evidence of active disease among these animals. However, pigs in porcine farms not deemed of high-health status are regularly colonized by \textit{S. suis} (3). Most cases of \textit{S. suis} disease in humans have been linked to accidental inoculation through skin injuries (3). The patient reported that in the days before his hospitalization, a pig died unexpectedly, and he removed it from the pen without using protective equipment such as gloves or safety glasses. However, there was no indication that this animal had died of a \textit{S. suis} infection. The patient also described transient worsening of his bilateral hearing loss during hospitalization. Hearing loss from \textit{S. suis} infection occurs frequently (9).

\textit{S. suis} zoonotic disease has emerged in Asia and occurs frequently in Europe among persons in close contact with pigs (3). In contrast, only 8 human \textit{S. suis} cases have been reported in Canada and the United States, which together are the second largest swine producers worldwide (3). This lower number of cases may be related to the lower virulence of \textit{S. suis} serotype 2 genotypes circulating in North America (ST25 and ST28) in comparison to serotype 2 genotypes circulating in Europe and Asia (ST1, ST7) (4,6,7). However, \textit{S. suis} infections may be underdiagnosed in North America. Our data and previous reports (10) show that the organism is sometimes misidentified as other \textit{α}-hemolytic streptococci by commercial identification systems. Here, the isolate was initially identified as viridans group \textit{Streptococcus}, and only the use of matrix-assisted laser desorption/ionization time-of-flight mass spectrometry at the reference laboratory permitted correct identification. While proper identification was unlikely to have led to a different treatment course in this case, our report underscores the need to increase awareness of \textit{S. suis} as a potential agent of human infections and serves as a reminder to routinely query patients about animal contact, particularly in areas with intensive pig farming operations.

Acknowledgments
We are grateful to the patient for consenting to publication of his clinical data. We thank Allison McGeer (Sinai Health System, Toronto) for critical reading of an earlier version of this manuscript. We also thank the staff of Public Health Ontario Genome Core for sequencing the genome of the isolate. We thank the staff of Public Health Ontario Toronto) for critical reading of an earlier version of this manuscript. We thank Allison McGeer (Sinai Health System, Toronto) for critical reading of an earlier version of this manuscript. We thank the staff of Public Health Ontario Genome Core for sequencing the genome of the isolate. We thank Allison McGeer (Sinai Health System, Toronto) for critical reading of an earlier version of this manuscript. We thank the staff of Public Health Ontario Genome Core for sequencing the genome of the isolate.

Dr. Gomez-Torres is an internist at Tillsonburg District Memorial Hospital. Her primary research interest is infectious diseases in hospital settings.

References

Address for correspondence: Nahuel Fittipaldi, Public Health Ontario Laboratory, 661 University Ave, Ste 17-100, Toronto, ON M5G 1M1, Canada; email: nahuel.fittipaldi@oahpp.ca

Moku Virus in Invasive Asian Hornets, Belgium, 2016

Mutien Garigliany, Bernard Taminiau, Noémie El Agrebi, Daniel Cadar, Gautier Gilliaux, Marie Hue, Daniel Desmecht, Georges Daube, Annick Linden, Frédéric Farnir, Michel De Proft, Claude Saegeerman

Author affiliations: Faculty of Veterinary Medicine, Liège, Belgium (M. Garigliany, B. Taminiau, N. El Agrebi, G. Gilliaux, M. Hue, D. Desmecht, G. Daube, A. Linden, F. Farnir, C. Saegeerman); Bernhard Nocht Institute for Tropical Medicine, Hamburg, Germany (D. Cadar); Walloon Agricultural Research Centre, Gembloux, Belgium (M. De Proft)

DOI: https://doi.org/10.3201/eid2312.171080

Emerging Infectious Diseases • www.cdc.gov/eid • Vol. 23, No. 12, December 2017

Page 2109
We report the detection of Moku virus in invasive Asian hornets (Vespa velutina nigrithorax) in Belgium. This constitutes an unexpected report of this flavivirus outside Hawaii, USA, where it was recently described in social wasps. Although virulence of Moku virus is unknown, its potential spread raises concern for European honeybee populations.

With their work estimated to have a global economic value of €153 billion, insects are critical pollinators of crops in agriculture, with the honeybee (Apis mellifera) being by far the major player in this process (1). Honeybee populations are decreasing dramatically worldwide, however, threatening food security. Environmental changes, pesticides, pathogens, and parasitic species are all recognized drivers of this decline (2). Among these, the Varroa mite (Varroa destructor) has been shown to have a critical effect on honeybee populations, both by its direct parasitic effects and through the transmission of pathogenic viruses such as deformed wing virus (2).

The Asian yellow-legged hornet (Vespa velutina nigrithorax), a natural predator of honeybees, has a native range spanning from India through China and as far as Indonesia (3). It is a particularly efficient invader because of its distinctive biology and behavior (4,5). The hornet was accidentally introduced from China into Europe, with sightings in France in 2004, and has rapidly spread to neighboring countries, including Belgium, since 2011 (6). In invaded areas, hornets’ feeding sites are primarily apiaries, which present an attractive, abundant, and defenseless prey source (5). V. velutina nigrithorax hornets not only contribute by hunting to the loss of honeybee colonies but also interact with the honeybees and can act as viral reservoirs, as V. destructor mites do, and infect the bees through spillover events (7,8). To explore the possibility of transmission of viruses from these hornets to honeybees, we performed a viral metagenomic analysis of Asian hornets collected in Belgium in 2016.

most viral sequences to phages (not represented) and viruses of the Partitiviridae and Paroviridae (Densosvirinae) families (online Technical Appendix Figure 1); however, a few reads pointed to a member of the Iflaviridae family, which contains such notable bee pathogens as deformed wing virus and slow bee paralysis virus (9). blastn alignment showed a positive match to Moku virus (9). Template-based assembly using Moku virus (GenBank accession no. KU645789) (9) permitted a near-full genome reconstruction from 1,215 matching reads out of 4,587,801. We used primer walking PCR and Sanger sequencing to fill the gaps in the genome (online Technical Appendix Figure 2).

The full viral genome sequence we obtained is 10,032 nt in length (GenBank accession no. MF346349) and has a mean nucleotide identity of 96.0% to the Hawaiian Moku virus strain (accession no. KU645789) (9), with both viruses showing an open reading frame of the same length (9,153 nt) sharing an amino acid identity of 99.0%. We performed an alignment to the full translated polyprotein amino acid sequence of representative iflaviruses available in GenBank using the Muscle aligner implemented in Geneious version 8.1.8 (Biomatters, Auckland, New Zealand). A maximum-likelihood phylogenetic analysis performed on the full-length polyprotein sequence yielded comparable results to that obtained on a conserved region of the RNA-dependent RNA polymerase (9), confirming the high identity of the Moku virus we obtained from the V. velutina nigrithorax hornet pools with the Hawaiian isolate of Moku virus, as well as its proximity to slow bee paralysis virus (Figure).

Our results show a large diversity of viruses in invasive Asian hornets collected in Belgium in 2016. Among these, we detected an iflavirus with high identity to the recently described Moku virus found in social wasps (Vespa pensylvanica), honeybees, and Varroa mites in Hawaii (9). Such a high nucleotide identity unequivocally places both strains in a single species. The potential pathogenicity of Moku virus for honeybees is currently unknown, but its relatively close relationship with the highly virulent slow bee paralysis virus warrants further studies (9). There is an urgent need to assess the presence of Moku virus in honeybees and Varroa mites in areas of Europe where the Asian hornet has become endemic, such as several regions in France. As highlighted by Mordecai et al. (9), the carriage of Moku virus in V. destructor mites in Hawaii is of great concern given the role played by this mite in the maintenance and transmission of viruses, including the deformed wing iflavirus, to honeybees. Furthermore, although Moku virus was shown to be highly dominant among viral species infecting V. pensylvanica wasps (9), suggesting that this species is a likely reservoir of the virus, we could not establish the same relationship for the Asian hornet V. velutina, in which Partitiviridae were much more abundant. It remains to be determined whether Moku virus is a virus of Vespuilidae or, more likely given the relatively low number of reads detected, could have been picked up by these hornets from their prey, such as honeybees. Further studies are needed to establish the origin, host range, and transmission route of Moku virus; its virulence; and the risks it may represent for European honeybee populations.

Acknowledgments
We thank the Fundamental and Applied Research for Animal and Health Center, Liège, Belgium, for the access to its metagenomics platform, and the Walloon Agricultural Research Centre, Gembloux, Belgium, for the access to historical specimens of the Asian hornet Vespa velutina.

Dr. Garigliany is a professor of pathology at the Veterinary Faculty of the University of Liège, Belgium. His research activities focus on the biology of viruses and related host-pathogen interactions.

References
Angiostrongylus cantonensis DNA in Cerebrospinal Fluid of Persons with Eosinophilic Meningitis, Laos

Damien K.Y. Ming, Sayaphet Rattanavong, Tehmina Bharucha, Onanong Sengvilaiapaseuth, Audrey Dubot-Pérès, Paul N. Newton, Matthew T. Robinson

Author affiliations: Imperial College London, London, UK (D.K.Y. Ming); Lao-Oxford-Mahosot Hospital Wellcome Trust Research Unit, Vientiane, Laos (D.K.Y. Ming, S. Rattanavong, T. Bharucha, O. Sengvilaiapaseuth, A. Dubot-Pérès, P.N. Newton, M.T. Robinson); University College London, London (T. Bharucha); Aix-Marseille Université, Marseille, France (A. Dubot-Pérès); University of Oxford, Oxford, UK (P.N. Newton, M.T. Robinson)

DOI: https://doi.org/10.3201/eid2312.171107

Definitive identification of Angiostrongylus cantonensis parasites from clinical specimens is difficult. As a result, regional epidemiology and burden are poorly characterized. To ascertain presence of this parasite in patients with eosinophilic meningitis in Laos, we tested samples from a cohort of 1,065 patients suspected of having CNS infection at Mahosot Hospital, Vientiane, Laos, during 2003–2013 by Giemsa staining and identified 54 CSF samples containing ≥10% eosinophils. Of these, 36 samples from 35 patients were available for this study (1 patient underwent lumbar puncture twice) and were tested by conventional and quantitative PCR (cPCR and qPCR). From the same cohort, we also performed qPCR testing on another 50 CSF samples with 1%–9% eosinophils.

DNA was extracted from 200 µL of CSF by using a QIAamp DNA Mini Kit (QIAGEN, Hilden, Germany) and eluted in 30 µL buffer. We ran the extract in parallel with positive samples (A. cantonensis DNA from experimentally infected rats, University of Sydney, Sydney, Australia) and negative controls by using published cPCR (7) and qPCR (8) protocols. We used Platinum PCR SuperMix (Thermo Fisher, Waltham, MA, USA) and performed assays on a Bio-Rad CFX96 (Bio-Rad, Watford, UK).

Among patients with CSF eosinophilia ≥10%, male patients predominated, although sex of patients did not differ significantly among patients with or without CSF eosinophilia (Table). Of the 36 CSF samples that contained ≥10% eosinophils, all were negative by cPCR, but 4 (11.1%) were positive for A. cantonensis DNA by qPCR; median quantification cycle was 35.9 (range 34.1–37.4). Sensitivity of qPCR was apparently higher than that of cPCR. The median duration of illness for patients with positive qPCR was 4 (range 0–10) days. Of 3 patients with a positive qPCR, 2 reported that they had eaten raw snails in the previous month.

Results from 2 CSF samples obtained from the same patient and tested by qPCR were discordant; CSF obtained after 7 days of illness was negative for A. cantonensis DNA, but a sample obtained on day 13 was positive (quantification cycle 34.1). This finding is consistent with previous observations (8), and it is plausible that during the acute stages of infection, insufficient nucleic material is present for detection. Although lumbar puncture is invasive, a high clinical suspicion of angiostrongyliasis in the context of a negative qPCR may therefore warrant a repeated lumbar puncture. All positive samples had CSF eosinophil proportions >40%, and all samples containing a 1%–9% eosinophil proportion tested negative by qPCR, supporting the conventional cutoff of a CSF eosinophilia ≥10% in the diagnosis of CNS angiostrongyliasis.