

# Nosocomial Transmission of Respiratory Syncytial Virus in an Outpatient Cancer Center



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## ABSTRACT

Respiratory syncytial virus (RSV) outbreaks in inpatient settings are associated with poor outcomes in cancer patients. The use of molecular epidemiology to document RSV transmission in the outpatient setting has not been well described. We performed a retrospective cohort study of 2 nosocomial outbreaks of RSV at the Seattle Cancer Care Alliance. Subjects included patients seen at the Seattle Cancer Care Alliance with RSV detected in 2 outbreaks in 2007–2008 and 2012 and all employees with respiratory viruses detected in the 2007–2008 outbreak. A subset of samples was sequenced using semi-nested PCR targeting the RSV attachment glycoprotein coding region. Fifty-one cases of RSV were identified in 2007–2008. Clustering of identical viral strains was detected in 10 of 15 patients (67%) with RSV sequenced from 2007 to 2008. As part of a multimodal infection control strategy implemented as a response to the outbreak, symptomatic employees had nasal washes collected. Of 254 employee samples, 91 (34%) tested positive for a respiratory virus, including 14 with RSV. In another RSV outbreak in 2012, 24 cases of RSV were identified; 9 of 10 patients (90%) had the same viral strain, and 1 (10%) had another viral strain. We document spread of clonal strains within an outpatient cancer care setting. Infection control interventions should be implemented in outpatient, as well as inpatient, settings to reduce person-to-person transmission and limit progression of RSV outbreaks.

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## INTRODUCTION

Respiratory syncytial virus (RSV) causes substantial morbidity and mortality among hematopoietic stem cell transplantation (HSCT) and oncology patients who are at high risk for progression to lower respiratory tract infection (LRTI)-associated respiratory failure and death [1]. Mortality rates for RSV-associated LRTI range from 15% to 70% [2,3]. Treatment regimens for RSV-associated LRTI include aerosolized ribavirin, often in combination with palivizumab or intravenous immunoglobulin; supplemental oxygen; and respiratory support [4–6]. However, antiviral therapies are expensive, difficult to administer, and have not reliably prevented progression to LRTI [7,8]. With no available vaccine or prophylaxis measures available for adults, infection control practices remain the only effective method to limit RSV outbreaks among adult cancer patients.

RSV may be acquired in a health care setting and has been implicated in outbreaks in inpatient hematology-oncology and transplant wards [9]. Hospital-based outbreaks of RSV

infection in HSCT recipients occur through introduction of circulating community strains as well as nosocomial transmission of identical viral strains [10]. The molecular epidemiology of RSV is characterized by sequencing a hypervariable region of the attachment (G) glycoprotein gene [11,12]. Evidence of acquisition of the same viral strain in inpatient cancer care settings has demonstrated the importance of specific infection control policies to prevent nosocomial RSV transmission, although data describing this in the outpatient setting are not available [13]. Studies have shown outpatient transmission of parainfluenza, a respiratory virus also associated with high mortality in immunocompromised cancer patients [14].

Efforts to enhance infection control to prevent RSV spread include strict hand hygiene, use of droplet precautions, cohorting of nursing staff, and symptom screening of employees and visitors [15,16]. Previous studies in inpatient settings have shown that the number of RSV-positive cases decreased after implementation of these infection control interventions [17]. However, most cancer care is now delivered in the outpatient setting. The routine use of antibiotic prophylaxis and hematopoietic growth factors has reduced many risks associated with prolonged neutropenia and prolonged hospital stays [18]. It has been assumed that outpatients generally acquire their respiratory infections in the community through routine daily activities, such as work and exposure to children, and not through contact within the health care setting. Few data are currently available on transmission of RSV infection in

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the outpatient cancer care setting. In this study molecular virologic methods were used to demonstrate nosocomial transmission of RSV during 2 RSV outbreaks at a large outpatient cancer care center.

**METHODS**

The Seattle Cancer Care Alliance (SCCA) is an inpatient and outpatient cancer care center based in Seattle, Washington. In 2012, 5599 patients were treated for cancer at the SCCA over the course of 72,300 visits. Patients at the SCCA are seen by teams of providers in different physical locations at a single site, divided by type of cancer or therapy, designated as Teams A through F. Allogeneic stem cell transplant recipients were seen by Teams A and B, autologous stem cell transplants were seen by Team C, pediatric patients were seen by Team D, patients in long-term follow-up were seen by Team E, and hematology-oncology patients were seen by Team F providers. The SCCA infection control team tracks incident RSV cases using an electronic system that identifies patients by provider team and location.

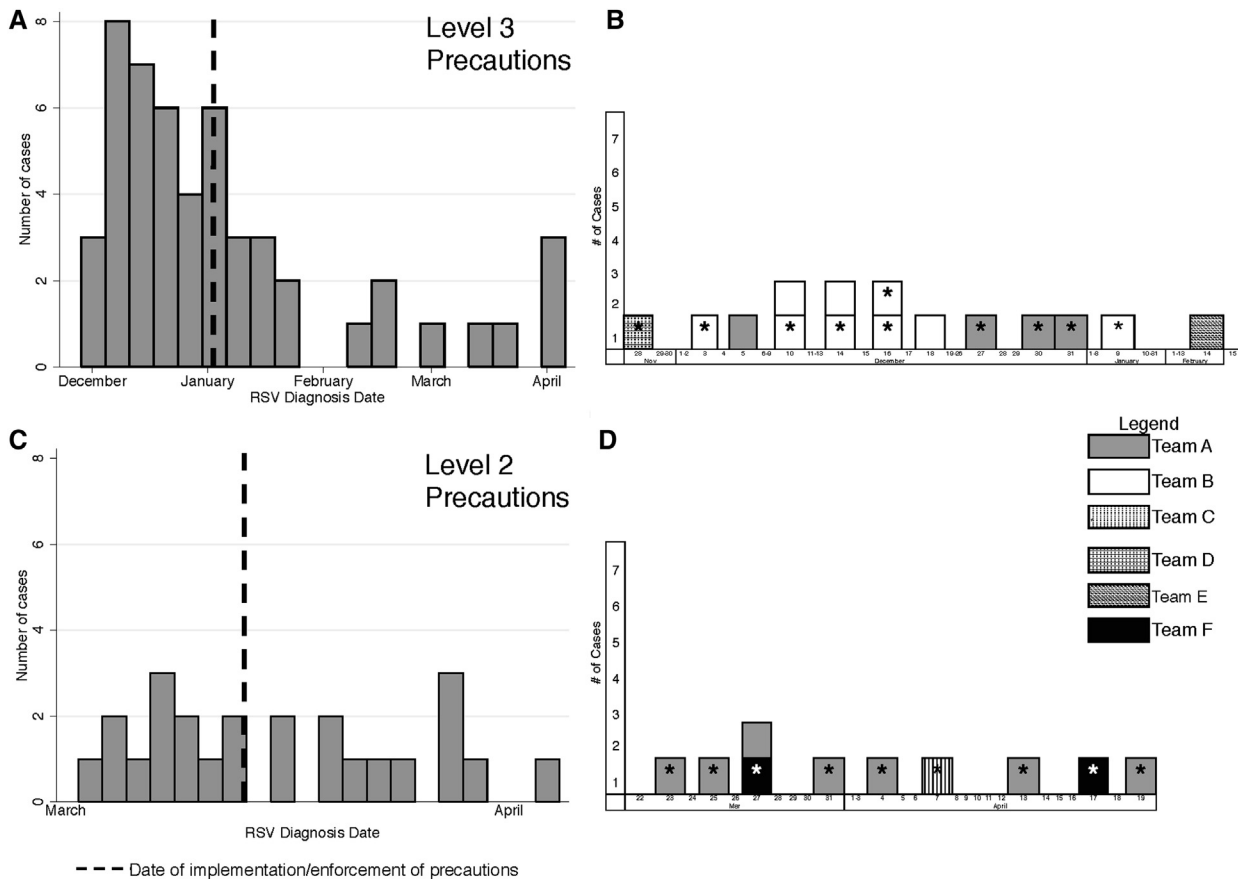
Electronic medical records were used to abstract sociodemographic and clinical data for patients with RSV detected during the 2 outbreak periods. Lymphopenia was defined as  $\leq 500$  cells/ $\mu\text{L}$  and severe lymphopenia as  $\leq 300$  cells/ $\mu\text{L}$ . Respiratory specimens were obtained in patients for testing by nasal washes or from bronchoalveolar lavage fluid when clinically indicated by the primary treatment team [19]. Direct fluorescent antibody detection was performed using RSV-specific mouse monoclonal antibodies (Chemicon, Temecula, CA) on all nasal wash samples before January 21, 2008, and reverse transcriptase PCR was performed afterward at the University of Washington Virology Laboratories using previously published methods [19,20]. All bronchoalveolar lavage samples undergo routine direct testing for RSV using direct fluorescent antibody, shell vial culture, and/or reverse transcriptase PCR testing.

During the 2007–2008 outbreak, all employees at the SCCA were administered a daily 12-symptom respiratory screening paper questionnaire for presence of runny nose, sinus congestion/stuffy nose, postnasal drip,

shortness of breath, cough, wheezing or chest tightness, sputum production, sore throat, sneezing, watery eyes, ear pain, or fever (temperature  $> 100.4^\circ\text{F}$ ) and had a nasal wash collected for respiratory viral testing per institutional policy at the time. Testing for RSV and 11 other respiratory viruses, including influenza A and B, human metapneumovirus, parainfluenza 1–4, rhinovirus, human coronavirus groups 1 and 2, and bocavirus were performed on employee samples using previously published methods [19,21]. Employees who had a positive nasal wash for any respiratory virus were not permitted to return to work until complete resolution of all symptoms and clearance by Occupational Health. To compare the outbreak with community data, rates of RSV detected in community samples from the Seattle and Pacific Northwest region were obtained from the University of Washington Diagnostic Virology Laboratory database (<http://depts.washington.edu/rspvirus/respiratory.htm>).

Sequencing was attempted from residual RSV-positive samples from the 2 respiratory seasons using a semi-nested PCR protocol targeting the second hypervariable region of the attachment glycoprotein coding region [22]. Random residual de-identified RSV-positive community samples collected from subjects seeking medical care during the same respiratory seasons were also sequenced to serve as control subjects. Sequences were submitted to GenBank with accession numbers KC565494 to KC565526. Sociodemographic, clinical, and virologic data were analyzed using Stata 12.0 (STATA Corp, College Station, TX). Nucleotide sequences for 233 and 212 base pair regions of the second hypervariable region of the RSV attachment glycoprotein coding region were aligned using ClustalX2 [23].

Phylogenetic trees were constructed separately for the 2007–2008 and 2012 outbreaks using MEGA5 with evolutionary distances calculated using the maximum likelihood method with 1000 bootstrap replicates [24]. This was performed using the Tamura-Nei model. The tree with the highest log likelihood is shown. When the number of common sites was 100 or less than one-fourth of the total number of sites, the maximum parsimony method was used; otherwise, the BioNJ method with MCL distance matrix was used. The trees were drawn to scale, with branch lengths measured in the number of substitutions per site.



**Figure 1.** Histogram of all RSV cases at the SCCA per day in the 2007–2008 outbreak (A) and only cases where the viral strain was sequenced (B). Histogram of all RSV cases at the SCCA per day in the 2012 outbreak (C) and only cases where the viral strain was sequenced (D). The asterisk represents an identical viral strain for that season. The pattern of the box represents the team of providers seen by the patient.

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