N2), A/swine/Hong Kong/273/94 (classic swine H1N1), A/swine/Hong Kong/168/93 (avian-like H1N1 from pigs), A/tern/South Africa/61 (H5N3), and A/Turkey/Oregon/71 (H7N3). Standard methods for HI testing were used, including treatment of serum specimens with receptor-destroying enzyme (Denka, Tokyo, Japan) [12]. HI titers are expressed as the reciprocal of the highest dilution of human serum that completely inhibited hemagglutination; for study purposes, titers ≥1:80 were considered to be evidence of recent infection.

Results

Retrospective Data Review

Puskesmas (government health center) data. Area-specific trend data for Kabupaten Jayawijaya are shown in figures 2–4. Evident was the geographic reach of the epidemic, involving the entire regency. Notable also was the rise in the rate of
Puskesmas records and census data) suggest a rise in attack rates with increasing elevation; the attack rate was 9,190 episodes/100,000 persons at elevations >6,000 feet above-sea-level (ASL), compared with 5,351 and 4,376 episodes/100,000 persons at elevations of 5,000–6,000 feet ASL and <5,000 feet ASL, respectively (figure 1).

There were 23,533 respiratory infection episodes documented during the outbreak period in a population of 421,169 (this figure is cumulative for all four outbreak months), for a monthly mean of 2,942. The excess morbidity attributed to the actual outbreak was estimated to be 4,338 episodes, as determined by adding the number of episodes reported from November 1993 through February 1994 (20,285) to the number from November 1994 through February 1995 (18,105) and then subtracting the mean value for these two reporting periods (19,195) from the number of episodes recognized from November 1995 through February 1996 (23,533).

**Village headmen data.** Outbreak (respiratory infection)–related deaths (from November 1995 through February 1996), as described by a sampling of village headmen from the affected area, indicate young adults (aged 15–44 years) accounted for a large proportion (28%) of the 287 reported deaths. The proportional distribution of outbreak fatalities for persons in the >45, 5–14, 1–4, and <1 year age categories was 34%, 7%, 17%, and 15%, respectively (table 2). Young adults (aged 15–44 years) accounted for the largest percentage of outbreak-related deaths in the Kecamatan (districts) of Okbibab/Borne (76% of 54 deaths) and Kelila/Wolo (54% of 13 deaths) (figure 1).

**Mission health center (Ninia and Soba) data.** Data recorded from the mission health centers in Ninia and Soba demonstrate the dramatic rise in clinically recognized episodes of respiratory illness during the outbreak period (figures 5, 6). In Soba, this was evident for reported cases of both URI and pneumonia; it was not possible to distinguish between the diagnostic classifications of URI and pneumonia from the records
maintained in Ninia. The monthly mean rates of respiratory illness were significantly lower in Soba ($P < .0001$) and Ninia ($P < .001$) before the outbreak than during the actual (4-month) outbreak period of November 1995 through February 1996: 5,796 vs. 22,782 episodes/100,000 persons (95% CI, 9,530–24,444/100,000) and 1,124 vs. 3,027 episodes/100,000 persons (95% CI, 937–2,879/100,000).

Age-specific attack rates in Ninia and Soba (table 3) were particularly high (and second only to those among young children, <5 years of age) in the age groups of 20–50 years ($P < .01$) and 15–44 years (202 episodes/1,000), respectively.

**Case-Control Field Study**

**Epidemiology.** One hundred twenty-two persons (95 cases and 27 controls) were interviewed in the case-control field study. An attack rate (for ≥1 respiratory illness episode during the outbreak period) of 779/1,000 persons was estimated. There was no significant difference ($P > .05$) in mean ages between cases (28 years, ranging from 1 to 60) and controls (33 years, ranging from 13 to 70). The male:female ratio was 1:1:1.

Analysis of the outbreak included 280 (92 case and 188 control) subjects: 158 absent and/or deceased (outbreak-related) family members, in addition to the 122 volunteer subjects interviewed, representing 71 families (or 4.0 persons/family). Overall, the estimated attack rate was 332/1,000 persons. An extrapolation of data reflects the epidemic situation in the entire regency, translating into 139,888 outbreak-related cases, or 33% of the overall population of Kabupaten Jayawijaya (421,169 persons).

The attack rate in Kurima (444/1,000 persons), which includes the communities of Ninia and Soba, was significantly ($P < .0001$) higher than in the Wamena-Kelila area (137/1,000 persons). The overall mean age of 91 cases (28 years) was significantly ($P < .01$) higher than for 175 controls (22 years) ($95\% \text{ CI}, 2.3–9.9$). This was true both in Wamena-Kelila ($P < .05$; 95% CI, 1.7–17.6) and in Kurima ($P < .01$; 95% CI, 2.9–12.2). There was little difference in the overall sex ratio between cases (1:1) and controls (1:1.3), in both the Wamena-Kelila and Kurima areas.

Outbreak data suggest significant familial impact. Overall, 66% of households had ≥1 cases ($95\% \text{ CI}, 0.5–0.8$). There was a significant difference ($P < .01$) in the proportion of affected households between the Wamena-Kelila (38%) and Kurima (89%) study areas. In addition, familial case clustering was evident in Kurima, where the mean number of cases per household (2.4) was significantly higher ($P < .01$) than that observed in Wamena-Kelila (1 case per household). In Kurima, 54% (20) of 37 households had ≥1 family case and 32% (12) had >2 family cases. Also notable was the high percentage of households in Kurima with ≥1 death (38%; 95% CI, 0.2–0.5).

**Clinical findings.** The most frequently described signs/symptoms recalled from the time of the outbreak by the 122 study volunteers interviewed clearly differentiated cases (95) from controls (27): 89% of cases vs. 11% of controls had headache, 87% vs. 7% had fever, 84% vs. 22% had cough, and 54% vs. 4% had shortness of breath. Clinical manifestations nominally associated with hemorrhagic conditions were negligible: bloody nose, bloody mouth, bloody urine, and bloody stool.

**Death data.** Retrospective information about deaths associated with the outbreak included 14 family-reported fatalities. The estimated case-fatality rate was 15.1% of outbreak cases. These data, applied to the whole of Kabupaten Jayawijaya, suggest that 20,983 deaths (15.1% of 139,888 outbreak cases, or 5.1% of the 421,169 population) may have been epidemic-related. However, this extrapolation assumes uniform epidemic
Table 2. Proportional distribution (by age group) of deaths associated with the outbreak of respiratory influenza A–like illness occurring in November 1995 through February 1996.

<table>
<thead>
<tr>
<th>Age group (y)</th>
<th>No. (%) of deaths* in indicated area²</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>17 (14)</td>
</tr>
<tr>
<td>1–4</td>
<td>5 (4)</td>
</tr>
<tr>
<td>5–14</td>
<td>1 (1)</td>
</tr>
<tr>
<td>15–44</td>
<td>25 (20)</td>
</tr>
<tr>
<td>&gt;45</td>
<td>75 (61)</td>
</tr>
<tr>
<td>Total</td>
<td>123 (100)</td>
</tr>
</tbody>
</table>

* As reported by village headmen.
² Kec. = Kecamatan (districts).

conditions throughout the regency and probably represents the high range of outbreak deaths; the actual number of case fatalities may have been measurably lower.

In addition, rates increased as did area-specific elevation: 13.9% of outbreak cases in the higher-elevated study area of Kurima vs. 1.1% of outbreak cases in Wamena-Kelila were fatal.

A relatively short interval (mean, 4.7 days; 95% CI, 3.32–6.08) was measured from onset of signs/symptoms until death. There was no significant difference (P > .05) between the mean ages of case fatalities (28 years; 95% CI, 15.2–40.3) and case survivors (29 years; 95% CI, 24.9–32.1). In addition, the proportion of fatalities among male cases (17%) did not differ significantly from that among females (13%).

Laboratory Findings

Serological test results showed negligible overall geometric mean titers of antibody (GMTs) to A/Taiwan/1/86, A/Johannesburg/33/94, and B/Harbin/07/94 ET influenza strains: 1:13.2, 1:16.2, and 1:8.8, respectively (table 4). However, the GMT for A/Taiwan/1/86 (based on titers >1:20) was comparatively higher (1:95.5) than those for A/Johannesburg/33/94 (1:61.2) and B/Harbin/07/94 ET (1:55.8) (P < .001). An important finding was that 34% of 180 samples had a GMT of >1:20 for A/Taiwan/1/86: 40% of the 78 from Wamena-Kelila and 16% of the 102 from Kurima (P < .05). No significant difference, by area, was found when titers (>1:20) of 1:5,120 were excluded from the calculation; five specimens with titers of 1:5,120 for A/Taiwan/1/86 all came from Wamena-Kelila. There was no significant difference in GMTs for influenza strain A/Taiwan/1/86 between study cases and controls.

For comparative purposes, 21 specimens collected from Okibil (figure 1) in 1990–1991 served as historical controls. Virus HI titers of 1:5,120 against A/Taiwan/1/86 (suggestive of acute disease) were detected in two sera. However, none of the 19 remaining samples registered titers >1:20.

Environmental Background

Evident from trend data in figures 2–4 is the annual cyclic rise of respiratory episodes, generally beginning in January and...
ending in March. Also evident is the lack of reported episodes (on the basis of Puskesmas data) during the month of December 1994 through 1996. This anomaly may reflect a reporting bias associated with the Christmas and New Year holiday period.

There was negligible variance in monthly temperatures over the 3 years of recorded measures from Kabupaten Jayawijaya. No weather data were available for February 1996, but the mean monthly temperature during the other outbreak months (19.6°C; range, 19.5–19.8°C) differed little from that during the preceding 31 months (19.1°C; range, 17.8–19.8°C). Rainfall, however, was relatively heavy during the 4 outbreak months; a monthly mean of 233 mm (range, 153–325 mm) of rain fell during the outbreak months, compared with 154 mm (range, 41–317 mm) during the previous 34 months ($P < .05$; 95% CI, 8.8–148).

Discussion

Data extracted from puskemas records highlight the occurrence of epidemic respiratory disease in Kabupaten Jayawijaya that affected a significant proportion of the area population and extended throughout the entire regency. The large number of outbreak-related respiratory illness episodes (4,338), expressed as a measure of excess morbidity, attests to the epidemic finding. This information provides the only regency-wide, systematically collected data for trend analysis. The community impact of this outbreak is further substantiated by information obtained from mission health centers in Ninia and Soba and findings from the case-control field study. Data extrapolated from the latter reflect the sizeable proportion (one-third) of outbreak-related respiratory illness cases among the population of Kabupaten Jayawijaya.

Historical evidence of epidemic influenza A and B transmission has been repeatedly documented in neighboring Papua New Guinea (figure 1). Influenza A was suspected in a large number of outbreak-related deaths in 1932; mortality estimates ranged from <1% in the coastal region to as high as 10% of the population in the remote highlands [13]. No fatalities were reported in the large western expatriate communities in the same areas.

Table 3. Area/age-specific rates of URI/pneumonia in Indonesia, November 1995–February 1996.

<table>
<thead>
<tr>
<th>Age group (y)</th>
<th>Ninia</th>
<th></th>
<th></th>
<th>Soba</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of persons</td>
<td>No. of cases/episodes</td>
<td>Attack rate (per/1,000)</td>
<td>No. of persons*</td>
<td>No. of cases/episodes</td>
<td>Attack rate (per/1,000)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–4</td>
<td>660</td>
<td>182</td>
<td>276</td>
<td>558</td>
<td>1,119</td>
<td>307</td>
<td></td>
</tr>
<tr>
<td>5–12</td>
<td>787</td>
<td>99</td>
<td>126</td>
<td>941</td>
<td>605</td>
<td>166</td>
<td></td>
</tr>
<tr>
<td>13–19</td>
<td>810</td>
<td>29</td>
<td>36</td>
<td>1,819</td>
<td>735</td>
<td>202</td>
<td></td>
</tr>
<tr>
<td>20–50</td>
<td>1,802</td>
<td>278</td>
<td>154</td>
<td>&gt;45</td>
<td>322</td>
<td>576</td>
<td></td>
</tr>
<tr>
<td>&gt;51</td>
<td>188</td>
<td>15</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE. Rates are based on disease episodes. Data are from mission health centers. URI = upper respiratory infection. * Extrapolated from 1995 census estimate (proportional distribution by age).
In 1962, >3,000 deaths in the highlands of Papua New Guinea were attributed to the A2/Hong Kong/68 strain of influenza A. Most notable was the rise in mortality rates with increasing elevation [13–15]. These findings are compatible with those described in this report: in the case-control field study in Kabupaten Jayawijaya, a high case-fatality rate (15.1% of outbreak cases) was demonstrated, and both puskesmas data and the case-control field study showed that these rates increase along with area-specific elevation.

Contributing to the theory that elevation may contribute to the death burden associated with influenza outbreaks is the probability that access to medical care is greater at lower altitudes, where there are more medical facilities, better transportation conditions, and higher temperatures [16]. The relationship between elevation and case morbidity/fatality, however, may be a function of nutritional status. Negligible consumption of vegetables and fruits (and their contained vitamins) characterizes the diet of the inhabitants of Kabupaten Jayawijaya, particularly at higher elevations. In addition, animal protein is noticeably lacking from the population’s diet, as evident from the scarcity of wild game and general absence of animal domestication, except for swine populations and limited attempts at rabbit breeding.

Pigs are found throughout the regency and in proximity with people, often sharing the same living spaces. These prized animals, however, are treated more as pets and generally are eaten only on special occasions, e.g., weddings. The principal (and often only) food staple of the more remote communities occupying the higher-elevated regions is sweet potato. Consequently, the added risk of respiratory-illness-related morbidity/mortality at higher altitudes may be predicated on poor nutritional status, as expressed by a limited diet (in the absence of nutritional indices). Although risk factors that lead to increased influenza-related morbidity and mortality, particularly in children and the elderly, have not been clearly identified, alterations in host immunity brought on by malnutrition may significantly contribute to severity of illness and poor outcome [16, 17].

A seemingly unique feature of this epidemic was the relatively high rates of morbidity (expressed as attack rates from mission health center data) and mortality (reported by village headmen) among the young adult population. These findings differ from what is generally described in influenza outbreaks; most accounts are from care centers for the very young and old [1–5]. This phenomenon, however, was documented in influenza outbreaks in the bordering (with Kabupaten Jayawijaya) highland region of Papua New Guinea, where adults were stricken in significant proportion [18]. In addition, the age-specific mortality curve depicting the “great pandemic” influenza A outbreak of 1918 in the United States was W-shaped as opposed to the normally observed U-type curve [1, 19, 20].

Notable among the death data was the short interval from onset of signs/symptoms until death. In addition, mission nursing staff described the dramatic clinical response in severe outbreak cases to antibiotic and antipyretic therapy. This information suggests a complicating bacterial component contributing to case fatalities. During the 1918 influenza pandemic, typical bacterial pneumonia was implicated for the rapidity with which death followed initial clinical presentation. Most deaths occurred among survivors of the first few days who went on to develop secondary bacterial infections, suspected as due to Haemophilus influenzae or a group A streptococcus, and died with typical bacterial pneumonia [1, 19].

The population of Kabupaten Jayawijaya can be characterized as “marginalized” from an immunologic perspective: first, naive with regard to exposure to western influences (including disease), which are usually a product of close social/commercial contacts, and second, compromised by the burden of background infections like malaria and/or poor nutritional status. In fact, most young children observed during the investigation had recognizable kwashiorkor. The effect of their non-immune status may have contributed to the heavy epidemic toll among the young adult population.

Laboratory diagnostic evidence did not confirm influenza A virus as the causative etiology in this epidemic event. In addi-

Table 4. Evidence of circulating influenza virus strain(s) in the study population, Wamena-Kelila and Kurima areas, April–May 1996.

<table>
<thead>
<tr>
<th>Virus strain, study area (n*)</th>
<th>No. (%) of persons with indicated titer of antibody to strain</th>
<th>Geometric mean titer of specimens (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≤20</td>
<td>40</td>
</tr>
<tr>
<td>A/Taiwan/1/86</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wamena-Kelila (78)</td>
<td>57 (73)</td>
<td>11 (14)</td>
</tr>
<tr>
<td>Kurima (102)</td>
<td>89 (87)</td>
<td>10 (10)</td>
</tr>
<tr>
<td>A/Johannesburg/33/94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wamena-Kelila (79)</td>
<td>67 (85)</td>
<td>6 (7)</td>
</tr>
<tr>
<td>Kurima (102)</td>
<td>52 (51)</td>
<td>30 (29)</td>
</tr>
<tr>
<td>B/ Harbin/07/94 ET</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wamena-Kelila (79)</td>
<td>73 (92)</td>
<td>4 (5)</td>
</tr>
<tr>
<td>Kurima (102)</td>
<td>83 (81)</td>
<td>12 (12)</td>
</tr>
</tbody>
</table>

* Includes cases and controls.
tion, HI titer responses suggestive of acute influenza strain A/Taiwan/1/86 were detected in historical control sera. Study findings do show, however, that A/Taiwan/1/86 was still circulating in the community at the time of this study. The epidemic effect of influenza A, as described in this report, is the same as previously detailed in Papua New Guinea [13–15, 18].

The presence of large pig populations occupying the same domicile (generally crowded and poorly ventilated) with humans suggests the potential for animal-to-person transmission. The occurrence of a large number of swine deaths was anecdotally reported from the regency many months before the start of the outbreak. No data regarding pig illness and/or associated mortality were collected before or during the actual outbreak.

There was no clinical evidence of pig illness during the investigation; clinical observations in veterinary examinations were made from among a sampling of two herds. Serological evidence of swine influenza virus was demonstrated during a 1976 outbreak among military recruits at Fort Dix, New Jersey [21]. Recently reported laboratory exposures (despite Animal Biosafety Level 3 containment practices) document that the transmission of swine H1N1 viruses to humans occurs directly and readily [22].

The conceptual approach to the study of emerging/reemerging disease should be expanded to include marginalized populations. Epidemic occurrence among susceptible persons like those living in the remote jungle highlands of Irian Jaya is different from the norm predicated on the Western model. Surveillance efforts targeting marginalized groups provide the best opportunity for recognizing and containing explosive epidemic events involving emerging/reemerging and endemic disease conditions. This investigation documents just such an outbreak of probable influenza A virus infection in a marginalized population.

References