We report mi-
larvae can eas-
H. tarandi
is mainly active on warm
during migration in host tis-
s, were detected in 5 of the symp-
tomatic patients (2,3).
H. tarandi eggs take 4–7 days to
hatch, depending on the temperature of the hair layer (4); thus, patients 8 and 9 were treated soon after ovipo-
sition and were seronegative. Newly
hatched H. tarandi larvae can eas-
ily dry, so their chance of survival is
higher when they are close to scalp
skin. Eggs from patient 2 were initially
misidentified as head lice eggs but
were eventually identified as H. tan-
di by T.G. Jaenson (Uppsala). Public-
ished photographs of the H. tarandi
eggs alongside the eggs of head lice (5) helped identify H. tarandi eggs in patients 8 and 9. According to
those patients, H. tarandi eggs could
not be removed from the hair with a
lice comb. The H. tarandi fly is well
adapted to sub-Arctic climate; nearly
all reindeer were found to be infested in
some districts of northern Finland and
Norway (6). Reindeer habitats at-
tract tourists, mostly during summer.
H. tarandi is mainly active on warm
summer days; warm weather perhaps
does not encourage persons to cover
their heads, which may predispose for
oviposition. Also, persons moving
around probably attract more flies than
do those staying still, and strong wind,
rain, and temperatures <10°C–12°C
are thought to inhibit the warble fly’s
flight activity and oviposition (7).

Awareness of human infestation
by H. tarandi warble flies increased in
Sweden and Norway after news
media in Sweden described patient
2 (5; www.lakartidningen.se/engine.
php?articleId=14643). This publi-
cation helped in the recognition of
symptoms and in shortening diag-
nostic delay in patients 3–6, 8, and
9. Of the 3 cases for which diagnos-
sis was not delayed, patients 4 and
5 were children of a physician who
read our publication and recognized
the symptoms; patient 6, herself a
physician, also read the article (5).
Increased awareness, rather than in-
creased incidence, explains the emer-
gence of new cases. Nine of 12 cases
of proven H. tarandi myiasis found
in the literature occurred in persons
who had ophthalmomyiasis interna
(3,8,9); migratory dermal swellings,
the clinical signature of hypodermo-
sis, have been reported only in 1 case
(10). Such swellings occurred in all
the patients reported here, suggesting
that clinicians overlooked this find-
ing, possibly because of the overtak-
ing severity of eye complications and
the reporting of most previous cases
by ophthalmologists (3,8,9). Persons
who seek care for migratory dermal
swellings during August–December
should be asked about recent travel to
reindeer habitats.

For 3 patients with ophthalmo-
myiasis reported here, ophthalmolo-
gists initially had difficulty establish-
ing a diagnosis, raising the possibility
that some cases of “idiopathic” uveitis
from H. tarandi–endemic areas may
be caused by H. tarandi. Ophthalmom-
myiasis should be considered in cases
of unilateral uveitis, lens subluxation,
and suspicion of intraocular foreign
body (3,8,9). Eosinophilia might be
absent and should not be used to
guide treatment.

Boris Kan, Kjetil Åsbakk,
Kristian Fossen, Arne Nilssen,
Rosario Panadero,
and Domenico Otranto
Table. Myiasis caused by warble reindeer fly (Hypoderma tarandi), Scandinavia, 1991–2012

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Case-patient no.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Age, y/sex</td>
<td>8/M</td>
</tr>
<tr>
<td>Signs and symptoms</td>
<td>Enlarged occipital and retroauricular lymph nodes</td>
</tr>
<tr>
<td>Destination</td>
<td>Norway, extreme northeast: patient resides in Troms County where reindeer are occasionally seen</td>
</tr>
<tr>
<td>Observed reindeer</td>
<td>Yes</td>
</tr>
<tr>
<td>Migratory swellings, no.</td>
<td>Temple, 1</td>
</tr>
<tr>
<td>Fever</td>
<td>Yes</td>
</tr>
<tr>
<td>Eosinophilia (highest value)†</td>
<td>Yes</td>
</tr>
<tr>
<td>Other signs and symptoms</td>
<td>Uveitis, failure to gain weight</td>
</tr>
<tr>
<td>Diagnostic delay, d#</td>
<td>74</td>
</tr>
<tr>
<td>Drugs received</td>
<td>Ivermectin</td>
</tr>
<tr>
<td></td>
<td>Anthistamines</td>
</tr>
<tr>
<td></td>
<td>Oral steroids</td>
</tr>
<tr>
<td>Outcome</td>
<td>After eye surgery</td>
</tr>
</tbody>
</table>

*Patients 8 and 9 are not included in the table because myiasis did not develop in them.
†Patients 4 and 5 are siblings.
‡Patient 7 was discovered by the father of patients 4 and 5 among his acquaintances, suggesting the possibility of additional unreported cases in the population.
§In Jukkasjärvi (Sweden), the child had visited an enclosure where the reindeer were agitated because of swarms of flies.
¶Referent 0–0.5 × 10^9/L.
#Interval between date of first visit for myiasis-associated symptoms and date when treatment began.
**The diagnosis could not be confirmed, but her clinical picture and response to treatment were similar to those of other patients.
†Ivermectin was given first after eye surgery.

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DOI: http://dx.doi.org/10.3201/eid1905.130145
Azole-Resistant Aspergillus fumigatus, Iran

To the Editor: Aspergillus fumigatus causes a variety of diseases in humans. The drugs recommended for treatment of Aspergillus diseases are the mold-active azole antifungal drugs (1). However, a wide range of mutations in A. fumigatus confer azole resistance, which commonly involves modifications in the cyp51A gene (2), the target for azole antifungal drugs.

Azole resistance is thought to be selected for as a result of patient therapy or exposure toazole compounds in the environment; resistance in clinical A. fumigatus isolates has been increasingly reported in several European countries, Asia, and the United States (3–7). The most frequently reported resistance mechanism is a 34-bp tandem repeat (TR<sub>L98H</sub>) in combination with a substitution at codon 98 (TR<sub>L98H</sub>/L98H) (4); this mechanism is believed to have been selected for through environmental exposure to azole fungicides.

Because routine in vitro susceptibility testing of clinical Aspergillus isolates is not common in many centers worldwide, the prevalence of azole resistance might be underestimated. We investigated the prevalence of azole resistance in clinical A. fumigatus isolates stored for 6 years (2003–2009) at Tehran University Mycology Reference Centre and Islamic Azad University, Ardabil Branch, Iran.

We investigated 124 clinical A. fumigatus isolates obtained from patients with Aspergillus diseases (online Technical Appendix Table 1, wwwnc.cdc.gov/EID/article/19/5/13-0075-Techapp1.pdf). We conducted strain identification, in vitro antifungal susceptibility testing, and sequence-based analysis of the Cyp51A gene, as described (4). We performed microsatellite genotyping of all A. fumigatus isolates for which the MIC of itraconazole was ≥16 mg/L (8) by using a short tandem repeat A. fumigatus assay, and we compared the results with those reported for the Netherlands (20 isolates) and other European countries (24 isolates) (online Technical Appendix Figure).

The distribution of azole-resistant and wild-type A. fumigatus isolates examined in this study, according to year of isolation, is shown in online Technical Appendix Table 1. Of 124 A. fumigatus isolates, 4 grew on the wells containing itraconazole and voriconazole, indicating a multidrug-resistant phenotype. Of these resistant isolates, 3 were from patients with chronic pulmonary aspergillosis and 1 was from a patient with allergic bronchopulmonary aspergillosis (Table).

Sequence analysis of the CYP51A gene indicated the presence of TR<br>L98H in 3 isolates and no mutations in the other isolate (Table). The first TR<br>L98H isolate had been recovered in 2005, which is relatively early compared with reported isolations in other countries (online Technical Appendix Table 2). Microsatellite typing of 6 short tandem repeat loci demonstrated identical patterns for 2 of the 3 azole-resistant isolates from Iran, but the TR<br>L98H isolates from Iran did not cluster with those from the Netherlands and other European countries, indicating no close genetic relatedness (online Technical Appendix Figure).

The TR<br>L98H azole resistance mechanism was first described in 1998 in the Netherlands; since then, its presence in clinical and environmental A. fumigatus isolates in multiple European countries and recently in Asia has been increasingly reported (online Technical Appendix Table 2) (3–7). In the study reported here, prevalence of azole resistance in clinical A. fumigatus isolates obtained from patients in Iran was 3.2%; most isolates exhibited the TR<br>L98H resistance mechanism. The fact that the first TR<br>L98H isolate was found relatively early, in 2005, underscores the possibility that prevalence

References