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human activities may correlate with the probability of pathogen introduction through introduced species (6), fomites, or other sources of infectious particles, with the likelihood of introduction higher for ranavirus. Certainly, both pathogens are presumed to be vectored in association with human activities (7,8), but B. dendrobatidis exhibits a greater host and geographic range and thus should exhibit greater prevalence if humans were mediating introduction across the range of our study. Second, human activities such as construction and industry, may directly or indirectly influence the basic reproductive number,  $R_0$ , of ranavirus to a greater extent than for that of B. dendrobatidis. Although ranavirus does exhibit optimal environmental ranges for replication and infection, the virulence of B. dendrobatidis can be directly influenced by the environment (2). Furthermore, infection by B. dendrobatidis occurs through a freeliving stage; ranavirus is more likely transmitted through direct contact, which suggests that B. dendrobatidis would be more sensitive to environmental factors. Last, human activities may influence host ability to mediate immune responses that have the capability to prevent infection. Evidence exists that amphibian host responses to ranavirus are predominantly acquired (9); those for B. dendrobatidis may be more innate and less prone to environmental manipulation (10). Although the observed correlation should be further tested and the disturbance index should be refined, we believe our observed pattern may reflect the influence of human activities and habitat modification in the dispersal of infectious diseases. With increasing evidence pointing towards the role of emerging infectious diseases in the decline of amphibian populations, management plans should therefore account for the indirect effects related to human activities.

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## Valerie St-Amour,\* Wai M. Wong,† Trenton W.J. Garner,† and David Lesbarrères\*

\*Laurentian University, Sudbury, Ontario, Canada; and †Institute of Zoology, London, UK

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Address for correspondence: David Lesbarrères, Biology Department, Laurentian University, Ramsey Lake Rd, Sudbury, Ontario P3B 3C8, Canada; email: dlesbarreres@laurentian.ca

## Avian Influenza Virus (H5N1) Mortality Surveillance

To the Editor: The highly pathogenic strain of avian influenza virus subtype H5N1 presents a major challenge to global public health systems. Currently, influenza (H5N1) infection is a zoonosis with a 60% case-fatality rate for affected persons over 3 continents; the virus could mutate to become directly transmissible among humans (1). This potential for pandemic transmission must be reduced through early detection of transmission foci, followed by rapid implementation of control measures (2). In the following analysis, we demonstrate that single carcasses of birds, mostly found by members of the public, were the primary indicators for avian influenza virus activity in Sweden and Denmark in 2006.

Influenza virus (H5N1) is amplified by commercial and backyard poultry and free-ranging birds. Whether captive birds (e.g., poultry) or wild birds are responsible for the spread of the virus remains a matter of debate (3). Initial spread from Southeast Asia before 2005 was likely the result of transport of infected poultry because the spread was not easily explained by natural bird movements (4,5). However, its spread to Western Europe in late 2005 could be explained by weatherinduced migration of waterfowl after a freeze in Eastern Europe (6,7). Since spreading to Sweden and Denmark in early 2006, the virus has been detected there in dead birds of numerous species (Table). Detections in carcasses of primarily free-ranging birds have become the principal means of tracking spread of the virus in Europe.

To better understand how avian mortality surveillance could be refined for monitoring the spread of influenza virus (H5N1), we analyzed the weekly official reports of such detections in Sweden and Denmark in 2006 (8). Virus surveillance in both countries includes both active cloacal swabbing of free-ranging wild birds and passive collection of tracheal swabs from bird carcasses. For the analysis, all carcasses of a single species collected on 1 day within a single locality constituted 1 record. For each record, we evaluated whether the carcasses were reported by a member of the public versus a civil servant, the number of carcasses tested, and the number of positive detections.

Our analysis evaluated 44 records; a total of 70 birds, of 14 species, tested positive for the virus in 22 localities of Sweden and Denmark. Almost all of these records ( $n\geq40$ , 91%) referred to dead birds found by members of the public rather than civil servants. A smaller portion than expected were Anseriformes (i.e., ducks, geese, or swans; n = 32, 73%). Other orders of birds represented were Falconiformes (hawks, falcons; n = 8, 18%), Strigiformes (owls; n = 2, 5%), Podicepidiformes (grebes; n = 1, 2%), and Charadriiformes (gulls, shorebirds; n = 1, 2%). In addition, birds of other orders tested positive in Denmark but were excluded from the analysis for lack of supporting data. Most (75%) of the records referred to singleton carcasses; the remaining 25% represented multiple detections, ranging from 2 to 9 individual birds of a single species. A majority (73%) of influenza virus (H5N1)-positive localities hosted solely singleton carcasses, whereas the other 27% hosted multiple dead birds. No virus activity was detected through active free-ranging bird surveillance, even though 9,260 live birds were captured and sampled during 2006 in Sweden and Denmark.

The pattern of virus activity observed in Sweden and Denmark was unexpected. Rather than die-offs of large numbers of waterfowl during winter when they congregate, small numbers (mainly singleton birds) were affected late in winter, just before spring migration. During the spring breeding season, less transmission was observed. The predictive power of detecting the virus in free-ranging migratory birds for forecasting poultry outbreaks or human disease remains undetermined. Some of these birds may have been infected in areas remote from the site of detection. However, several of the affected birds in this report were either resident nonmigratory species (eagle owl, Eurasian magpie) or captive domesticated species (muscovy, peafowl, chicken), which indicates local transmission. Health authorities will be better prepared to prospectively minimize transmission in new regions with early warning provided by singleton carcass surveillance.

Surveillance results from Sweden and Denmark highlight the importance of public participation in avian mortality surveillance for influenza virus (H5N1); the preponderance of detections from singleton carcasses; and

Table. Bird species testing positive for highly pathogenic avian influenza virus subtype H5N1, Sweden and Denmark, 2006\*

		No. carcasses positive	
Avian order and species	Scientific name	Sweden	Denmark
Podicepidiformes (great crested grebe)	Podiceps cristatus	0	1
Anseriformes			
Mute swan	Cygnus olor	2	4
Whooper swan	Cygnus cygnus	0	3
Greylag goose	Anser anser	0	1
Goose spp	Anser spp.	1	0
Muscovy duck	Cairina moschata	0	2
Mallard	Anas platyrhynchos	1	0
Greater scaup	Aythya marila	3	0
Tufted duck	Aythya fuligula	25	26
Common merganser	Mergus merganser	2	0
Smew	Mergus albellus	1	0
Falconiformes			
Common buzzard	Buteo buteo	1	6
Rough-legged hawk	Buteo lagopus	0	1
Peregrine falcon	Falco peregrinus	0	1
Galliformes			
Common peafowl	Pavo cristatus	0	1
Domestic chicken	Gallus gallus	0	1
Charadriiformes (herring gull)	Larus argentatus	1	0
Strigiformes (eagle owl)	Bubo bubo	2	0
Passeriformes (Eurasian magpie)	Pica pica	0	1
All birds		39	48
*Sources: (6,7).			

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the broad spectrum of affected species, particularly raptors. A raptor was the index case in Denmark (7). Current surveillance efforts in regions free from the virus favor investigation of significant death events of waterfowl and active sampling of healthy waterfowl as the means for early detection (e.g., 9). Many national surveillance programs are heavily influenced by the influenza virus (H5N1) outbreak in 2005 at Qinghai Lake in China, where hundreds of geese, gulls, and cormorants died during the breeding season (10). However, large die-offs may be anomalous or restricted to communal breeding sites of waterfowl where juvenile birds amplify and spread the virus within the breeding colony. Testing of public-reported singleton carcasses provides a more sensitive and robust means of early detection of this virus.

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## Nicholas Komar\* and Björn Olsen†

\*Centers for Disease Control and Prevention, Fort Collins, Colorado, USA; and †Uppsala University, Uppsala, Sweden

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Address for correspondence: Nicholas Komar, Centers for Disease Control and Prevention, Arbovirus Diseases Branch, 3150 Rampart Rd, Fort Collins, CO 80521, USA; email: nkomar@ cdc.gov

# *Klebsiella pneumoniae* Carbapenemase-2, Buenos Aires, Argentina

**To the Editor:** The activity of carbapenem has been compromised because of the emergence of carbapenemases (1). Since 1995, carbapenem resistance has been identified among 77 *Klebsiella pneumoniae* isolates and 1 *Citrobacter freundii* clinical isolate

in Argentina (WHONET-Argentina Network). However, until now, none had produced a carbapenemase.

*K. pneumoniae* carbapenemase-1 (KPC-1) was first detected in a *K. pneumoniae* strain isolated in North Carolina in 2001 (1). Since that time, several reports of KPCs worldwide have been made, including in South America (1). We report on KPC-2 –producing *K. pneumoniae* and *C. freundii* clinical isolates in Argentina.

A 36-year-old woman with systemic lupus erythematosus and chronic renal failure was admitted to the Sanatorio Mitre in Buenos Aires in September 2006 for a kidney transplant. Two months after the transplant, intraabdominal collection obtained during a surgical procedure vielded a carbapenem-susceptible Escherichia coli isolate, after which meropenem therapy was initiated (1 g/day). After 16 days of treatment, an infection developed at the patient's surgical site (per US Centers for Disease Control and Prevention criteria, available from www.cdc.gov/ncidod/dhqp/pdf/guide lines/SSI.pdf). C. freundii M9169 and K. pneumoniae M9171 were both isolated from the same specimen obtained from the surgical site. Because carbapenemase production was suspected, carbapenem treatment was stopped, and the infection was treated with local antiseptic and drainage for 20 days; the patient was discharged from the hospital in January 2007. Neither the patient nor her relatives or hospital staff had been in the United States before the emergence of these strains.

By using disk diffusion (2) (Mueller-Hinton agar and disks obtained from Difco and BBL, respectively; Becton, Dickinson and Co., Franklin Lakes, NJ, USA), we determined that *K. pneumoniae* M9171 was resistant to all antimicrobial drugs except amikacin, tetracycline (3), and tigecycline (US Federal Drug Administration criteria, susceptible  $\geq$ 19 mm). *C. freundii* M9169 remained susceptible