Rhinovirus is a common cause of exacerbations of cystic fibrosis (CF) and is usually considered a self-limiting infection. We report a case of chronic infection with rhinovirus A type 33 in a 43-year-old male with CF which has persisted for over 2 years.

CASE REPORT

A 43-year-old man with cystic fibrosis (CF) was diagnosed with chronic rhinovirus A infection through a combination of PCR assays and genetic sequencing. His CF genotype was F508del/G542X, and he had moderate to severe CF-related lung disease (forced expiratory volume in 1 s, 42% [predicted at baseline]). The patient was known to have allergic bronchopulmonary aspergillosis (ABPA) and chronic endobronchial infection with *Pseudomonas aeruginosa*. He had received high-dose inhaled corticosteroids (fluticasone at 1,000 μg per day) and oral itraconazole (100 mg) twice daily for the previous 4 years.

In January 2011, the patient enrolled in an observational study investigating the role of respiratory viruses in adults with CF (1). At recruitment to the study, he was clinically stable and had no symptoms suggestive of an acute respiratory tract infection. In-house PCR assays for a total of nine respiratory viruses were performed on sputum and nose and throat swab specimens following total nucleic acid extraction using a QIAamp Virus Biorobot MDx instrument (Qiagen, Hilden, Germany). The rhinovirus PCR assay targeted the 5′-noncoding region in line with the method reported by Scheltinga et al. (2). Details of the primers and probes are given in Table 1. The sputum sample was PCR positive for rhinovirus, with a cycle threshold value of 33 cycles. Both upper-airway swabs were negative for viral pathogens.

During the following 12 months, the patient provided respiratory samples for virological analysis on a total of eight occasions. At seven of the study visits, rhinovirus was identified in at least one respiratory tract sample (see Table 2). On the eighth occasion, nose and throat swabs were negative for rhinovirus but the patient was unable to expectorate sputum. The patient’s clinical course during this period was complicated by the diagnosis of adrenal insufficiency in August 2011. Itraconazole treatment was stopped, and the dose of inhaled fluticasone was reduced to 400 μg daily. Replacement oral hydrocortisone was commenced and has continued to the present day.

In March 2013, more than 2 years after its initial identification, the patient provided a further sputum sample which was again PCR positive for rhinovirus. Overall, only three of the patient’s rhinovirus-positive episodes were associated with symptoms of an upper respiratory tract infection and just one met predefined criteria for a pulmonary exacerbation of CF lung disease (3).

Genetic sequencing of the rhinovirus 5′-untranslated region was performed in five of the patient’s specimens using an Applied Biosystems 3130xl Genetic Analyzer and following the method reported by Lee et al. (4). Details of the primers used...
TABLE 2 Serial rhinovirus PCR results and symptomatology from an adult with cystic fibrosis

<table>
<thead>
<tr>
<th>Date of specimen collection</th>
<th>Symptom(s) of URTI?</th>
<th>Pulmonary exacerbation?</th>
<th>Rhinovirus PCR result (cycle threshold value [specimen no.])</th>
<th>Sample sequenced?</th>
</tr>
</thead>
<tbody>
<tr>
<td>21 January 2011</td>
<td>No</td>
<td>No</td>
<td>Neg (41 [M11914546])</td>
<td>No</td>
</tr>
<tr>
<td>4 March 2011</td>
<td>Yes</td>
<td>Yes</td>
<td>Pos (38 [M11914547])</td>
<td>Yes</td>
</tr>
<tr>
<td>3 June 2011</td>
<td>No</td>
<td>No</td>
<td>Neg (33)</td>
<td>No</td>
</tr>
<tr>
<td>8 July 2011</td>
<td>Yes</td>
<td>Yes</td>
<td>Neg (34)</td>
<td>No</td>
</tr>
<tr>
<td>31 August 2011</td>
<td>Yes</td>
<td>No</td>
<td>Neg (33)</td>
<td>No</td>
</tr>
<tr>
<td>2 November 2011</td>
<td>No</td>
<td>No</td>
<td>Neg (31 [M11960169])</td>
<td>Yes</td>
</tr>
<tr>
<td>30 November 2011</td>
<td>Yes</td>
<td>No</td>
<td>Neg (34)</td>
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<tr>
<td>4 January 2012</td>
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<td>No</td>
<td>Pos (39)</td>
<td>Yes</td>
</tr>
<tr>
<td>21 March 2013</td>
<td>No</td>
<td>No</td>
<td>Pos (35 [M13907462])</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*Specimen identity numbers beginning with “M” identify the samples shown in Fig. 1. All samples from the patient were PCR negative for adenovirus, influenza A and B virus, metapneumovirus, parainfluenza 1 to 3, and respiratory syncytial virus. URTI, upper respiratory tract infection; Neg, negative; Pos, positive.*
infection are contagious and represent an infection control risk. Until such issues are addressed, this case report serves to highlight that CF lung disease may be complicated by chronic respiratory infection with viruses as well as bacterial and fungal pathogens.

REFERENCES
2. Scheltinga SA, Templeton KE, Beersma MF, Claas EC. 2005. Diagnosis...


