arthropod-based transmission *F. philomiragia* has not been suspected. However, *F. philomiragia* DNA was found in 19% of a sample of dog ticks (*Dermacentor reticulatus*) in France (9). This finding suggests that *D. reticulatus*, which is now broadly distributed across Europe because of global warming and increased travel with pets, may have a role in the life cycle and transmission of *F. philomiragia* (10). The patient did not own a dog and did not recall having had contact with dogs. However, his job (a municipal gardener) constituted a risk factor for tick bites in urban green spaces.

Although multiple points for *F. philomiragia* to enter this patient were suspected, none were laboratory confirmed. Further investigation is needed to better define the natural life cycle of this organism, especially the role of tick species in its transmission.

Acknowledgments
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References

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Severe Ocular Cowpox in a Human, Finland

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To the Editor: We describe cowpox with corneal involvement in a 31-year-old atopic woman who lived in southern Finland and was unvaccinated for smallpox. In August 2009, she noticed irritation and edema in her right eye and sought care from a local physician; she started topical antimicrobial drug therapy and oral cephalaxin 2 days later. Over the following week, fever developed (37.6°C–39.0°C), edema developed on half her face, the eye became increasingly painful, and visual acuity decreased. The conjunctiva was severely chemotic and hyperemic, but the cornea was clear and the other eye unaffected.

Microbiologic samples taken from the eye 11 days after onset showed neither bacteria nor respiratory viruses. Orbital tomography results were normal. The patient was hospitalized, and broad-spectrum intravenous antimicrobial treatment (meropenem, vancomycin, valacyclovir, and fluconazole) was started, combined with topical corticosteroids and antimicrobial drugs. Within 2 weeks, the conjunctiva showed necrosis, and epithelial erosions appeared in the lower cornea, but visual acuity normalized (online Technical Appendix Figure, panels A, B, http://wwwnc.cdc.gov/EID/article/21/12/15-0621-Techapp.pdf).

A strong cytopathic effect was observed in Vero cells infected with conjunctival swab (online Technical Appendix
Table 1), but the virus was unidentifiable by routine methods. In electron microscopy, cell culture and tear fluid samples contained particles with typical orthopoxvirus (OPV) morphology. PCRs for hemagglutinin (1) and 14-kDa genes (2) verified OPV infection. Additional PCRs and sequencing confirmed zoonotic cowpox virus (CPXV) with strain designation FIN/K2009. Nucleotide sequences of the hemagglutinin, thymidine kinase, and A-type inclusion body protein genes were identical to those of CPXV strains T2000 and E1989 previously identified in Finland (3). In phylogenetic analysis (Figure), CPXV/FIN/K2009 clustered with strains from Austria and shared ancestry with vaccinia virus. OPV IgG and IgM were detected by immunofluorescence assay (3) in serum samples up to 5 months after symptom onset (online Technical Appendix Table 1).

The patient was started on intravenous polyclonal gammaglobulin and topical trifluorothymidine with in vitro anti-OPV effects; nevertheless, corneal erosions enlarged, corneal stromal edema ensued, and intraocular pressure increased (online Technical Appendix Figure, panel C), suggesting trabeculitis. Topical autologous serum drops had no effect. Periocular edema slowly resolved, but corneal erosions persisted. Amniotic membrane transplantation (AMT) (4) was performed 2.5 months after onset. The inferior cornea melted, and the cornea lost transparency (online Technical Appendix Figure, panel D). AMT was repeated twice at 1-month intervals because of corneal thinning.

At 3.5 months after symptom onset, tecovirimat (400 mg 2×/d) was given orally for 14 days. Despite treatment, ocular OPV PCR test results remained positive until 9 months after onset (online Technical Appendix Table 1), and corneal melting progressed (online Technical Appendix Figure, panel E). Corneal collagen cross-linking and a fourth AMT were performed at 5 months after onset with partial success (online Technical Appendix Figure, panel F).

At 1 year after symptom onset, corneal limbal stem cell deficiency with deep corneal neovascularization was evident. Autologous limbal stem cell transplantation from the patient’s other eye and another AMT were performed, resulting in stable corneal surface 2 months later (online Technical Appendix Figure, panel G). Neovascularization regressed, the cornea cleared, and vision improved (online Technical Appendix Figure, panels H, I).

Cowpox is transmitted to humans sporadically from rodents or cats (5). We snap-trapped 23 wild rodents from the yard of the patient’s home and from an adjacent meadow and trapped 136 rodents from 3 other regions 30–100 km from the patient’s home (online Technical Appendix Table 2). We also collected 8 environmental samples from the patient’s storehouse. In accordance with the Finnish Act on Use of Animals for Experimental Purposes (62/2006) and the Finnish Animal Experiment Board’s later decision (May 16, 2007), the animal capture technique used is not an animal experiment and requires no ethics license.

Diluted blood for IFA was collected from all rodents (6), and DNA was extracted from rodent liver and lungs and from environmental samples. One vole and 1 mouse from the meadow were seropositive for OPV; however,
no CPXV DNA was amplifiable in the samples from the liver, lungs, or environment (online Technical Appendix Table 2).

CPXV infection may manifest in severe ocular forms along with self-limiting cutaneous pocks (5). Our patient had keratitis with no other identifiable cause but CPXV. Culture and PCR from early conjunctival samples and serology confirmed the etiologic diagnosis.

Our case and that of another report (7) highlight the challenges of treating cowpox keratitis. Topical and systemic antiviral drugs and AMT appear ineffective during the acute phase. Corneal melting and scarring continued as long as CPXV was observed and until combined limbal stem cell and AMT treatment had favorable outcomes. Amnesia of therapy-resistant keratitis should include information on rodent contacts.

We dated the infection to mid-August (incubation 7–21 days). Catching OPV-IgG–positive rodents close to the patient’s home 2 months after onset showed that OPVs were circulating in the local rodent population and indicated the putative role of CPXV-infected voles as the source of infection.

The latest cowpox outbreak in Central Europe involved several humans and pets (8). This patient was born in 1977, after Finland ceased smallpox vaccinations. Declining cross-reactive smallpox-vaccination immunity enables emergence of unusual cowpox infections in humans (9).

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Human Alveolar Echinococcosis, Czech Republic, 2007–2014

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DOI: http://dx.doi.org/10.3201/eid2112.150743

To the Editor: Human alveolar echinococcosis (AE) is a zoonotic parasitic disease caused by larvae of Echinococcus multilocularis tapeworms that manifests most often in the host’s liver, although it can infect the lungs, brain, and other organs. Clinical symptoms usually develop after a long incubation period (5–15 years), causing considerable diagnostic difficulties and delay in treatment. The disease is reported in all countries neighboring the Czech Republic: Slovakia, Poland, Austria, and Germany (1,2). To complete data from central Europe, we present results on the occurrence of AE in the Czech Republic collected by the National Reference Laboratory for Tissue Helminthoses during 2007–2014.

Epidemiological data on AE was collected at the Department of Internal Medicine, Charles University First Faculty of Medicine, Prague, Czech Republic, from 2007 to 2014. AE is considered in patients presenting with abdominal or chest pain, fever, or weight loss and found to have cystic masses in the liver, lungs, or other organs. CT images (n = 24) and MRI images (n = 18) were reviewed by radiologists and specialists. Liver biopsies (n = 5) and lung biopsies (n = 1) were examined by pathologists. The diagnosis of AE is based on the finding of protoscoleces in the cystic material and the presence of the E. multilocularis DNA in samples from the patients (3–5).

Our study adds to the data on AE in Central Europe. This information will be helpful in guiding public health authorities in the Czech Republic, as well as in other countries neighboring the Czech Republic, in their surveillance efforts and control strategies for AE.

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Severe Ocular Cowpox in a Human, Finland

Technical Appendix

Technical Appendix Table 1. Orthopoxvirus and cowpox virus diagnostic findings of the patient in Finland*

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Months after symptom onset, no.</th>
<th>0.5</th>
<th>1</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>8</th>
<th>≥9</th>
</tr>
</thead>
<tbody>
<tr>
<td>OPV IgG titer</td>
<td>1,280</td>
<td>1,280</td>
<td>320</td>
<td>320</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>OPV IgM titer</td>
<td>320</td>
<td>1,280</td>
<td>40</td>
<td>40</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Real-time PCR from conjunctival swab/ contact lens, HA gene, Cycle threshold values</td>
<td>Swab 33.7</td>
<td>Swab 35.7</td>
<td>Swab 41.4/Lens 43.1</td>
<td>Swab 35.4/Lens 38.2</td>
<td>Swab 48.3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Virus isolation: CPE in Vero cells and electron microscopy</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*OPV, Orthopoxvirus; NA, Not analyzed; CPE, Cytopathogenic effect; HA, Hemagglutinin, 1,091 bp, GenBank accession no. KR054112; TK, Thymidine kinase, 342 bp, KR054113; ATIP, A-type inclusion body protein, 1,590 bp, KR054111; +, positive; –, negative.

Technical Appendix Table 2. Results from the environment and rodents from the yard of the patient’s home, its vicinity, and more distant regions

<table>
<thead>
<tr>
<th>Sample description</th>
<th>No. samples</th>
<th>No. positive in IFA (%)</th>
<th>Comments on positive samples</th>
<th>No. positive in PCR* (HA and 14 kDa protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental samples from the patient’s outdoor storehouse†</td>
<td>8</td>
<td>0</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Dead insect, pieces of dry leaves, rubbish, dust, small pieces of paper</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rodents trapped in patient’s yard and neighborhood</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bank vole, Myodes glareolus</td>
<td>9</td>
<td>1 (11.1)</td>
<td>Male; antibodies had high avidity; mildly icteric.</td>
<td>0</td>
</tr>
<tr>
<td>Yellow-necked mouse, Apodemus flavicollis</td>
<td>14</td>
<td>1 (7.1)</td>
<td>Male; because of low titer, avidity was indeterminable.</td>
<td>0</td>
</tr>
<tr>
<td>Total rodents</td>
<td>23</td>
<td>2 (8.7)</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Rodents trapped elsewhere in southern Finland (distance from patient’s home)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mäntsälä (30 km)</td>
<td>33</td>
<td>7 (21.2)</td>
<td>Bank voles</td>
<td>NA</td>
</tr>
<tr>
<td>Riihimäki (50 km)</td>
<td>55</td>
<td>3 (5.5)</td>
<td>Bank voles, yellow-necked mouse</td>
<td>NA</td>
</tr>
<tr>
<td>Heinola (100 km)</td>
<td>48</td>
<td>41 (85.4)</td>
<td>Bank voles, field voles (Microtus agrestis)</td>
<td>NA</td>
</tr>
</tbody>
</table>

*HA, Hemagglutinin; NA, Not analyzed.
†Patient had cleaned the storehouse 1 week before onset.

Technical Appendix Figure. Patient’s facial image and a series of progressive cornea images after onset of disease. A) Facial image at 2 weeks. B) Cornea image at 2 weeks; C) at 2 months; D) at 3 months; E) at 5 months; F) at 7 months; G) at 15 months; H) at 30 months; I) at 4 years.