Food Environ Virol (2010) 2:41–46
DOI 10.1007/s12560-010-9026-5

ORIGINAL PAPER

The Prevalence of Human Parainfluenza Virus 1 on Indoor Office Fomites

Stephanie A. Boone · Charles P. Gerba

Received: 28 November 2009 / Accepted: 24 January 2010 / Published online: 9 February 2010

Abstract The objective of this study was to evaluate the potential role of fomites in human parainfluenza virus 1 (HPIV1) transmission by assessing the occurrence of HPIV1 on surfaces in an adult setting (office). In 2004, a total of 328 fomites from 12 different office buildings in five different cities were evaluated for HPIV1 viral RNA. HPIV1 was isolated using reverse transcriptase–polymerase chain reaction (RT–PCR) and detected on 37% of all office fomites. HPIV1 RNA was frequently isolated on desk tops (47%), and infrequently isolated on light switches (19%). Data revealed a statistically significant difference between the percentage of HPIV1 positive fomites in office cubicles and conference rooms (Chi-square $P < 0.011$, Fisher's Exact $P = 0.054$). A statistically significant difference was also found among positive fomites in different buildings (Chi-square $P < 0.011$). HPIV1 was consistently isolated on various indoor fomites in the 12 office buildings assessed during 2004, a low HPIV incident year.

Keywords Parainfluenza 1 virus · Respiratory virus · Fomites · Offices · RT–PCR

C. P. Gerba (✉)
Department of Soil Water and Environmental Science, University of Arizona, Tucson, AZ 85721, USA
e-mail: gerba@ag.arizona.edu

Present Address:
S. A. Boone
Southern Regional Research Center, 1100 Robert E. Lee Blvd, New Orleans, LA 70124, USA

Introduction

Acute respiratory tract infection is the most common illness in humans (Monto 2002). Human parainfluenza viruses (HPIV) are medically important respiratory pathogens second only to respiratory syncytial virus in causing major lower respiratory tract disease in infants and young children (Glezen and Floyd 1997; Echevarria et al. 1998, Hendrickson 2003; CDC 2009). There are four types of parainfluenza with type 1 being the most common cause of infection in humans and typically causes outbreaks of respiratory disease in the fall (Glezen and Floyd 1997). The Center for Disease Control (CDC) reports that human parainfluenza virus 1 (HPIV1) is the leading cause of croup in the United States (Hendrickson 2003; CDC 2009). HPIV are considered community acquired pathogens without ethnic, gender, age, or geographic boundaries (Hendrickson 2003). Immunity to HPIV is incomplete and viral infection occurs throughout life (Hendrickson 2003; CDC 2009). Thus far, HPIV infections are commonly associated with pediatric illness, and less is known about adult infections (Hendrickson 2003).

Adult respiratory tract infections are caused by several different viruses that often have clinically indistinguishable symptoms. Due to a lack of laboratory diagnosis, the HPIV adult morbidity data available is limited (Glezen and Floyd 1997). As a result, the true prevalence and incidence of HPIV-related respiratory disease in adults is unknown. The majority of studies investigating adult HPIV infections and illness have concentrated on elderly or immunocompromised populations (Falsey et al. 1995, Falsey 1991; Perz et al. 2001). In a 10-year study conducted by Vilchez et al. (2002) examining immunocompromised adult lung transplant recipients, HPIV was cited as the leading cause of lower respiratory tract illness in patients with a median age

of 48. HPIV is also suspected to play a role in bacterial pneumonia and the death of elderly nursing home residents (Fiore et al. 1998). To date, very few studies have focused on parainfluenza viral illness in healthy adults. In 1999 a respiratory tract illness outbreak among an international tourist group was diagnosed as HPIV1 after evaluation of nasopharyngeal and throat swabs. During the outbreak attack rates ranged from 100% in adults 65 years and older, to 45% in adults less than 65 years of age (Perz et al. 2001). A study conducted at the San Francisco University Medical Center during the 2002 influenza season found HPIV1 in healthy adults with respiratory infections after using polymerase chain reaction (PCR) for diagnosis (Louie et al. 2005).

Rational infection control of any virus requires a clear understanding of pathogen survival and environmental transmission (Goldman 2000). HPIV1 disease prevalence has been documented at consistently low levels throughout most months of the year with the highest incidence occurring during October of odds years (Alguilar et al. 2000; Ansari et al. 1991; Henrickson et al. 1994; Monto 2004). HPIV environmental survival and infectivity is influenced by temperature, humidity and pH. HPIV environmental stability is greatest at 4°C and at physiological pH (7.4–8.0) with viral survival decreasing significantly at 37°C and low humidity (Ansari et al. 1991; Donaldson and Ferris, 1976). When HPIV is held at room temperature on nonporous surfaces its survival may vary from 2 h to 1 week depending environmental conditions (Hendrickson 2003). Average survival of HPIV on porous surfaces is about 4 h (Aitken and Jefferies 2001; Ansari et al. 1991; Barker et al. 2001; and Brady et al. 1990).

Studies investigating HPIV transmission indicate small particle aerosol spread is unlikely as documented by the aerosol studies of Mclean et al. (1976) where only 2 of 40 children were infected with HPIV1 when exposed at a distance of 60 cm. In 1991, Ansari et al. demonstrated that parainfluenza virus could be transferred from stainless steel surfaces to clean fingers, and that HPIV3 remained viable on hands longer than 1 h. The Ansari and Mclean studies suggest that fomites may play a role in the spread of HPIV. CDC publications also suggest that HPIV can be spread through contact with contaminated respiratory secretions from an infected individual or a contaminated surface (CDC 2009). Pathogenic virus contamination of fomites has been documented in the indoor environments of hospitals (coronavirus, RSV, influenza A, parainfluenza, norovirus), day care facilities (influenza A, rotavirus, coronavirus, astrovirus), restaurants, and bars (adenovirus, HAV) (Barker et al. 2001; Boone and Gerba 2005; Goldman 2000; Hota 2004, Ward et al. 1991). Presently there are only limited studies investigating HPIV transmission (Ansari et al. 1991; Barker et al. 2001). Additionally, there appear to be no studies documenting the prevalence of HPIV or other viruses on surfaces in office settings. The aim of this study was to evaluate the prevalence of HPIV1 on indoor surfaces in an adult setting (office) during an HPIV1 low incident year to better assess the potential role of fomites in its transmission.

Methods

Sample Plan

Selected fomites were sampled in office buildings located in Atlanta, GA, Chicago, IL, San Francisco, CA, New York, NY, and Tucson, AZ. A total of 328 samples from 12 different office buildings were evaluated over a 6-week period from September to October 2004. Work areas were sampled once during regular work hours; however, the occupant was asked to leave while the fomites were swabbed. Surfaces sampled included 76 telephone receivers, 62 computer mouse, 51 office and cubical desktops, 26 conference room table tops, 50 chair arms, 42 door knobs or door handles, 21 light switches, totaling 328 samples. For flat surfaces (desktops, table tops, arm chairs) approximately 100 cm^2 was sampled. For the other objects the entire surface was sampled. Several pens, pencils, and audiovisual remote controls were also tested but not included in data analysis due to the small sample number. The samples were obtained by swabbing each individual surface with a sterile polyester fiber-tipped transport system collection swab moistened in transport medium (BBL culture swabs, Becton, Dickinson and Company, Sparks, MD). One person in each city was trained and appointed to complete all surface swabbing for sample collection standardization and consistency.

RNA Extraction and Reverse Transcription

Environmental Protection Agency (EPA) Quality Assurance Quality Control guidance for laboratories performing PCR analysis (USEPA 2004) was used throughout both RNA extraction and the RT–PCR procedures to prevent sample and product contamination. RNA extraction and RT–PCR procedures were performed in separate rooms and in separate PCR hoods. Samples and reagents were also kept in separate rooms and separate freezers. Parainfluenza 1 human paramyxovirus was obtained from American Type Culture Collection (ATCC, Manassas, VA) and used as the HPIV1 positive control throughout the procedure. QIAmp Viral RNA Mini kits and procedure from Qiagen Inc. (Valencia, CA) were used as recommended by the manufacturer to extract and concentrate viral RNA from fomite and control samples. An initial volume of 420 μL was used

in the RNA extraction process to produce a final volume of 80 μL.

All reagents used for reverse transcriptase and polymerase chain reaction were obtained from Applied Biosystems (Roche Molecular Systems Inc. Branchburg, NJ). The reverse transcriptase reaction mixture contained 10 μl of sample RNA extract, 3.5 μl of 25 mM $MgCl_2$ solution, 1.5 μl of Amplitaq GeneAmp 10× PCR buffer, 4.0 μl of 2.5 mM of dNTP mix, 0.5 μl of 50 mM Random Hexamers, 0.5 μl of 20 U/μl RNase Inhibitor, and 0.5 μl of 50 U/μl MuLV Reverse Transcriptase to yield reverse transcriptase mixture totaling 20.5 μl per sample. The reaction mixture was then placed in an Applied Biosystems Gene Amp PCR System 9700 (Roche Molecular Systems Inc. Branchburg, NJ) thermocycler for 10 min at 24°C, 60 min at 44°C, 5 min at 99°C, and 5 min at 5°C.

PCR and Product Detection

The primers used were based on a surface protein gene segment that is highly conserved among the subtypes of parainfluenza 1 virus (Fan and Hendrickson 1996). The viral primers amplify a 179 base pair product with the following nucleotide sequences: 5′ATT TCT GGA GAT GTC CCG TAG GAG AAC-3′ (upstream), and 5′-CAC ATC CTT GAG TGA TTA AGT TTG ATG A-3′ (downstream) as shown in Fan and Hendrickson (1996). PCR was performed using 18.2 μl of Rnase free water (Promega Madison, WI), 3.0 μl of 25 mM $MgCl_2$ solution, 4.0 μl of Amplitaq with GeneAmp 10× PCR buffer, 3.0 μl of 2.5 mM of dNTP mix, 0.4 μl of 200 μM (upstream and downstream) primers, and 0.50 μl of 5 U/μl Amplitaq DNA Polymerase per sample. All sample cDNA products (20.5 μl) from the reverse transcriptase reaction were added to the PCR master mix, which resulted in a final mixture volume of 50 μl. The PCR mixture was placed in an Applied Biosystems Gene Amp PCR System 9700 thermocycler Roche Molecular Systems Inc. Branchburg, NJ) for amplification at following times and temperatures: initial step 94°C for 3 min then three cycles of; denaturation 94°C for 30 s, annealing 53°C for 30 s, elongation 72°C for 30 s, and 37 cycles of each; denaturation 94°C for 30 s, annealing 60°C for 30 s, elongation 72°C for 30 s, and final elongation 72°C for 7 min.

Ten microliters of PCR product was detected using agarose gel electrophoresis. The 2% agarose gel was stained by adding 5 μl of molecular grade ethidium bromide solution 10 mg/ml (Promega, Madison, WI) to the liquid gel buffer mixture. An Alpha Imager 2000 (Alpha Innotech Company, San Leandro, CA) was used to visualize the resulting 179 base pair HPIV1 product bands. Thirty microliters of positive sample were purified using a QIAquick PCR Purification Kit from Qiagen Inc. (Valencia, CA). Positive samples were sequenced for verification of parainfluenza 1, and to verify genetic discrimination from the positive control. Sample genomic sequencing was conducted at the University of Arizona Genomic Analysis Technical Center using a 377 ABI sequencer from Applied Biosystems (Roche Molecular Systems Inc. Branchburg, NJ).

Systat 9.0 Chi-square analysis and the Fisher's Exact test were used for statistical analysis of the sample test results.

Results

HPIV1 was detected on one of the two pens, and one of the six remotes tested; but these data were not included in the analysis due to the small number of object samples collected. Overall, 37% of the samples tested were positive for HPIV1 (Fig. 1). HPIV1 was detected more often on the desktops (47%), the computer mouse (46%), and the phone (45%) (Fig. 1). Viruses were isolated least often on door handles (26%) and light switches (19%) as seen in Fig. 1. The quantity of positive surfaces varied within object category from 20% (New York phone) to 66% (Atlanta phone) as seen in Fig. 2. Data indicated a statistically significant difference within the computer mouse category between the cities of Atlanta (0%) and New York (100%), Chi-square $P < 0.0000$ and Fisher's Exact test $P = 0.000005$ (Fig. 2). Differences between the total quantities of positive fomites per building were also assessed (Fig. 3). There was a statistically significant difference found between the occurrence of HIPV1 in Arizona building 1 (86%) and building 5 (14%), Chi-square analysis $P < 0.003$ and Fisher's Exact test $P = 0.0017$. Additionally, results indicated a variation in the quantity of positive fomites detected in cubicles, offices, and conference rooms (Fig. 4). Data also revealed a statistically significant difference between the total quantity of positive fomites found in cubicles and conference rooms, Chi-square analysis $P < 0.011$ and Fisher's Exact test $P = 0.054$ as seen in Fig. 4.

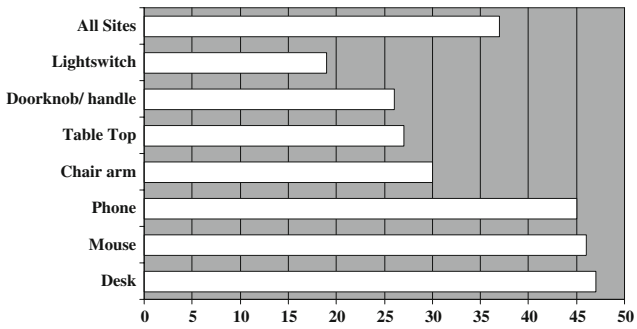

Fig. 1 Percentage of fomites positive for HPIV1

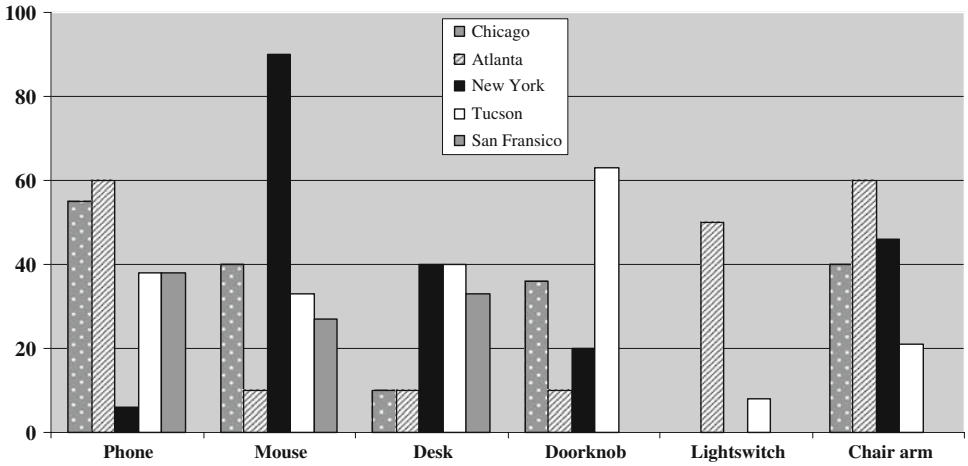

Fig. 2 Fomites positive for HPIV1 in each city

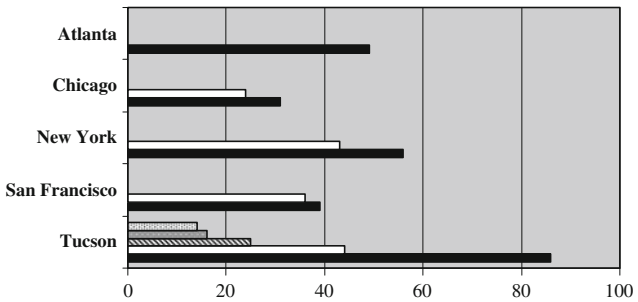

Fig. 3 Percentage of HPIV1 positive fomites by buildings in each city. Two buildings were assessed in each city except Tucson (five buildings) and Atlanta (one building)

Discussion

This study is the first to investigate the occurrence of HPIV1 or any virus on indoor office surfaces. HPIV1 was detected in all of the office buildings tested during 2004, a low incidence year. Positive fomite samples obtained during a year with low HPIV1 incidence reinforces the theory that viruses can be spread easily through closed indoor environments (Goldman 2000). Previous research showed that nasal secretion droplets containing infectious viral particles generated via coughing, sneezing, and talking are easily transmitted over considerable distances (Barker et al. 2001). Studies by Zhao et al. (2005) indicated that an average sneeze can travel up to 3 m at a speed of 20 m/sec. In addition, several studies have shown that viruses can be transferred from surfaces to hands, and vice versa (Aitken and Jefferies 2001; Ansari et al. 1991, 1988; Barker et al. 2001; Goldman 2000). The average survival of HPIV is 10 h on nonporous surfaces, and over an hour on hands (Aitken and Jefferies 2001; Ansari et al. 1991; Barker et al. 2001; Brady et al. 1990). The majority of positive fomites in this study appear to have resulted from the transfer of contaminated nasal secretions to fomites by touching (hand to nose or mouth, surfaces, etc.), sneezing, coughing, and/or talking. Research by Couch et al. (1974) indicated that up to 10^7 infectious virions can be shed per ml of respiratory secretions. Viral shedding may occur asymptomatically,

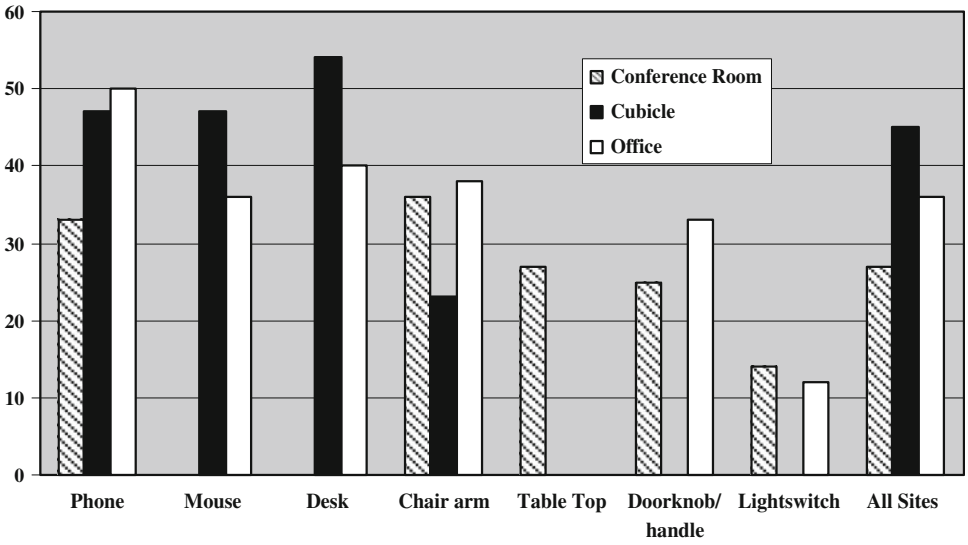

Fig. 4 Percentage of HPIV1 positive fomites by work area

before symptom onset or continue for several days or weeks after symptoms have ceased (Barker et al. 2001; Goldman 2000). Consequently, the shed and spread of HPIV1 by adult individuals with active infections is indicated by the occurrence of HPIV1 RNA on office surfaces.

Studies investigating the survival of viruses on surfaces indicate very little variation in viral decay rates on nonporous surfaces (Boone and Gerba 2007). Viruses typically survive for longer periods of time on nonporous surfaces as compared to porous surfaces (Abad et al. 1994). Virus survival on surfaces can also be influenced by environmental changes in humidity and temperature depending on the virus (Boone and Gerba 2007). However, research indicates that temperature and humidity variations within 10° centigrade have little effect on viral viability (Abad et al. 1994 Sattar et al. 1987). All the surfaces sampled in this study were nonporous indoor surfaces, as a result the variables of surface, temperature, and humidity were considered to have an equal impact on the presence of viral RNA. In general, surface exposure or frequency of use has a significant impact on surface contamination (Sattar 2001). Normally, conference rooms are used less frequently than offices or cubicles. Study data indicated more surfaces positive for HPIV1 in cubicles and offices as compared to the conference rooms. Also, there was a significant difference between the percentages of positive surfaces detected in cubicles when compared to conference rooms. Light switches in office buildings tested were usually turned off at the beginning and the end of the day, and therefore used infrequently. On average doors were only opened and closed at the beginning and end of the day indicating infrequent use. As seen in Fig. 1, both door knobs and light switches were found to be contaminated with HPIV1 less often than more frequently used surfaces like the phone, desk, or the computer mouse.

The offices sampled in Arizona indicated a greater variation in the total percentage of HPIV1 positive surfaces per building (Fig. 3). Variation in the number of positive surfaces detected per office building may have been due to dissimilarities in office building population infection rate, and/or a reflection of surface usage. Arizona office buildings varied in exposure to incoming students or patrons and the sharing of office space. The Arizona office buildings with the most HPIV positive surfaces were offices in the athletic department, which experience a high volume of incoming student patrons, and work space is shared with more than two people. In addition, several workers were reported to be ill simultaneously during the sampling period. The Arizona office buildings with the fewest positive surfaces were offices in accounting, with no incoming student patrons and work space used by only one person. Surfaces in high traffic office areas consequently may be exposed to more infected individuals.

In addition to surfaces usage, variations in HPIV1 positive surfaces may also be a reflection of office cleaning practices (frequency of wiping surfaces to remove matter) and surface disinfection (use of a product that kills or inactivates microorganisms). Offices with fewer HPIV1 positive surfaces may use disinfectants that degrade viral RNA (i.e., chlorine- or peroxide-based disinfectants) or may clean surfaces more frequently and thus removing the virus from the surface. In contrast an increase in surface contamination may also occur if the office is being cleaned with inefficient sanitizers or disinfectants, which can spread viruses and cross-contaminate clean surfaces (Barker et al. 2004). The positive samples obtained in this study are reflective of both infective and noninfective HPIV1 viruses due to the detection method used (PCR), and it was not determined if the virus was infective. Additional research is needed to assess the infectivity of the virus when transferred from fomites. Office surfaces like the telephone receiver, mouse, and desk tops are frequently used, but infrequently cleaned or disinfected. General cleaning with disinfectants that degrade viral RNA can reduce the possibility of viral transfer, transmission, and possible infection (Aitken and Jefferies 2001; Ansari et al. 1991; Brady et al. 1990; Stienmann 2001).

References

Abad, F. X., Pinto, R. M., & Bosch, A. (1994). Survival of enteric viruses on environmental fomites. *Applied Environmental Microbiology, 60*, 3704–3710.

Aitken, C., & Jefferies, D. J. (2001). Nosocomial spread of viral disease. *Clinical Microbiology Reviews, 14*, 528–546.

Alguilar, J. C., Perez-Brena, M. P., Garcia, M. L., Cruz, N., Erdman, D. D., & Echevarria, J. E. (2000). Detection and identification of human parainfluenza viruses 1, 2, 3 and 4 clinical samples of pediatric patients by multiplex reverse transcription–PCR. *Journal of Clinical Microbiology, 38*, 1191–1195.

Ansari, S. A., Sattar, S. A., Springthrope, G. A., Wells, G. A., & Tostowaryk, W. (1988). Rotavirus survival on human hands and transfer of infectious virus to animate and non-porous inanimate surfaces. *Journal of Clinical Microbiology, 26*, 1513–1518.

Ansari, S. A., Springthrope, V. S., Sattar, S. A., Rivard, S., & Rahman, M. (1991). Potential role of hands in the spread of respiratory viral infections: Studies with human Parainfluenza virus 3 and Rhinovirus 14. *Journal of Clinical Microbiology, 29*, 2115–2119.

Barker, J. D., Stevens, D., & Bloomfield, S. F. (2001). Spread and prevention of some common viral infections in community facilities and domestic homes. *Journal of Applied Microbiology, 91*, 7–21.

Barker, J. D., Vipond, I. B., & Bloomfield, S. F. (2004). Effects of cleaning and disinfection in reducing the spread of norovirus contamination via environmental surfaces. *Journal of Hospital Infection, 58*, 42–49.

Boone, S. A., & Gerba, C. P. (2005). The occurrence of influenza A virus on day care and household fomites. *Journal of Infection, 51*, 103–109.

Boone, S. A., & Gerba, C. P. (2007). Significance of fomites in the spread of respiratory and enteric viral disease. *Applied and Environmental Microbiology, 73*, 1687–1696.

Brady, M. T., Evans, J., & Cuartas, J. (1990). Survival and disinfection of parainfluenza viruses on environmental surfaces. *American Journal of Infection Control, 8*, 18–23.

CDC Center for Disease Control. (2009). National Center for infectious diseases, respiratory and enteric branch; human parainfluenza viruses. http://www.cdc.gov/ncidod/dvrd/revb/respiratory/hpivfeat.htm. Accessed November, 2009.

Couch, R. B., Kasel, J. A., Gerin, J. L., Schulman, J. L., & Kibourne, E. D. (1974). Induction of partial immunity to influenza by a neuraminidase-specific vaccine. *Journal of Infectious Diseases, 129*, 411–420.

Donaldson, A. I., & Ferris, N. P. (1976). The survival of some airborne animal viruses in relation to relative humidity. *Veterinary Microbiology, 1*, 413–420.

Echevarria, J. E., Erdman, D. D., Swierkosz, E. M., & Holloway, B. P. (1998). Simultaneous detection and identification of human parainfluenza viruses 1, 2, and 3 from clinical samples by multiplex PCR. *Journal of Clinical Microbiology, 36*, 1388–1391.

Falsey, A. R. (1991). Non-influenza respiratory infection in long-term care facilities. *Infection Control Hospital Epidemiology, 12*, 602–608.

Falsey, A. R., Cunningham, C. K., Baker, W. H., Kouides, R. W., Yuen, J. B., Menegus, M., et al. (1995). Respiratory syncytial virus and influenza A infections in hospitalized elderly. *Journal of Infectious Disease, 172*, 389–394.

Fan, J., & Hendrickson, K. J. (1996). Rapid diagnosis of human parainfluenza virus type 1 infection by quantitative reverse transcription–PCR-enzyme hybridization assay. *Journal of Clinical Microbiology, 34*, 1914–1917.

Fiore, A. E., Iverson, C., Messmer, T., Erdman, D., Lett, S. M., Talkington, D. F., et al. (1998). Outbreak of pneumonia in a long-term care facility; antecedent human parainfluenza virus 1 infection may predispose to bacterial pneumonia. *Journal of American Geriatric Society, 46*, 1112–1117.

Glezen, W. P., & Floyd, W. D. (1997). Parainfluenza viruses. In A. S. Evans & R. Kaslow (Eds.), *Viral infections in humans, epidemiology and control* (4th ed., pp. 552–554). New York: Plenum Press.

Goldman, D. A. (2000). Transmission of viral respiratory infections in the home. *Pediatric Infectious Disease Journal, 19*, S97–S102.

Hendrickson, K. J. (2003). Parainfluenza viruses. *Clinical Microbiology Reviews, 16*, 2242–2264.

Henrickson, K. J., Kuhn, S. M., Savatski, L. L., & Sedmak, J. (1994). Recovery of human parainfluenza virus types one and two. *Journal of Virological Methods, 46*, 189–206.

Hota, B. (2004). Contamination, disinfection and cross-colonization: Are hospital surface reservoirs for nosocomial infection? *Clinical Infectious Disease, 39*, 1182–1189.

Louie, J. K., Hacker, J. K., Gonzales, R., Mark, J., Maselli, J. H., Yagi, S., et al. (2005). Characterization of viral agents causing acute respiratory infection in a San Francisco University medical center clinic during influenza season. *Clinical Infectious Diseases, 41*, 822–828.

Mclean, O. M., Bannatyne, R. M., & Givan, K. F. (1967). Myomxovirus dissemination by air. *Journal of Canadian Medical Association, 96*, 1449–1453.

Monto, A. S. (2002). Epidemiology of viral respiratory infections. *The American Journal of Medicine, 112*, S4–S12.

Monto, A. S. (2004). Occurrence of respiratory virus: Time, place and person. *Pediatric Infectious Disease Journal, 23*, S58–S64.

Perz, J. F., Craig, A. S., & Schaffner, W. (2001). Mixed outbreak of parainfluenza type 1 and influenza B associated with tourism and air travel. *International Journal of Infectious Disease, 5*, 189–191.

Sattar, S. A. (2001). Survival of microorganisms on animate and inanimate surfaces and their disinfection. In W. A. Rutala (Ed.), *Disinfection, sterilization and antisepsis: Principles and practices in healthcare facilities* (pp. 195–205). Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc.

Sattar, S. A., Karim, Y. G., Springthorpe, V. S., & Johnson-Lussenburg, C. M. (1987). Survival of human rhinovirus type 14 dried onto nonporous inanimate surfaces: Effect of relative humidity and suspending medium. *Canadian Journal of Microbiology, 33*, 802–806.

Stienmann, J. (2001). Some principles of virucidal testing. *Journal of Hospital Infection, 48*, S15–S17.

USEPA United States Environmental Protection Agency. (2004). Quality assurance/quality control guidance for laboratories performing PCR analyses on environmental samples. http://www.epa.gov/microbes/qa_qc_pcr10_04.pdf. Accessed November, 2009.

Vilchez, R., McCurry, K., Dauber, J., Iaconco, A., Keenan, R., Griffith, B., et al. (2002). Influenza and parainfluenza respiratory viral infection requiring admission in adult lung transplant recipients. *Transplantation, 73*, 1075–1078.

Ward, R., Bernstein, D., Knowlton, D., Sherwood, J., Young, E., Cusack, T. M., et al. (1991). Prevention of surface to human transmission of rotavirus by treatment with disinfectant spray. *Journal of Clinical Microbiology, 29*, 1991–1996.

Zhao, B., Zhao, Z., & Xianing, L. (2005). Numerical study of the transport of droplets or particles by respiratory systems indoors. *Building and Environment, 4*, 1032–1039.