
To the Editor: African swine fever (ASF) is a highly contagious and deadly hemorrhagic disease of domestic pigs caused by African swine fever virus (ASFV), a double-strand DNA virus of the family Asfarviridae and genus Asfivirus (1). Twenty-two ASFV genotypes (I–XXII) have been identified on the basis of nucleotide sequencing of the variable 3′-end of the B646L gene encoding the major capsid protein p72 (2,3).

Historically, all ASFV p72 genotypes have been circulating in eastern and southern Africa, and genotype I has been circulating in Europe, South America, the Caribbean, and western Africa (2,3). Spread of ASFV beyond traditional geographic boundaries occurred with incursion of p72 genotype II into the Republic of Georgia and its subsequent spread into Armenia, Azerbaijan, and Russia (4,5) and incursion of genotype IX into western Africa (6). ASFV circulating in Tanzania has p72 genotypes X, XV, and XVI (7–10). We describe incursion and persistent circulation in Tanzania of a highly virulent p72 genotype II ASFV that is identical to the Georgia 2007/1 isolate in the 3′-end of the B646L gene.

An outbreak of ASF in domestic pigs occurred in November 2010 in the Kyela District of the Mbeya region in southwestern Tanzania, which coincided with another outbreak in a neighboring district of Karonga in northern Malawi (Figure, panel A). ASF continued to spread from Mbeya and ultimately reached the neighboring region of Iringa (Ludewa District) in February 2011 through feeding of pigs with swill from Mbeya. By March 2011, ASF had spread to Chunya, Ileje, Mbarali, Rungwe, and Tukuyu districts within Mbeya. The disease spread within the region because of the lack of zoosanitary measures and illegal movement of animals despite the quarantine in place. An outbreak on 1 farm in the Temeko District of the Dar es Salaam region in eastern Tanzania occurred in March 2011 after a farmer obtained pig stock from Mbeya. No further spread of the disease in Dar es Salaam was observed after early diagnosis, removal of affected pigs, and zoosanitary measures.

In October 2011, the disease spread to the Sumbawanga District of the Rukwa region through feeding of swill and illegal movement of animals. ASF was reported in February 2012 in Ifakara in the Kilombero District in the Morogoro region, and in July 2012 in the Kilosa District within this region. The disease spread into Kilombero District after 1 farmer purchased pigs for stock from the Iringa region. As of July 2012, ASF was reported again in the Mbeya and Iringa regions, from which it had been eliminated. This unique ASF outbreak in Tanzania persistently circulated for more than a year; previous outbreaks have been sporadic and resolved after shorter durations (8–10).

Mortality rates of 100% caused by ASF were recorded in domestic pigs of all ages in all outbreak areas. Affected pigs showed pyrexia and anorexia, dragged their hind legs, and then showed recumbence. In addition, affected animals had severe cutaneous hemorrhages, especially on medial and lateral sides of the pinna, forelimbs above the carpal joint, facial region, scrotum, and mammary glands (Figure, panels B and C). Postmortem lesions included darkening and enlargement of the spleen, severe hemorrhages of mesenteric and gastrohepatic lymph nodes, and hemorrhagic enteritis (Figure, panels D–G).

DNA was extracted from spleens of animals that either died of the disease or were killed at slaughterhouses during 2010–2012. The variable 3′-
end of the B646L (p72) gene was amplified by using p72U/p72D primers (2) and subjected to automated dideoxynucleotide cycle sequencing by using Big Dye Terminator Cycle Sequencing Kit Version 3.1 (Applied Biosystems, Foster City, CA, USA) in a 24-capillary DNA sequencer (Genetic Analyzer 3500 xL; Applied Biosystems).

All obtained ASFV p72 nucleotide sequences (GenBank accession nos. JX391987 [TAN/10/Kyela], JX391988 [TAN/10/Tukuyu], JX391989 [TAN/11/Chunya], JX391990 [TAN/11/Ludewa], JX391991 [TAN/11/Temeke], JX391992 [TAN/12/Ifakara]) were 100% identical. BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi) analysis of 2010–2012 ASFV p72 nucleotide sequences from Tanzania in GenBank showed 100% nucleotide identity with the Georgia 2007/1 ASFV isolate (GenBank accession no. FR682468) at nt positions 103,594–104,070.

The Georgia 2007/1 ASFV isolate was detected in Georgia in 2007 and has caused ASF outbreaks in Armenia, Azerbaijan, and Russia (5). This ASFV isolate belongs to p72 genotype II (5) and clusters with ASFV isolates from Mozambique, Zambia, Madagascar, Mauritius, and Georgia (2,3,7). Although the 2010–2012 outbreak in Tanzania coincided with the outbreak in Malawi in 2010, no ASFV belonging to p72 genotype II has been described in Malawi. ASFV isolates from the 2010 outbreak in Malawi should be sequenced to establish their relatedness to ASFV isolates from the 2010–2012 outbreak in Tanzania and determine an epidemiologic link between these outbreaks. Incursion and persistent circulation of a highly virulent p72 genotype II ASFV identical to the Georgia 2007/1 isolate has implications for transboundary spread of ASF.

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**Controlling Highly Pathogenic Avian Influenza, Bangladesh**

**To the Editor:** Highly pathogenic avian influenza (HPAI) A(H5N1) virus is a deadly zoonotic pathogen. Since 2003, HPAI infections have been reported in millions of poultry and wild birds from 63 countries (1) and in 598 humans, among whom there have been 352 reported deaths in 15 countries (2). HPAI (H5N1) virus is endemic in Bangladesh, and the first outbreak occurred in March 2007. Since then, the virus has spread to 49 of 64 districts in Bangladesh, and samples from 536 farms have tested positive for the virus. Bangladesh now ranks among countries worldwide with the highest reported number of HPAI outbreaks (1). Intermittent outbreaks in Bangladesh and clusters of disease across the border in northeastern India are dramatic reminders that the emergence of new, mutant viruses in developing countries could lead to a pandemic among humans. Six cases of nonfatal HPAI (H5N1) infection have been reported in Bangladesh (2). Live bird markets that are in poor physical condition and that lack or have poor biosecurity are probable sources of HPAI transmission to humans and for bird-to-bird transmission (3–5).

In 2008, a global project of the United States Agency for International Development, Stamping Out Pandemic and Avian Influenza (STOP AI), was initiated in Bangladesh. The project began with biosecurity training for veterinarians and livestock science graduates on some large-scale commercial farms. The local STOP AI office was established in Dhaka, the capital of Bangladesh, in February 2009, and the organization managed the project through its completion in September 2010 (online Technical Appendix Figure 1, wwwnc.cdc.gov/EID/pdfs/12-0635-Techapp.pdf).

STOP AI initially organized 7 highly successful live bird market biosecurity training programs in 5 geographic divisions of Bangladesh; later, STOP AI piloted cleaning and disinfection activities in 2 live bird markets, Mohammadmurar and Kaptan Bazaar, in Dhaka by working closely with the United Nations’ Food and Agriculture Organization. The Food and Agriculture Organization subsequently conducted cleaning and disinfection activities in 24 other markets within Dhaka and other districts in Bangladesh.

We focused on understanding the inter-relationships among household poultry producers, commercial farmers, suppliers, transporters, processors, and consumers that facilitate the process of producing and moving poultry, i.e., the entire poultry value chain (PVC). We describe how improved biosecurity on poultry farms and hygienic standards in live bird markets can reduce HPAI outbreaks. In resource-limited countries, like Bangladesh, these improvements can be made through training, technical support, financial assistance for infrastructure renovations, and incentive-driven trust-building between service providers and the population.