



Figure. Comparison of transport for London absenteeism rates from influenza data to syndromic surveillance indicators of influenza-like illness rates, London, United Kingdom, 2009. A) National Pandemic Flu Service (NPFS); B) Royal College of General Practitioners (RCGP); and C) QSurveillance. Vertical black line indicates when the World Health Organization declared a pandemic on June 11, 2009. Source: Health Protection Agency, London, and Transport for London.

absenteeism was highly correlated with laboratory-confirmed influenza, and such information could be used to provide surveillance alerts up to 2 weeks before other traditional influenza surveillance data sources (3).

The use of workplace absenteeism data, particularly from large employers, has the potential for overcoming the major limitation of school-based absenteeism data in detecting outbreaks of ILI: the effects of school holidays and local planned school closures. Near real-time workplace absenteeism is an effective surveillance tool and should be more widely incorporated in influenza surveillance systems.

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Zoonotic Ascariasis, United Kingdom

To the Editor: *Ascaris lumbricoides/suum* is a complex of closely related enteric roundworms that mainly infect humans and pigs (1). Transmission occurs through ingestion of fecally excreted ova. *A. lumbricoides* worms usually infect humans, mainly in regions with poor sanitation, where the environment is contaminated with human feces. In industrialized countries, human ascariasis is uncommon and cases are generally believed to have been imported (2). By contrast, *A. suum* infection of pigs occurs worldwide; in the United Kingdom, 3.4%–6.5% of pigs at slaughter have evidence of infection (3). Sporadic zoonotic infection with *A. suum* in the industrialized world is described (4–6) but poorly quantified. We describe probable zoonotic transmission of *Ascaris* spp. roundworms in Cornwall, a rural county in southwestern England.

Incidence rates for ascariasis in Cornwall and the rest of England were calculated from local and national laboratory data. From 2004 through 2008, a total of 18 cases were

identified in Cornwall, and 314 from the rest of England were reported to the Health Protection Agency; annual rates were 0.87 and 0.12 cases per 100,000 population, respectively.

From 1995 through 2010, a total of 63 ascariasis cases were identified in Cornwall, and details of patient age, sex, and place of residence were collected. Patients from Cornwall were younger (mean age 22 years) than those from other parts of England (mean age 31 years), and the proportion of patients <5 years of age in Cornwall (35.5%) was greater than that in the rest of England (19.7%). Similar proportions (61% vs. 65%) of patients from Cornwall and England were female.

The possibility of zoonotic transmission in Cornwall was investigated by comparing risk factors for ascariasis and enterobiasis (caused by an enteric helminth that infects only humans). From 1995 through 2010, the laboratory in Cornwall identified 38 cases of *Enterobius* infection. Patient mean age was 24 years (range 1–95 years); 2 (5.7%) patients were <5 years of age and 23 (60.5%) were female. The following risk factors were considered for statistical analysis: age <5 years, female sex, and residence near pig herds. Residence was determined by comparing the postcodes of case-patients with postcodes of pig holdings registered with the Department of Environment, Food and Rural Affairs. The UK postal service allots a maximum of 80 households to a postcode. In rural areas like Cornwall, the number is much smaller. Consequently, sharing

a postcode with a pig holding implies proximity to pig herds. Of the 50 ascariasis patients with a Cornwall postcode, 11 (22%) shared that postcode with a pig holding. Of the 35 enterobiasis patients in Cornwall, only 2 (5.7%) shared a postcode with a pig holding.

Odds ratios were calculated for all 3 risk factors, and the Fisher exact test was used to determine their significance. We calculated p values by using 2-tailed models for age and sex and a 1-tailed model to test the association with residence near a pig holding (Table). Significant associations were found for age <5 years (odds ratio 6.42, $p = 0.0037$) and living near pigs (odds ratio 4.65, $p = 0.036$) but not for female sex.

Further evidence for zoonotic transmission comes from molecular analyses of DNA extracted from 11 *Ascaris* spp. worms recovered from patients in Cornwall. Results were compared by PCR-linked restriction fragment length polymorphism and sequence analysis with those from 35 reference worms from pigs in the United Kingdom, Denmark, Uganda, Guatemala, and the Philippines and from 20 worms from humans in Uganda, Tanzania, and Nepal. We used the PCR-linked restriction fragment length polymorphism method described by Nejsum et al. (5). Briefly, the ribosomal internal transcribed spacer region was amplified, and the products were digested with the restriction enzyme *Hae*III and separated into bands by agarose gel electrophoresis. All worms from humans and pigs in the

United Kingdom had 3- or 4-banded genotypes, typically found in worms from pigs (4,5,7). By contrast, a 2-banded genotype predominated in worms collected from humans living in *A. lumbricoides*-endemic areas. Similarly, sequence analysis, as described by Nejsum et al. (8), of amplified mitochondrial *cox1* genes using primers by Peng et al. (9) showed that all worms from humans in Cornwall clustered with worms from pigs (i.e., had pig-like DNA sequences).

Compared with the rest of the United Kingdom, incidence of human ascariasis is high in Cornwall, especially among children <5 years of age. Because of the retrospective nature of our study, we have little travel or clinical information for these case-patients. However, because such young case-patients would probably not travel much and because postcode data identified place of residence as a risk factor, the data suggest a focus of locally acquired *A. suum* infection in humans in Cornwall. Molecular evidence implicates pigs as the source. Further studies are needed, but if pigs are confirmed to be the source, control and prevention of this emerging infection will probably depend more on modifications of animal husbandry and fecal waste disposal rather than on human sanitation.

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Table. Risk factors for ascariasis versus enterobiasis in residents of Cornwall, United Kingdom, 1995–2010

Risk factor	Ascariasis, no. with risk factor/total no. cases*	Enterobiasis, no. with risk factor/total no. cases†	Odds ratio (95% confidence interval)	p value‡
Age <5 y	22/62	3/38	6.42 (1.77–23.30)	0.0037
Female sex	35/62	22/38	0.845 (0.372–1.920)	0.83
Pig holding within same postcode	11/50	2/35	4.65 (0.962–22.500)	0.036

*n = 63 patients. Postcode not available for 12 patients; 1 postcode was outside of Cornwall. Thus, only 50/63 patients were included in postcode analysis. Sex not indicated for 1 patient; age not indicated for 1 patient. Thus, only 62/63 patients were included in age and sex analyses.

†n = 38 patients. Postcode outside of Cornwall for 3 patients. Thus, only 35/38 patients were included in postcode analysis.

‡By Fisher exact test. **Boldface** indicates significance.

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Minority K65R Variants and Early Failure of Antiretroviral Therapy in HIV-1-infected Eritrean Immigrant

To the Editor: Genotypic drug resistance testing before initiation of first-line antiretroviral therapy (ART) is recommended to detect drug-resistant viruses and to avoid treatment failure caused by preexisting drug-resistant viruses (1). However, standard resistance testing cannot detect drug-resistant HIV-1 minority variants unless they represent 20%–25% of the population (2). Approximately 15% of those who underwent seroconversion in the acute phase in industrialized settings harbor drug-resistant HIV-1 minority

variants, while standard resistance testing did not detect drug-resistant viruses in those patients (3). We report the case of a treatment-naive HIV-1-infected patient with early treatment failure because of preexisting minority K65R-harboring HIV-1 variants.

A 32-year-old immigrant to Switzerland from Eritrea with a recently diagnosed HIV-1 subtype C infection was seen at University Hospital, Zurich. On the basis of the low CD4+ T-cell count of 69 cells/μL (15%) and high HIV-1 viral load of 980,000 copies/mL plasma, we started directly observed ART with tenofovir and emtricitabine plus nevirapine. Genotypic resistance testing showed no evidence of resistance. Within the first 4 weeks of ART, the viral load decreased to 540; however, 4 weeks later it increased to 15,000, and then 12 days later to 71,000 HIV-1 RNA copies/mL (Figure). Resistance testing at this time revealed the reverse transcriptase (RT) mutations K65R, K103N, and M184V, which confer resistance to all prescribed drugs. ART was changed to lamivudine/zidovudine, darunavir/ritonavir, and etravirine, and subsequently viremia decreased and remained undetectable.

We hypothesized that preexisting drug-resistant HIV-1 minority variants might have caused this early treatment failure. Thus, we performed clonal analysis of the RT gene before and during ART. At baseline, 17/222 clones (7.7%) carried the K65R mutation, synonymous to ≈51,000 HIV-1 RNA copies. Later, the K65R mutation was comprised in all clones. Further preexisting drug-resistance mutations have been detected in single, K65 wild-type, separate clones: K70R, V106A, and V108I. However, neither the K103N nor the M184V mutation was detected in any of those 222 clones, but both mutations were rapidly selected during early treatment failure.

Thus, the presence of the K65R mutation in a substantial fraction