

in Lanzhou, China (8). Variation may be due to different geographic and age distributions of the virus. Another study reported that frequencies of WUPyV in URTIs (6.7%) and LRTIs cases (7.1%) were comparable (10). However, we found the incidence of WUPyV in patients with LRTIs (16.1%) was higher than in patients with URTIs (4.4%). Among WUPyV-infected patients with LRTIs, 71.4% were <1 year of age, which was comparable to populations investigated in other studies (2,3,6). Although ≈60% of outpatients with URTIs were >5 years of age, none was WUPyV positive. This finding suggests WUPyV may play a major role in young children, especially infants, with LRTIs.

Most WUPyV infection has been detected during later winter and early spring (2,4,5) although other research showed no seasonal distribution (6). We found 2 peaks, in April and December 2008 (L. Xiaoyan et al., unpub. data). We also detected 1 WUPyV-infected case in September 2008, which suggests WUPyV could also occur in summer months.

Frequency of WUPyV co-infection with other pathogens varied from 42.1% to 79.7% (4–6). Although we showed a co-infection rate of 71.4%, there were 8 (28.6%) of 28 patients with respiratory illness in whose specimens we detected only 1 virus, WUPyV. No WUPyV was detected in samples from 43 control patients, whereas in patients with LRTIs and URTIs, infection rates were 16.1% and 4.4%, respectively. These findings suggest WUPyV may be a potential pathogenic agent in children with acute respiratory tract infections. More comprehensive case–control investigations are needed to determine the association of WUPyV infections with respiratory diseases.

This work was supported by Tianjin Municipal Science and Technology Commission (grant no. 07SYSYSF05100).

**Xiaoyan Li, Jinying Chen,  
Mei Kong, Xu Su, Ming Zou,  
Hua Zhang, and Yumin Han**

Author affiliations: Tianjin Centers for Disease Control and Prevention, Tianjin, People's Republic of China (X. Li, M. Kong, X. Su, M. Zou); Tianjin Medical University, Tianjin (X. Li, J. Chen); Tianjin Children's Hospital, Tianjin (H. Zhang); and Tianjin Xi'anhuo Hospital, Tianjin (Y. Han)

**References**

1. Gaynor AM, Nissen MD, Whiley DM, Mackay IM, Lambert SB, Wu G, et al. Identification of a novel polyomavirus from patients with acute respiratory tract infections. *PLoS Pathog.* 2007;3:e64. DOI: 10.1371/journal.ppat.0030064
2. Le BM, Demertzis LM, Wu G, Tibbets RJ, Buller R, Arens MQ, et al. Clinical and epidemiologic characterization of WU polyomavirus infection, St. Louis, Missouri. *Emerg Infect Dis.* 2007;13:1936–8.
3. Wattier RL, Vázquez M, Weibel C, Shapiro ED, Ferguson D, Landry ML, et al. Role of human polyomaviruses in respiratory tract disease in young children. *Emerg Infect Dis.* 2008;14:1766–8. DOI: 10.3201/eid1411.080394
4. Neske F, Blessing K, Ullrich F, Prötzel A, Wolfgang Kreth H, Weissbrich B. WU polyomavirus infection in children, Germany. *Emerg Infect Dis.* 2008;14:680–1. DOI: 10.3201/eid1404.071325
5. Bialasiewicz S, Whiley DM, Lambert SB, Jacob K, Bletchly C, Wang D, et al. Presence of the newly discovered human polyomaviruses KI and WU in Australian patients with acute respiratory tract infection. *J Clin Virol.* 2008;41:63–8. DOI: 10.1016/j.jcv.2007.11.001
6. Payungporn S, Chieochansin T, Thongmee C, Samransamruajkit R, Theamboolers A, Poovorawan Y. Prevalence and molecular characterization of WU/KI polyomaviruses isolated from pediatric patients with respiratory disease in Thailand. *Virus Res.* 2008;135:230–6. DOI: 10.1016/j.virusres.2008.03.018
7. Dalianis T, Ramqvist T, Andreasson K, Kean JM, Garcea RLKI. WU and Merkel cell polyomaviruses: a new era for human polyomavirus research. *Semin Cancer Biol.* 2009;19:270–5. DOI: 10.1016/j.semcancer.2009.04.001
8. Yuan XH, Jin Y, Xie ZP, Gao HC, Xu ZQ, Zheng LS, et al. Prevalence of human KI and WU polyomaviruses in children with acute respiratory tract infection in China. *J Clin Microbiol.* 2008;46:3522–5. DOI: 10.1128/JCM.01301-08
9. Allander T, Tammi MT, Eriksson M, Bjerkner A, Tiveljung-Lindell A, Andersson B. Cloning of a human parvovirus by molecular screening of respiratory tract samples. *Proc Natl Acad Sci U S A.* 2005;102:12891–6. DOI: 10.1073/pnas.0504666102
10. Norja P, Ubillos I, Templeton K, Simmonds P. No evidence for an association between infections with WU and KI polyomaviruses and respiratory disease. *J Clin Virol.* 2007;40:307–11. DOI: 10.1016/j.jcv.2007.09.008

Address for correspondence: Xiaoyan Li, Tianjin Centers for Disease Control and Prevention, Institute of Microbiology, No. 76 Hualong St, Hedong District, Tianjin 300011, People's Republic of China; email: xiaoyanli1291@163.com

## Intestinal Capillariasis, Western Mindanao, the Philippines

**To the Editor:** Capillariasis is caused by the foodborne nematode *Capillaria philippinensis*. Infection causes severe diarrhea and protein loss resulting in dehydration, cachexia, and eventually death. Infected patients may also have borborygmi, abdominal pain, weight loss, anorexia, vomiting, and bipedal edema (1).

*C. philippinensis* was first reported in 1963 in Bacarra, Ilocos Norte Province in the northern Philippines (2). Since then, additional endemic foci of *C. philippinensis* have been identified. The most recent focus is in Monkayo, Compostela Valley, in the southern Philippines (3). In the past several years, suspected unconfirmed cases have been reported from Zamboanga del Norte Province in western Mindanao. In 1999, an epidemic of gastroenteritis in Piñan Municipality was reported; it resulted in 42 deaths. The schistosomiasis team of the De-

partment of Health Regional Office conducted stool examinations and suspected the presence of *Capillaria ova* in symptomatic patients (4). In November 2007, several deaths caused by chronic diarrhea were reported in Siayan Municipality. These deaths were attributed to capillariasis, but their cause was never confirmed (5).

In February 2008, we obtained 205 stool specimens from residents of Katipunan who had a history of diarrhea of >2 weeks duration and abdominal disturbance. These samples were processed by using the formalin–ether concentration technique (6) and examined by expert microscopists. One hundred fifty-one (73.3%) persons were infected with  $\geq 1$  organism; 67 (32.5%) had 1 parasitic infection and 84 (40.8%) had multiple parasitic infections. Ninety-one (44.2%) persons had  $\geq 1$  soil-transmitted helminth infection, and 93 (45.2%) had  $\geq 1$  protozoan infection. Ten (4.9%) persons were confirmed to have *Capillaria* infections. The distribution of organisms observed is shown in the Table.

Among the 10 persons who had capillariasis, 8 were from Barangay (smallest administrative region) Matam, 1 from Barangay Dabiak, and 1 from Barangay Carupay, a nearby barangay. Six cases were in male patients and 4 were in female patients. Ages of infected persons ranged from 5 to 54 years (mean 29.2 years, SD 17.1 years). Three of the reported case-patients (a 5-year-old boy, an 8-year-old boy, and a 48-year-old woman) were from the same household.

A total of 24 persons in Katipunan were interviewed regarding history of capillariasis and their eating habits. Fourteen residents reported having eaten kinilaw (raw freshwater fish soaked in vinegar and garnished with salt, ginger, and lime). Seven of the persons interviewed had a diagnosis of capillariasis, and 6 had  $\geq 1$  relative with a diagnosis of capillariasis. All of the previously diagnosed case-patients were treated with albendazole (400 mg tablets). Most patients were instructed to take 1 tablet 1 $\times$ /day for 20 days; others were instructed to take 1 tablet 2 $\times$ /day for 5 or 10 days.

The drug of choice for treating patients with capillariasis is mebendazole, 200 mg 2 $\times$ /day for 20–30 days. An alternative treatment is albendazole, 400 mg 1 $\times$ /day for 10 days (7,8). Variations in the treatment regimen used for patients with capillariasis at the study site suggest a need to train health professionals on the diagnosis, treatment, and follow-up of cases, and on disease prevention and control. Guidelines on proper laboratory techniques for diagnosis of capillariasis; treatment protocols and supportive measures; and protocols for detection, follow-up, and treatment for relapse cases must be developed.

Rates of infection with protozoans and soil-transmitted helminths at the study site are high, which indicate fecal contamination of food and water. A review of records from the Katipunan rural health unit indicated that 76% of households in this municipality have access to toilets. Only 11% of house-

holds have water connections (level III). Fifty-seven percent of households have access to communal faucets (level II), and 31% have access only to rivers or springs (9). Therefore, increased access to toilets and safe water is needed. Local ordinances concerning ownership and use of toilets must be strictly enforced, and evaluation and rehabilitation of existing toilet and water systems must be conducted.

In spite of efforts concerning information, education, and communication on capillariasis, many residents continue to eat raw or poorly cooked freshwater fish. Concurrent infection among household members, including those in younger age groups, was observed in this study. These findings result from the fact that consumption of kinilaw has become widely accepted and is consumed as a viand (choice food) by families. Thus, information, education, and communication campaigns must be intensified. A promising approach is through collaboration with other agencies. For example, the Department of Education in the Philippines may become involved in dissemination of information on capillariasis to students and in early detection and treatment of infected school children.

#### Acknowledgments

We thank the National Center for Disease Prevention and Control, the National Epidemiological Center, and the Center for Health Development for Western Mindanao of the Department of Health, the local government units of the province of Zamboanga del Norte, and the municipality of Katipunan for technical assistance and logistics support; Johnson and Johnson Philippines, Inc., for providing partial support for conducting field work; and Joanne Ramirez and Edward Castelo for assistance with the field work.

**Vicente Y. Belizario Jr,  
Francis Isidore G. Totañes,  
Winifreda U. de Leon,  
Julius R. Migrño Jr,  
and Lino Y. Macasaet**

Table. Parasites detected in Katipunan, Zamboanga del Norte, the Philippines, February 2008\*

Parasite	No. (%)
<i>Trichuris trichiura</i>	64 (31.1)
<i>Entamoeba coli</i>	49 (23.8)
<i>Ascaris lumbricoides</i>	46 (22.3)
<i>Endolimax nana</i>	14 (19.9)
Hookworm	34 (16.5)
<i>Blastocystis hominis</i>	21 (10.2)
<i>Giardia lamblia</i>	19 (0.2)
<i>Entamoeba histolytica</i>	14 (6.8)
<i>Capillaria philippinensis</i>	10 (4.9)

\*A total of 205 parasites were detected by using the formalin–ether concentration technique.

Author affiliations: University of the Philippines–National Institutes of Health, Manila, the Philippines (V.Y. Belizario Jr, F.I.G. Totañes, J.R. Migrño Jr); University of the Philippines College of Public Health, Manila (W.U. de Leon); and National Center for Disease Prevention and Control, Manila (L.Y. Macasaet)

DOI: 10.3201/eid1604.080483

## References

1. Cross J, Belizario VY Jr, Capillariasis. In: Murrel KD, Fried B, editors. Food-borne parasitic zoonoses, fish and plant-borne parasites. Vol. 11. In: Black S, Seed RJ, editors. World class parasites. New York: Springer Science and Business Media, LLC; 2007. p. 209–32.
2. Chitwood MB, Velasquez C, Salazar NG. *Capillaria philippinensis* (Nematoda: Trichinellida) from intestine of man in the Philippines. *J Parasitol.* 1968;54:368–71. DOI: 10.2307/3276953
3. Belizario VY Jr, de Leon WU, Esparar DG, Galang JM, Fantone J, Verdadero C. Compostela Valley: a new endemic focus for *Capillariasis philippinensis*. *Southeast Asian J Trop Med Public Health.* 2000;31:478–81.
4. Icao C. Capillariasis report. Interagency Conference on Capillariasis in Zamboanga del Norte. 2008 Feb 21. Dipolog City (the Philippines): Zamboanga del Norte Provincial Health Office; 2008.
5. Alipala J. Philippine Daily Inquirer. 70 dead in parasite infection; whole Zambo village afflicted [cited 2008 Feb 27]. [http://services.inquirer.net/print/print.php?article\\_id=103456](http://services.inquirer.net/print/print.php?article_id=103456)
6. World Health Organization. Training manual on the diagnosis of intestinal parasites; 2004. Geneva: The Organization [cited 2008 Mar 7]. [http://www.who.int/worm-control/documents/benchaid/en/training-manual\\_sip98-2.pdf](http://www.who.int/worm-control/documents/benchaid/en/training-manual_sip98-2.pdf)
7. Abramowicz M. Drugs for parasitic infections. *The Medical Letter.* 2004;46:e1–e12.
8. World Health Organization. WHO model prescribing information: drugs used in parasitic diseases, 2nd ed. Geneva: The Organization; 1995.
9. Katipunan Rural Health Unit. Annual report for 2007. Katipunan, Zamboanga del Norte (the Philippines): The Unit; 2007.

Address for correspondence: Vicente Y. Belizario Jr, University of the Philippines–National Institutes of Health, Pedro Gil St, Ermita 1000 Manila, the Philippines; email: vbelizar@yahoo.com

## Buruli Ulcer Lesions in HIV-Positive Patient

**To the Editor:** *Mycobacterium ulcerans* disease (Buruli ulcer) is a neglected and emerging tropical disease (1). It often leads to extensive destruction of skin and soft tissue with the formation of large ulcers (2). In 2004, the World Health Organization (WHO) recommended the combination treatment of rifampin/streptomycin for patients with this disease (3). According to WHO, development of new antimicrobial drug treatment is one of the major advances since the establishment of the Global Buruli Ulcer Initiative (1). Treatment with rifampin/streptomycin for  $\geq 4$  weeks can inhibit the growth of *M. ulcerans* in preulcerative lesions (4). In other patients, despite 4 weeks of treatment, lesions may deteriorate. Whether this treatment is less efficacious in persons with HIV infection is unknown.

In August 2008, a 35-year-old man was referred to the Medical Centre of the Democratic Republic of Congo for assessment of chronic ulcers. Lesions had appeared 12 months earlier when the patient was living in Kafufu/Luremo, a new focus of Buruli ulcer in Angola (5). Tissue specimens were subjected to Ziehl-Neelsen staining, culture, and PCR. All results were positive for *M. ulcerans*. Histopathologic analysis of formalin-fixed tissue confirmed the diagnosis of active Buruli ulcer. We treated the patient with a combination of rifampin (10 mg/kg/day, orally) and streptomycin (15 mg/kg/day, by intramuscular injection). Wound dressings containing an aqueous solution of chloramine/metronidazole/nitrofurantoin were changed daily (6). For logistic reasons, surgery (large excision) under general anesthesia was not possible.

Characteristics of the patient are shown in the online Technical Appendix ([www.cdc.gov/EID/content/16/4/](http://www.cdc.gov/EID/content/16/4/)

738-Techapp.pdf). At the start of treatment, the patient had a large ulcer on the right leg and thigh, a nodule 2 cm in diameter on the left thigh, and a plaque 8 cm in diameter on the left thigh. After 2 weeks of treatment, the size of the large ulcer had increased. After 4 weeks, the nodule became an ulcer 6 cm in diameter, and the plaque became a large ulcer 15 cm in diameter with a satellite ulcer 2 cm in diameter. After 8 weeks, we observed enlargement of all lesions and the appearance of an ulcer on the left wrist. Treatment was continued for an additional 4 weeks (total 12 weeks). Radiologic investigation did not disclose any bone destruction.

The patient was positive for HIV by the Determine HIV-1/2 test (Abbott Laboratories, Dainabot Co. Ltd., Tokyo, Japan), the Uni-Gold HIV test (Trinity Biotech PLC, Bray, Ireland), and the Genie II HIV-1/HIV-2 test (Bio-Rad, Marnes-la-Coquette, France). Results of PCR for *M. ulcerans* and Ziehl-Neelsen staining were positive for all specimens obtained during the 8 weeks of initial treatment. The patient died 2 weeks after treatment ended, just when antiretroviral treatment had been scheduled to begin.

Although the patient did not respond clinically to treatment with rifampin/streptomycin, whether the treatment also failed microbiologically is more difficult to prove. Results of PCRs performed during treatment remained positive. However, PCR does not differentiate between living and dead *M. ulcerans* bacteria. Therefore, our positive results suggest, but do not prove, treatment failure. The positive culture after 2 weeks of treatment also suggests treatment failure but cultures obtained at 4 and 8 weeks were contaminated. However, culturing *M. ulcerans* bacteria is difficult, especially if samples must be transported (7).

Patients with Buruli ulcer may also be infected with HIV. In a study conducted during January 2002–Au-