

Address for correspondence: Yong Poovorawan, Center of Excellence in Viral Hepatitis Research, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand; email: yong.p@chula.ac.th

Laboratory Exposure to Influenza A H2N2, Germany, 2004–2005

To the Editor: From November 2004 to February 2005, a company contracted by the College of American Pathologists (CAP) sent influenza quality assurance samples containing live influenza A H2N2 viruses (A/Japan/305/57) to 3,748 international laboratories (1,2). Of these, 3,686 (98%) were located in Canada or the United States. In Germany, 6 laboratories received at least 1 of 3 samples, 2 for virus antigen detection and 1 for virus culture; all contained live virus and were formatted identically. No information on infectivity or virulence of the samples was available. Because of the absence of human-to-human influenza A H2N2 virus transmission since 1968, this situation provided the rare opportunity to investigate whether infections with this strain had occurred in any of the laboratories.

We used a standardized questionnaire to obtain from the laboratories information on when the CAP samples had been received, which of the 3 quality assurance specimens they contained, and how many employees had been involved in their handling. A second questionnaire was distributed to personnel in microbiology and virology departments. This elicited information regarding routine laboratory activities, contact with CAP sam-

ples, tasks performed in conjunction with handling of the samples, and any influenzalike symptoms (sudden onset of fever, cough, headache, and muscle pain) within the respective time frame. Persons who had worked in a receiving laboratory from September 1, 2004, to April 15, 2005, and had performed routine procedures in virology (defined as transport of samples, preparation of samples for diagnostic testing, antigen testing, nucleic acid amplification testing, and virus isolation) were eligible for the study. From May 4 to May 19, 2005, we visited the laboratories to interview supervisory personnel regarding routine work-up of samples and to collect blood from study participants for serologic testing.

The national reference laboratory for influenza at Robert Koch Institute performed serologic testing for antibodies against A/Singapore/1/57 (H2N2) virus by hemagglutination inhibition. We compared antibody titers of laboratory workers who worked with a CAP sample with those who did not. However, this comparison might not show a difference if (silent) virus transmission had occurred among laboratory staff. To exclude this possibility, we also compared titers of workers born before 1969 with those in a group of volunteers from Robert Koch Institute also born before 1969. Titers <10 were assigned a value of 1.

Of 47 laboratory workers, 18 either declined to participate or were excluded because they did not perform any of the defined routine procedures during the defined period. Thus, 29 (62%) workers were included in the study, of whom 14 (48%) reported having worked with CAP samples. Of these 14 workers, 12 (2 exclusively) transported samples and 11 (2 exclusively) prepared the samples; 9 (1 exclusively) performed antigen testing, 2 (0) performed nucleic acid amplification tests and 4 (0) performed virus isolation. Fourteen

workers (48%) reported no contact with the samples, and 1 (3%) was unsure. None of the 29 participants reported any event that could have led to release of infectious material during the respective time frame, such as broken test tubes or dropped culture plates. Participating laboratories reported that all procedures were performed under appropriate hygienic and safety precautions. No person had ≥ 3 symptoms typical for influenzalike illness in the 4 days after having worked with a CAP sample.

Specific influenza A H2N2 antibody titers were determined in 25 study participants. None had a titer >80, two (8%) had a titer of 80, three (12%) had titers of 40, two (8%) had titers of 20, and the remaining 18 (72%) had titers ≤ 10 . Three (21%) of 14 workers exposed to CAP samples and 4 (40%) of 10 who denied exposure had titers ≥ 20 . All 7 were born before 1969. The geometric mean of titers of all participating workers born before 1969 did not differ significantly from that of the Robert Koch Institute employees ($p = 0.28$; Figure).

In summary, no evidence was found for laboratory infections with the influenza A H2N2 virus. The risk for laboratory-acquired influenza infections is unknown. Severe acute respiratory syndrome coronavirus and *Mycobacterium tuberculosis* are infectious agents whose transmission characteristics are similar to those of influenza. For severe acute respiratory syndrome coronavirus, laboratory-acquired infections are well documented (3,4). For *M. tuberculosis*, there are strong indications that they occur (5–7). From a public health perspective and in view of the current importance given to influenza and a possible pandemic, accurate characterization of the risk for influenza infections in laboratory settings is needed. The small number of persons included in this study limits the conclusions that can be drawn. Potentially exposed workers were

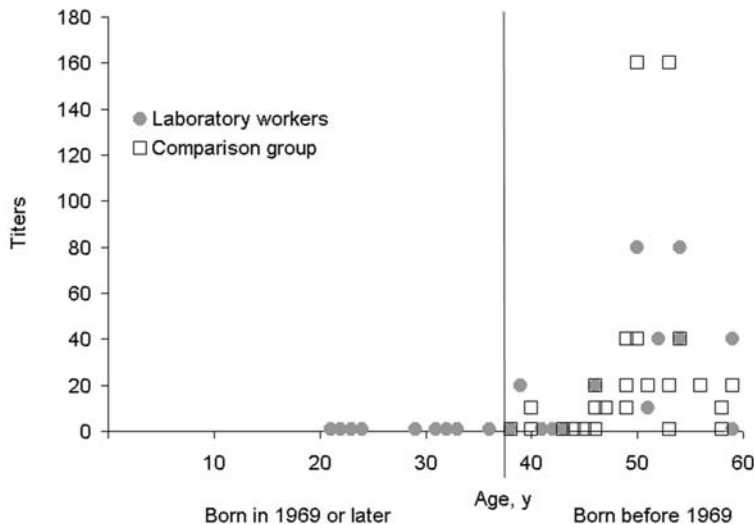


Figure. Titers of antibodies to influenza A H2N2 virus in laboratory personnel (n = 25; 13 born before 1969) and a comparison group born before 1969 (n = 32). The age listed is that in 2005. Titers <10 were assigned a value of 1.

presumably tested in other laboratories involved, but we are not aware of any publications to this end. The lack of evidence for laboratory-acquired infection with A H2N2 in our study suggests that the risk was low under controlled laboratory conditions. However, only a large-scale serologic study (which might still feasibly be undertaken) could further substantiate this finding.

Acknowledgments

We thank all assisting public health professionals and study participants for their support.

Annette Schrauder,*

Brunhilde Schweiger,*

Udo Buchholz,* Walter Haas,*

Daniel Sagebiel,*

Adrienne Guignard,*

and Wiebke Hellenbrand*

*Robert Koch Institute, Berlin, Germany

References

1. Eurosurveillance editorial team, Meijer A, Brown C, Paget J. Worldwide laboratory distribution of influenza A/H2N2 virus similar to 1957–58 pandemic strain, labs asked to destroy all samples immediately. *Eurosurveillance*. 2005;10. [cited 2006 Sep

15]. Available from <http://www.eurosurveillance.org/ew/2005/050414.asp>

2. World Health Organization. International response to the distribution of a H2N2 influenza virus for laboratory testing: risk considered low for laboratory workers and the public. 2005 [cited 2006 May 22]. Available from www.who.int/csr/disease/influenza/h2n2_2005_04_12/en/.
3. Normile D. Mounting lab accidents raise SARS fears. *Science*. 2004;304:659–61.
4. Lim PL, Kurup A, Gopalakrishna G, Chan KP, Wong CW, Ng LC, et al. Laboratory-acquired severe respiratory syndrome. *N Engl J Med*. 2004;350:1740–5.
5. Seidler A, Nienhaus A. Epidemiologie der berufsbedingten tuberkulose: ein literatur-review zu konventionellen studien. In: Nienhaus A, Brandenburg S, Teschler H, editors. *Tuberkulose als berufskrankheit: ein leitfaden zur begutachtung*. Landsberg (Germany): Ecomed Verlag; 2003. p. 142–69.
6. Jacobson JT, Orlob RB, Clayton JL. Infections acquired in clinical laboratories in Utah. *J Clin Microbiol*. 1985;21:486–9.
7. Kao AS, Ashford DA, McNeil MM, Warren NG, Good RC. Descriptive profile of tuberculin skin testing programs and laboratory-acquired tuberculosis infections in public health laboratories. *J Clin Microbiol*. 1997;35:1847–51.

Address for correspondence: Udo Buchholz, Department for Infectious Disease Epidemiology, Robert Koch Institute, Seestrasse 10, 13353 Berlin, Germany; email: buchholz@rki.de

Methicillin-resistant *Staphylococcus aureus* in Cat and Owner

To the Editor: A 3-year-old, neutered male, domestic shorthaired cat was referred for treatment to a veterinary specialty clinic in San Francisco, California, with a 1-year history of multifocal patches of crusted and well-demarcated ulcers on the trunk. Initially, small crusts suspected to be associated with flea allergy and pyoderma were present; however, response was poor to multiple treatments, including repeated corticosteroid therapy and antimicrobial therapy with amoxicillin–clavulanic acid and enrofloxacin. The owner reported having skin abscesses and pneumonia 3 months earlier, although no microbiologic testing was performed.

Cytologic evaluation of exudate from the cat's lesions identified neutrophils and eosinophils with engulfed cocci. Leukocytosis with eosinophilia was found on a complete blood cell count. No notable abnormalities were present on thoracic radiograph, abdominal ultrasonograph, urinalysis, and tests for feline leukemia and immunodeficiency virus. Skin biopsy specimens were collected for histologic examination, and swabs of the exudates were submitted for bacterial culture. Histopathologic findings demonstrated ulcers and dermal granulation tissue with linearly arranged eosinophils, mast cells, neutrophils, and plasma cells between dense, homogeneous collagen bundles (sclerosing dermatitis). This pattern of inflammation is distinct from most staphylococcal infections of the skin, and it has been suggested that this uncommon histologic finding in cats is associated with methicillin-resistant staphylococcal infection (1).

Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated