

References

1. Sánchez-Seco MP, Rosario D, Domingo C, Hernández L, Valdés K, Guzmán MG, et al. Generic RT-nested-PCR for detection of flaviviruses using degenerated primers and internal control followed by sequencing for specific identification. *J Virol Methods*. 2005;126:101–9. DOI: 10.1016/j.jviromet.2005.01.025
2. Prince HE, Lape-Nixon M, Moore RJ, Hogrefe WR. Utility of the Focus Technologies West Nile virus immunoglobulin M capture enzyme-linked immunosorbent assay for testing cerebrospinal fluid. *J Clin Microbiol*. 2004;42:12–5. DOI: 10.1128/JCM.42.1.12-15.2004
3. Echevarría JM, Martínez-Martín P, Téllez A, de Ory F, Rapún JL, Bernal A, et al. Aseptic meningitis due to varicella-zoster virus: serum antibody levels and local synthesis of specific IgG, IgM, and IgA. *J Infect Dis*. 1987;155:959–67.
4. Kramer LD, Li J, Shi PY. West Nile virus. *Lancet Neurol*. 2007;6:171–81. DOI: 10.1016/S1474-4422(07)70030-3
5. Dauphin G, Zientara S, Zeller H, Murgue B. West Nile virus: worldwide current situation in animals and humans. *Comp. Comp Immunol Microbiol Infect Dis*. 2004;27:343–55. DOI: 10.1016/j.cimid.2004.03.009
6. Zeller HG, Schuffenecker IBB. West Nile virus: An overview of its spread in Europe and Mediterranean basin in contrast to its spread in the Americas. *Eur J Clin Microbiol Infect Dis*. 2004;23:147–56. DOI: 10.1007/s10096-003-1085-1
7. Nash D, Mostashari F, Fine A, Miller J, O’Leary D, Murray K, et al. The outbreak of West Nile virus infection in the New York City area in 1999. *N Engl J Med*. 2001;344:1807–14. DOI: 10.1056/NEJM200106143442401
8. Charles PE, Zeller H, Bonnotte B, Descasimacker AL, Bour JB, Chavanet P, et al. Imported West Nile virus Infection in Europe. *Emerg Infect Dis*. 2003;9:750.
9. Gebhardt DO. Another case of West Nile fever in the Netherlands: a man with encephalitis following a trip to Canada. *Ned Tijdschr Geneesk*. 2003;147:1336.
10. Hubalek Z, Lukacova L, Halouzka J, Srucek P, Januska J, Precechtelova J, et al. Import of West Nile virus infection in the Czech Republic. *Eur J Epidemiol*. 2006;21:323–4. DOI: 10.1007/s10654-006-0019-5

Address for correspondence: Rogelio López-Vélez, Ramón y Cajal Hospital–Infectious Diseases, Carretera de Colmenar 9,1, Madrid 28230, Spain; email: rlopezvelez.hrc@salud.madrid.org

Outbreak of Pertussis, Kabul, Afghanistan

To the Editor: Infectious diseases are the main cause of illness for armed forces in conflict (*I*), resulting in decreases in operational efficiency. The International Security Assistance Force (ISAF) in Afghanistan is a multinational force operating under the auspices of the North Atlantic Treaty Organization (NATO). As part of ISAF, French troops operate in Kabul and its surroundings, within a 70-km radius. French medical facilities consist of a French field hospital and a primary care center. The facilities support 4,000 soldiers, 1,048 of whom are French.

Troop disease, including acute respiratory disease (ARD), is routinely monitored through French Army and NATO surveillance systems. We report an outbreak of ARD in the multinational force in which pertussis cases were identified by using laboratory tests and epidemiologic criteria.

In November 2006, a significant increase of ARD was detected in soldiers of different nationalities (Figure), with a 10-fold increase among French troops at week 51. Patients with persistent cough or dyspnea were referred to the field hospital, in a nonrandomized manner, and those with a 2-week history of cough underwent serologic tests. Samples were sent to France and were analyzed at Hôpital Saint Anne, Toulon, France. Immunoglobulin (Ig) G antibodies to *Bordetella pertussis* antigens (pertussis toxin, filamentous hemagglutinin, and adenylcyclase) were determined by a Western blot assay (MarDx Diagnostics, Carlsbad, CA, USA). Recent infection was diagnosed by finding high levels of antibodies to pertussis toxin compared to results for standardized positive and negative samples, in concurrence with the fact that no soldier had been vaccinated against pertussis after childhood.

IgG and IgA antibodies to *Chlamydia pneumoniae* were determined by a semiquantitative method that assessed samples’ absorbance value in optical density (SeroCP Quant IgG and Quant IgA, Savyon Diagnostics, Ashdod, Israel). Recent infection to *Mycoplasma pneumoniae* was assessed by detecting IgM antibodies with a specific enzyme immunoassay (Platelia *Mycoplasma pneumoniae*, Biorad, Hercules, CA, USA) and by using a semiquantitative method to detect IgM and IgG antibodies with patented gelatin particles sensitized with cell membrane components of *M. pneumoniae* (Serodia Myco II, Fujirebio, Malvern, PA, USA). *Coxiella burnetii* infection was assessed by indirect immunofluorescence assay (*Coxiella burnetii* Spot IF, bioMérieux, Marcy l’Etoile, France).

Statistical analysis was performed with Epi Info v3.4 software package (Centers for Disease Control [CDC], Atlanta, GA, USA). Quantitative variables were compared by using the Kruskal-Wallis test.

From the third week of December 2006 until the third week of January 2007, 209 French soldiers sought treatment at the French medical facilities for stereotyped acute febrile respiratory infection, which represents a cumulative attack rate of 20% on clinical grounds. Thirty-nine French soldiers and 10 non-French soldiers or local civilian workers were then referred to the field hospital. All patients had a 24-h history of fever >38.5°C and nonspecific ear, nose, and throat symptoms, mainly a sore throat. Cough was unremarkable, without whoops. Fourteen of the 49 patients were hospitalized for severe dyspnea. Median age was the same for inpatients (26 [range 20–57] years) and outpatients (36 [range 21–53] years, $p = 0.15$).

Twenty-seven blood samples were taken, 24 from French troops, 2 from British troops, and 1 from Polish patients. Six patients, including 3 French soldiers, had recent pertussis. No difference in age was found between

patients with pertussis and those with non-pertussis ARD (36 [range 27–51] versus 33 [range 20–63] years; $p = 0.39$). No pertussis patient had been vaccinated against the illness since childhood.

One patient had evidence of recent infection with *M. pneumoniae*, and another with *C. pneumoniae*. No recent infection involved *C. burnetii*. All patients with ARD had a favorable outcome.

This outbreak of ARD among troops in Afghanistan highlights the importance of nontraumatic illness in wartime when military field conditions enhance exposure to, and incidence of, endemic diseases. Although our study did not include systematic laboratory confirmation for all cases of ARD in soldiers due to field conditions, this outbreak was mainly due to pertussis: most cases were defined by a cough lasting ≥ 2 wk, took place in an outbreak setting, and were (for 6 patients) confirmed by laboratory tests. CDC requirements were followed to ascertain confirmed cases (2). This outbreak also involved British troops; after the 2 cases we described, 2 additional serologically confirmed cases and 1 prob-

able confirmed case were discovered among symptomatic British returnees (3). Pertussis, which remains endemic in developing countries (4), was reported in northeastern Afghanistan in 2002 (5), nor was it ever biologically ascertained nor reported in Kabul.

This outbreak elicits 3 main questions. First, how can ARD transmission be stopped under field conditions? Besides prophylactic antibiotherapy, isolation of suspected case-patients is not achievable because of limited number of beds in medical facilities and high-person density in barracks and dining halls. To minimize transmission, patients and caregivers should wear masks.

Second, what prophylactic antibiotherapy should be given? We recommend a 3-day regimen of azithromycin because it is as efficient as erythromycin in preventing spread pertussis (6), targets most intracellular bacteria involved in ARD, and offers the best compliance (7).

Finally, should soldiers be vaccinated against pertussis for overseas campaigns? In France, no booster vaccination is given after 13 years of age (8). Because acellular vaccines do not

ensure immunity for >6 years (5), no French soldier has immunity to pertussis. We therefore advocate booster vaccination before overseas campaigns. Pertussis vaccination is widely available in combination with vaccination against, at minimum, diphtheria and tetanus, but these combination vaccines can only be performed once in an adult's life and only 2 years after previous vaccination against diphtheria or tetanus. Monovalent vaccines against pertussis must be made more widely available for multinational troops in field conditions.

Acknowledgments

We are indebted to Martin Powell for helpful comments in preparing the manuscript.

**Emmanuel Sagui,*
Lénaïck Ollivier,†
Tiphaine Gaillard‡
Fabrice Simon,*
Patrick Brisou,‡
Philippe Puech,§
and Alain Todescot‡**

*Hôpital d'Instruction des Armées Laveran, Marseille, France; †Institut de Médecine Tropicale du Service de Santé des Armées, Marseille Armées, France; ‡Hôpital d'Instruction des Armées Saint Anne, Toulon Naval, France; and §Etat Major 4, Marseille Armées

DOI: 10.3201/eid1407.071329

References

- Sanders JW, Putnam S, Frankart C, Frenck R, Monteville M, Riddle M, et al. Impact of illness and non-combat injury during operations Iraqi Freedom and Enduring Freedom (Afghanistan). *Am J Trop Med Hyg.* 2005;73:713–9.
- Centers for Disease Control and Prevention. Case definitions for infectious conditions under public health surveillance. *MMWR Recomm Rep.* 1997;46:1–55.
- Cooper NK, Bricknell MCM, Holden GR, McWilliam C. Pertussis—a case finding study amongst returnees from Op Herrick. *J R Army Med Corps.* 2007;153:114–6.
- Singh M, Lingappan K. Whooping cough? The current scene. *Chest.* 2006;130:1547–53. DOI: 10.1378/chest.130.5.1547

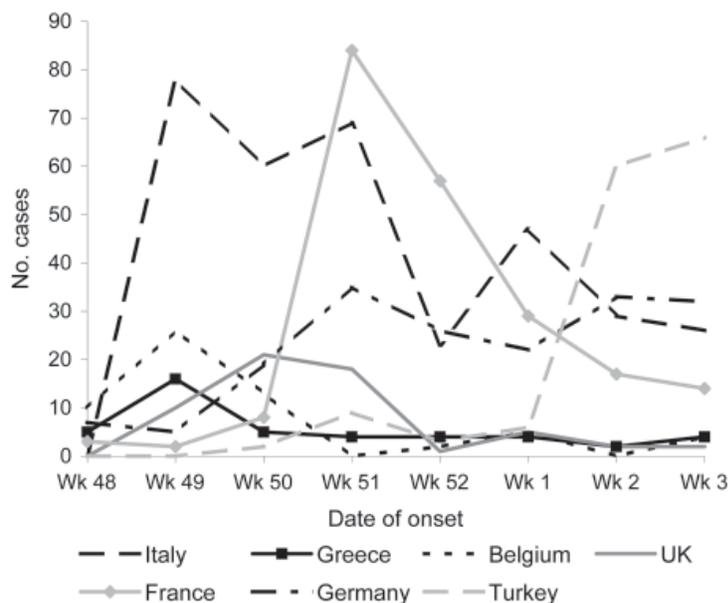


Figure. Number of acute respiratory diseases cases, according to troop nationality. UK, United Kingdom. A color version of this figure is available online: www.cdc.gov/EID/content/14/7/1173-G.htm

5. World Health Organization. Epidemic and pandemic alert and response: pertussis [cited 2007 Aug 5]. Available from <http://www.who.int/csr/don/archive/disease/pertussis/en>
6. Wirsing von König CH, Halperin S, Riffelmann M, Guiso N. Pertussis of adults and infants. *Lancet Infect Dis.* 2002;2:744–50. DOI: 10.1016/S1473-3099(02)00452-8
7. Altunajji S, Kukuruzovic R, Curtis N, Massie J. Antibiotics for whooping cough (pertussis). *Cochrane Database Syst Rev.* 2005;1:CD004404.
8. Calendrier vaccinal 2006. *Bulletin épidémiologique hebdomadaire.* 2006;29–30: 212–26.

Address for correspondence: Emmanuel Sagui, Service de Neurologie, Hôpital d'Instruction des Armées Laveran, BP 50, 13013 Marseille, France; email: emmanuel.sagui@laposte.net

Anthropogenic Influence on Prevalence of 2 Amphibian Pathogens

To the Editor: Although the relationship between the emergence of zoonotic diseases and human influenced landscapes is accepted (1–3), the relationship between human-influenced landscapes and wildlife disease is less so. Evidence does support correlations between human activities and environmental conditions affecting wildlife disease emergence (2,3). These studies assume relationships between component(s) of human hab-

itat modification and the virulence of disease, and derive estimates of virulence from counts of the visibly diseased or those that have seroconverted (3). This explains only part of the host and pathogen dynamic; it seems reasonable to extend the relationship to include prevalence of infection. Data supporting this extension are lacking. Here we present data from a study examining the correlations between human influences on habitat and prevalence of 2 amphibian pathogens (*Batrachochytrium dendrobatidis* and ranavirus FV3) in populations of *Rana clamitans* in central and northeastern Ontario, Canada.

We sampled an average of 25 animals (standard deviation \pm 6.16) from 11 populations during summer 2005. We washed equipment in bleach and air-dried equipment between visits and sites. All animals were kept individually to avoid cross-contamination, euthanized with MS22, and assessed for infection using molecular diagnostics. We tested for ranavirus infection of livers by amplifying the major capsid protein using standard PCR (4). We tested for infection with *B. dendrobatidis* by using a quantitative real-time PCR (5). Prevalence for each pathogen was estimated as the proportion of animals testing positive at a pond.

Site coordinates were determined by using global positioning satellite (GPS), and 4 quantitative measures of human habitat modification were also assessed. GPS coordinates were used to map sites and to measure distance to the nearest road, industrial activity (agriculture, mine, paper mill), and human habitation; all measurements

were in meters. We further assigned a qualitative measure of human influence on each breeding pond by assigning ponds to each of the following categories: 1) human presence without human habitat modification or extensive disturbance; 2) recreational activities (fishing, boating); 3) property development (housing or commercial buildings); 4) agricultural activity; and 5) industrial activity. Each of the 5 categories was assigned a 0/1 score; scores for each pond were summed 1–5 (by definition no site scored 0 due to sampling strategy) to derive the final measure of human influence. We modeled the relationship between prevalence and human habitat modification or influence using general linear models (GLM) with prevalence as the dependent variable and with all human influence variables log-transformed to meet assumptions of normality. A type III model structure was used to account for the influence of all explanatory variables in each analysis.

Eight ponds exhibited signs of FV3 infection (range 0%–63% prevalence); 6 ponds contained frogs infected with the amphibian chytrid (range 0%–36% prevalence). GLM did not show any relationship between the prevalence of chytrid infection and all of our explanatory variables (Table). In contrast, 3 of our explanatory variables had a significant influence of ranavirus prevalence. Distance to industrial activity ($p < 0.05$), to human habitation ($p < 0.05$), and degree of human influence ($p < 0.01$) all had a significant effect on the dependent variable (Table).

The disparity between results for the 2 pathogens generates several possible hypotheses. First, proximity to

Table. General linear models for the relationships of amphibian emerging infectious disease prevalence and anthropogenic variables

Data point	Degrees of freedom	Ranavirus		<i>Batrachochytrium dendrobatidis</i>	
		Mean squares	F value	Mean squares	F value
Intercept	1	0.06	8.47 ($p < 0.05$)	0.05	4.61 ($p < 0.1$)
Human disturbance	1	0.24	35.35 ($p < 0.01$)	0.0009	0.08
Distance to road	1	0.03	4.11 ($p < 0.1$)	0.03	2.82
Distance to industry	1	0.06	8.82 ($p < 0.05$)	0.07	5.89 ($p < 0.1$)
Distance to housing	1	0.06	8.08 ($p < 0.05$)	0.06	4.87 ($p < 0.1$)
Error	5	0.01		0.01	