Control of zoonoses in emergency situations: lessons learned during recent outbreaks (gaps and weaknesses of current zoonoses control programmes)

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Summary

In emergency situations, domestic animals and wildlife are, like people, exposed to infectious diseases and environmental contaminants in the air, soil, water and food. They can suffer from acute and/or chronic diseases from such exposure. Often animals serve as disease reservoirs or early warning systems for the community in regard to the spread of zoonotic diseases. Over 100 years of experience have shown that animal and human health are closely related. During the past few years, emergent disease episodes have increased; nearly all have involved zoonotic agents. As there is no way to predict when or where the next important new zoonotic pathogen will emerge or what its ultimate importance might be, investigation at the first sign of emergence of a new zoonotic disease is particularly important. Today, in many emerging situations, different activities involving zoonotic disease control are at risk because of failed investigative infrastructures or financial constraints. Considering that zoonotic diseases have their own characteristics, their prevention and control require unique strategies, based more on fundamental and applied research than on traditional approaches. Such strategies require cooperation and coordination between animal and public health sectors and the involvement of other disciplines and experts such as epidemiologists, entomologists, environmentalists and climatologists. Lessons learned from the avian influenza pandemic threat, the Crimean-Congo haemorrhagic fever and rabies outbreaks are presented and the gaps and weakness of current control programmes are discussed.

Keywords

Animal, Control, Disease, Emergency, Outbreak, Public health, Zoonoses.

Controllo delle zoonosi nelle situazioni di emergenza: la lezione appresa nel corso dei recenti focolai (lacune e debolezze degli attuali programmi di controllo delle zoonosi)

Riassunto

Nelle situazioni di emergenza gli animali domestici e quelli selvatici, al pari dell'uomo, sono esposti a patologie infettive e contaminanti ambientali derivanti dall'aria, dal suolo, dall'acqua e dagli alimenti. A causa di tali esposizioni possono contrarre patologie acute e/o croniche. Sovente gli animali rappresentano un serbatoio di malattie o anche un sistema di allerta rapido per la comunità allorquando si diffondono patologie zoonosiche. Più di 100 anni di esperienza dimostrano che la salute umana e animale sono strettamente correlate. Nel corso degli ultimi anni sono aumentati gli episodi di patologie emergenti e quasi tutti coinvolgono agenti zoonotici. Dal momento che non è possibile prevedere quando o dove si manifesterà il prossimo nuovo e importante patogeno agente di zoonosi o

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quale potrà essere la sua gravità, è particolarmente importante investigare sin dal primo segnale di manifestazione della patologia. Oggi, in molte situazioni di emergenza, sono a rischio molte attività che coinvolgono il controllo delle patologie zoonotiche a causa di carenti infrastrutture investigative o vincoli finanziari. Dal momento che le zoonosi hanno peculiari caratteristiche, la loro prevenzione e il loro controllo necessitano di strategie mirate, basate più sulla ricerca fondamentale e applicata che su approcci tradizionali. Tali strategie richiedono cooperazione e coordinamento tra settori della sanità animale e della sanità pubblica e il coinvolgimento di altre discipline nonchè di esperti epidemiologi, entomologi, ambientalisti e climatologi. Nel presente lavoro vengono illustrate le lezioni apprese nel corso della minaccia di pandemia di influenza aviaria, di Crimean-Congo haemorrhagic fever e dei focolai di rabbia e si discutono lacune e debolezze degli attuali programmi di controllo.

Parole chiave
Animale, Controllo, Emergenza, Focolaio, Malattia, Sanità Pubblica, Zoonosi.

Introduction

In the past few years, emerging disease episodes have increased globally. The list of the most significant of these diseases is impressive indeed and, given what we know about disease ecology, the will continue to grow. Nearly all of these disease episodes have involved zoonotic infectious agents, that is, they have involved the transmission of the aetiological agent to humans from an ongoing reservoir life cycle in animals or arthropods, without the permanent establishment of a new life cycle in humans. Fewer episodes have involved the jumping of the species barrier by the aetiological agent; that is, they derive from an ancient reservoir life cycle in animals but have subsequently established a new life cycle in humans that no longer involves an animal reservoir.

The relationship between natural disasters and zoonotic diseases is frequently misconstrued. The risk of the occurrence of outbreaks is often presumed to be very high in the chaos that follows natural disasters, a fear that is most likely derived from a perceived association between dead bodies and epidemics. However, the risk factors in regard to outbreaks after the occurrence of a disaster are associated primarily with population displacement. The availability of safe water and sanitation facilities, the degree of crowding, the underlying health status of the population and the availability of health care and veterinary services all interact within the context of the local disease ecology to influence the risk for zoonotic diseases and death in the affected population. There is a strong need to outline the risk factors for outbreaks after a disaster and to review the zoonotic diseases that are likely to be important. Furthermore, it is necessary that priorities are clearly established to address zoonotic diseases in disaster settings.

Natural disasters are catastrophic events with atmospheric, geological and hydrological origins. Disasters include earthquakes, volcanic eruptions, landslides, tsunamis, floods and drought. Natural disasters can have a rapid or slow onset, with serious health, social and economic consequences. During the past two decades, natural disasters have killed millions of people and animals, adversely affected the lives of at least one billion more people and many animal populations. They have also resulted in substantial economic damage. Developing countries are disproportionately affected because they often lack resources, infrastructure and disaster-preparedness systems.

The risk of zoonotic disease transmission in emergency situations is associated primarily with the size and characteristics of the population that has been displaced, specifically the proximity of safe water and functioning latrines, the nutritional status of the displaced populations, the level of immunity to vaccine-preventable diseases and the access to health care and veterinary services. Outbreaks are less frequently reported in disaster-affected populations than in conflict-affected areas, where two-thirds of deaths may be caused by communicable and zoonotic diseases. Malnutrition increases the risk of death from zoonotic diseases and is more common in conflict-affected populations, particularly if displacement is related to long-term conflicts.

Outbreaks that occur after flooding are better documented than those that occur after earthquakes, volcanic eruptions, tsunamis and natural disasters (regardless of the type of disaster) which do not result in the displacement of populations. Historically, the large-scale displacement of populations as a result of natural disasters is not common; this is likely to contribute to the low risk of outbreaks occurring and to the variability in risk among disasters of different types.

Risk factors for zoonotic disease transmission

Responding effectively to the needs of a population affected by disaster requires an accurate zoonotic disease risk assessment. The efficient use of humanitarian funds depends on implementing priority interventions on the basis of this risk assessment.

A systematic and comprehensive evaluation should identify the following:

- endemic and epidemic diseases that are common in the affected area
- living conditions of the affected population, including number, size, location and density of settlements
- availability of safe water and adequate sanitation facilities
- underlying nutritional status and immunisation coverage among the population
- degree of access to health care, veterinary services and to effective case management.

Zoonotic diseases associated with natural disasters

Water-related zoonotic diseases and vector-borne diseases have been associated with populations displaced by natural disasters. These diseases should be considered when post-disaster risk assessments are performed.

Water-related zoonotic diseases

Access to safe water can be jeopardised by a natural disaster. Outbreaks of diarrhoeal disease can occur after drinking water has been contaminated and reports have been made after flooding and related displacement. An outbreak of diarrhoeal disease after flooding in Bangladesh in 2004 involved >17 000 cases; *Vibrio cholerae* (O1 Ogawa and O1 Inaba) and enterotoxigenic *Escherichia coli* were isolated. A large (>16 000 cases) cholera epidemic (O1 Ogawa) in West Bengal in 1998 was attributed to preceding floods and floods in Mozambique in January-March 2000 led to an increase in the incidence of diarrhoea.

In an extensive study undertaken in Indonesia in 1992-1993, flooding was identified as a significant risk factor for diarrhoeal illnesses caused by *Salmonella enterica* serotype Paratyphi A (paratyphoid fever). In a separate evaluation of risk factors for infection with *Cryptosporidium parvum* in Indonesia in 2001-2003, case-patients were more than four times more likely than controls to have been exposed to flooding.

The risk of diarrhoeal disease outbreaks occurring after natural disasters is greater in developing countries than in industrialised countries. In the Aceh Province of Indonesia, a rapid health assessment in the town of Calang two weeks after the December 2004 tsunami revealed that 100% of the survivors drank from unprotected wells and that 85% of residents reported diarrhoea in the previous two weeks. In Muzaffarabad, Pakistan, an outbreak of acute watery diarrhoea occurred in an unplanned, poorly equipped camp of 1 800 people after the 2005 earthquake. The outbreak involved >750 cases, mostly adults, and was controlled after adequate water and sanitation facilities were provided.

Leptospirosis is an epidemic-prone zoonotic bacterial disease that can be transmitted by direct contact with contaminated water. Rodents shed large quantities of leptospires in their urine and transmission occurs through contact of the skin and mucous membranes with water, damp soil or vegetation (such as sugar cane), or mud contaminated with rodent urine. Flooding facilitates spread of the micro-organisms because of the proliferation of rodents and the proximity of rodents to humans on shared high ground. Outbreaks of leptospirosis occurred in Taiwan, associated with Typhoon Nali in 2001, in Mumbai (India), after flooding in 2000, in Argentina after flooding in 1998 and in the Krasnodar Region of the Russian Federation in 1997. After a flooding-related outbreak of leptospirosis in Brazil in 1996, spatial analysis indicated that incidence rates of leptospirosis doubled within the flood-prone areas of Rio de Janeiro.

Vector-borne diseases

Natural disasters, particularly meteorological events such as cyclones, hurricanes and flooding, can affect the breeding sites of vectors and vector-borne disease transmission. While initial flooding may wash away existing mosquito breeding sites, standing water caused by heavy rainfall or the overflow of rivers and climate change can create new breeding sites. This situation can result (typically with a delay of a few weeks) in an increase of the vector population and potential for disease transmission, depending on the local mosquito vector species and its preferred habitat. The crowding of infected and susceptible hosts, a weakened public health infrastructure and interruptions of ongoing control programmes are all risk factors for vector-borne disease transmission.

Malaria outbreaks in the wake of flooding are a well-known phenomenon. An earthquake in Costa Rica's Atlantic Region in 1991 was associated with changes in habitat that were beneficial for breeding and preceded an extreme rise in malaria cases. In addition, periodic flooding linked to the El Niño-Southern Oscillation has been associated with malaria epidemics in the dry coastal region of northern Peru.

Dengue transmission is influenced by meteorological conditions, including rainfall and humidity, and often shows strong seasonality. However, transmission is not directly associated with flooding. Such events may coincide with periods of high risk for transmission and may be exacerbated by the increased availability of the vector's breeding sites (mostly artificial containers) caused by a disruption in the basic water supply and solid waste disposal services. The risk of outbreaks can be influenced by other factors, such as changes in human behaviour (increased exposure to mosquitoes while sleeping outside, movement from areas that are not endemic to endemic areas of dengue, a pause in disease control activities, overcrowding) or changes in the habitat that promote mosquito breeding (landslides, deforestation, river damming and rerouting of water).

Distinct prevention and control strategies

Prevention and control strategies for diseases caused by zoonotic agents are different from those required for diseases the aetiological agent of which has long relied on human-to-human transmission for survival.

In general, there is no way of predicting when or where the next important new zoonotic pathogen will emerge or what its ultimate importance might be. A pathogen might emerge as the cause of a geographically limited curiosity, intermittent disease outbreaks, or a new epidemic. Consequently, investigation at the first sign of emergence of a new zoonotic disease is particularly important, although the investigation usually resembles a field- and laboratory-based research project rather than a typical case-control-based outbreak investigation. This reality must drive strategic planning to deal with new zoonotic diseases.

Factors contributing to the emergence of zoonotic diseases

Many elements can contribute to the emergence of a new zoonotic disease, in particular: microbial/virological determinants, such as mutation, natural selection and evolutionary progression, individual host determinants, such as acquired immunity and physiologic factors, host population determinants, such as host behavioural characteristics and societal, transport, commercial and iatrogenic factors and environmental determinants, such as ecological and climatological influences.

The emergence of new zoonotic pathogens seems to be increasing for several reasons, namely: global human and livestock animal populations have continued to grow, bringing increasingly greater numbers of people and animals into close contact, transportation has advanced enormously, making it possible to circumnavigate the globe in less than the incubation period of most infectious agents; ecological and environmental changes brought about by human activity are massive and, in most instances, the infectious agents of choice seem to be zoonotic.

Ecological factors that contribute to the emergence of zoonotic diseases

Among the factors that contribute to the emergence of zoonotic diseases is the capacity of microorganisms and viruses to adapt to extremely diverse and changing eco-niches. One of the most complex sets of adaptations concerns the arboviruses and their transmission by specific arthropods. When ecosystems are altered, disease problems of humans and animals follow. Population movements and the intrusion of humans and domestic animals into arthropod habitats have resulted in emergent disease episodes, some of which resemble fiction.

Deforestation and settlement of new tropical forest and farm margins have exposed farmers and domestic animals to new arthropods and the viruses they carry. Increased long-distance air travel facilitates the movement of infected people and exotic arthropod vectors around the world. Increased long-distance livestock transportation facilitates the movement of viruses and arthropods (especially ticks) across the globe. The introduction, emergence and the possible transport of Crimean-Congo hemorrhagic fever (CCHF) virus or other tick-borne pathogens have been moved to new locales. Ecological factors pertaining to uncontrolled urbanisation and environmental pollution are contributing to many emergent disease episodes. Arthropod vectors breeding in accumulations of water (e.g. tin cans, old tyres) and sewage-laden water are a problem worldwide. Environmental chemical toxicants (herbicides, pesticides, residues) can also affect vector-virus relationships either directly or indirectly. Ecological factors related to expanding primitive irrigation systems are becoming important in virus disease emergence. New routes of long-distance bird migrations, brought about by new man-made water impoundments, represent an important yet still untested risk of introduction of arboviruses into new areas. Global warming, which affects sea level, estuarine wetlands, fresh water swamps and human habitation patterns, may also be affecting vector-virus relationships throughout the tropics. However, data are scarce and long-term programmes to study the effect of global warming have too often not included the participation of tropical and sub-tropical medicine and veterinary experts.

Of all the ecological factors that contribute to arthropod-borne zoonotic viral disease emergence, uncontrolled urbanisation is the most important. The mega cities of the tropics and subtropics, with their lack of sanitary systems, serve as incubators for emerging zoonoses – they represent the greatest zoonotic disease threat of this century. Who will pay for the control of disease in these cities? How will the World Health Organization (WHO) and its Mediterranean Zoonoses Control Programme (MZCP) serve the needs of the people in these cities? Lessons from the past suggest that we need more extended national and international cooperation to deal with emerging zoonoses in such settings and, more than ever before, we need an adaptable enterprise, one that can adjust rapidly to diverse episodes.

Lessons learned

Crimean-Congo haemorrhagic fever

Although an inactivated, mouse brain-derived vaccine against CCHF has been developed and used on a small scale in Eastern Europe, there is no safe and effective vaccine that is widely available for human use.

Tick vectors are numerous and widespread and tick control with acaricides (chemicals manufactured to kill ticks) is only a realistic option for well-managed livestock production facilities. People living in endemic areas should use personal protective measures that include avoidance of areas where tick vectors are abundant and at times when they are active (spring to autumn), regular examination of clothing and skin for ticks and their removal and use of repellents. People who work with livestock or other animals in endemic areas can take practical measures to protect themselves. Protective measures include the use of repellents on the skin and clothing (e.g. permethrin) and wearing gloves or other protective clothing to prevent skin contact with infected tissues or blood.

When patients with CCHF are admitted to the hospital, there is a risk of nosocomial spread of infection. In the past, serious outbreaks have occurred in this way and it is imperative that adequate infection control measures be observed to prevent this disastrous outcome, in particular:

patients with suspected or confirmed CCHF should be isolated and cared for using barrier nursing techniques

specimens of blood or tissues taken for diagnostic purposes should be collected and handled using universal precautions

sharp objects (needles and other penetrating surgical instruments) and body waste should be safely disposed of using the appropriate decontamination procedures

Health care workers are at risk of acquiring infection from injuries caused by sharp objects during surgical procedures and, in the past, infection has been transmitted to surgeons who operate patients to determine the cause of the abdominal symptoms in the early stages of infection (i.e. at that stage undiagnosed). Health care workers who have had contact with tissues or blood from patients with suspected or confirmed CCHF should be monitored (checking daily temperature and symptoms) for at least 14 days after putative exposure.

Rabies epidemics

Rabies provides many lessons on how viral adaptation contributes to emergence in new eco-niches. Often, the necessary ecological elements are in place and the recipe for emergence simply involves the introduction of a virus. A dramatic illustration was the appearance of epidemic raccoon rabies in the eastern United States. The epidemic was traced to raccoons imported from Florida to West Virginia in 197. As usual, human perturbation of an ecosystem, in this instance involving the transport of wild raccoons from an endemic site, caused the problem. One key to our understanding of this episode was the discovery that the rabies virus is not one virus; but rather, it is a set of different genotypes, each transmitted within a separate reservoir host eco-niche. In North America, there are six terrestrial animal genotypes, including the raccoon virus genotype. Raccoons bite raccoons that bite raccoons and, after some time, the virus becomes a distinct genotype that is highly adapted to the host cycle. Many mysteries of rabies ecology were clarified when the full significance of this discovery was realised. The lesson here is that modern virological research is the key for prevention and control programmes such as those carried out by the Centers for Disease Control Rabies Laboratory and the Texas State Health Department that is achieving much success with its coyote vaccination programme.

Avian influenza pandemic threat

Guidelines on disease prevention and control have been issued as joint recommendations of the World Organisation for Animal Health (*Office International des Épizooties*: OIE), the Food and Agriculture Organization and the World Health Organization. These recommendations, however, need to be put into practice in a variety of different field situations. The applicability of one system rather than another in a given situation must be evaluated, weighing the benefits of a successful result against the drawbacks of failure.

Until recently, highly pathogenic avian influenza (HPAI) infections caused by viruses of the H5 and H7 subtypes have occurred rarely and vaccination was not considered because stamping-out was the recommended control option. Primarily for this reason, vaccine procedures for HPAI have not grown at the same rate as for other infectious animal diseases. Data is being generated from experimental and field research in HPAI vaccine applications, but the rather complex task of vaccinating poultry in different farming and ecological environments still has areas of uncertainty.

Vaccination of fowl can be a powerful tool to support eradication programmes if used in conjunction with other control methods. Vaccination has been shown to increase resistance to field challenge, reduce shedding levels in vaccinated birds and reduce transmission. All these effects of vaccination contribute to controlling HPAI. However, experience has shown that, to be successful in controlling and ultimately in eradicating the infection, vaccination programmes must be part of a wider control strategy that includes biosecurity and monitoring the evolution of infection.

Control and eradication should be based on the elimination of insects and mice, depopulation of flocks and destruction of carcasses, removal of manure down to bare concrete, high pressure spraying to clean equipment and surfaces, spraying with residual disinfectants, enforcement of import restrictions, surveillance including appropriate biosecurity, controlling human traffic, introducing new birds into flocks, avoiding open range rearing in waterfowl prevalent areas, in addition to education of the poultry industry and prompt response to HPAI outbreaks.

In all these lessons, one of the most important points is the need for greater epidemiological resources and professionals who are better trained to deal with human and animal diseases or with the zoonotic interface between the two. This training component requires consideration of all steps along the discovery-to-control continuum.

The discovery-to-control continuum as applied to zoonotic diseases

Initial investigation at the first sign of emergence of a new zoonotic disease must focus on practical characteristics, such as death rate, severity of disease, transmissibility and remote spread, all of which are important predictors of epidemic potential and societal risk. Various elements of a discovery-to-control continuum are usually called for, in particular: discovery, recognition of a new zoonotic disease in a new setting; epidemiological field investigation; aetiological investigations, diagnostics development; focused research; technology transfer, training and outreach and, ultimately, control, elimination and eradication. Of course, not all of these elements are appropriate in every emerging zoonotic disease episode – decisions must be made and priorities must be set.

In the initial phases in the discovery-to-control continuum, people outside the 'family' (the traditional community of investigators and officials) must be recognised – local clinicians, pathologists (including medical examiners and forensic pathologists), veterinarians and animal scientists, ecologists, wildlife scientists, as well as local public health officials, many of whom have not been enamoured of their experiences in dealing with those 'inside' the family. The important early role of primary diagnostic laboratories and the reference laboratory networks that support them must also be recognised. In this era of the primacy of molecular microbiology and virology, it bears reminding that many of the early investigative activities surrounding the identification of a possibly emergent zoonotic disease must be performed in the field, not in the laboratory.

This phase may include expansion of many elements, such as technology transfer involving diagnostics development and proof testing, vaccine and drug development and proof testing, sanitation and vector control, medical and veterinary care activities and their adaptation to the circumstances of the disease locale, commercialisation, where appropriate, of diagnostics, vaccines and therapeutic agents in quantities required and provision of these materials through non-government organisations or government sources, training, outreach, continuing education and public education, each requiring professional expertise and adaptation to the special circumstances of the disease locale, communications, employing the technologies of the day such as the Internet and professional expertise.

More expensive and specialised expertise and resources come into play in the final phases of the discovery-to-control continuum, namely: public health systems, including rapid case-reporting systems, surveillance systems, vital records and disease registers, staffing and staff support, logistic support, legislation and regulation and expanded administration; special clinical systems, including isolation of cases, quarantine and patient care, public infrastructure systems, including sanitation and sewerage, safe food and water supplies and reservoir host and vector control.

The question of the need for facilities is an element of our capacity to fulfil the discovery-to-control continuum. What about biosafety level (BSL) 3+ and BSL-4 laboratory facilities Plans for a few small BSL-4 laboratories in academic centres may help to expand basic research supported by competitive grants, but they will not support expanded field-based research.

Further reading

1. Aggarwal R. & Krawczynski K. 2000. Hepatitis E: an overview and recent advances in clinical and laboratory research. *J Gastroenterol Hepatol*, **15**, 9-20.
2. Ahern M., Kovats R.S., Wilkinson P., Few R. & Matthies F. 2005. Global health impacts of floods: epidemiologic evidence. *Epidemiol Rev*, **27**, 36-46.
3. Barcellos C. & Sabroza P.C. 2001. The place behind the case: leptospirosis risks and associated environmental conditions in a flood-related outbreak in Rio de Janeiro. *Cad Saude Publ*, **17** (Suppl.), 59-67.
4. Brennan R.J. & Kimba K. 2005. Rapid health assessment in Aceh Jaya District, Indonesia, following the December 26 tsunami. *Emerg Med Aust*, **17**, 341-350.
5. Boumandouki P., Formenty P., Epelboin A., Campbell P., Atsangandoko C., Allarangar Y., Leroy E.M., Kone M.L., Molamou A., Dinga-Longa O., Salemo A., Kounkou R.Y., Mombouli V. Ibara J.R., Gaturuku P. Nkunku S., Lucht A. & Feldmann H. 2005. Clinical management of patients and deceased during the Ebola outbreak from October to December 2003 in Republic of Congo [article in French]. *Bull Soc Pathol Exot*, **98**, 218-223.
6. Campanella N. 2001. Infectious diseases and natural disasters: the effects of Hurricane Mitch over Villanueva municipal area, Nicaragua. Public Health Rev, 1999, **27**, 311-319.
7. Centers for Disease Control and Prevention 2005. Infectious disease and dermatologic conditions in evacuees and rescue workers after Hurricane Katrina – multiple states, August-September, 2005. *MMWR*, **54**, 961-964 (www.cdc.gov/mmwr/preview/mmwrhtml/mm54d926a1.htm accessed on 17 July 2008).
8. Centers for Disease Control and Prevention 2005. Norovirus outbreak among evacuees from Hurricane Katrina – Houston, Texas, September 2005. *MMWR*, **54**, 1016-1018 (www.cdc.gov/MMWR/preview/mmwrhtml/mm5440a3.htm accessed on 17 July 2008).
9. Centers for Disease Control and Prevention 2006. Two cases of toxigenic *Vibrio cholerae* O1 infection after Hurricanes Katrina and Rita – Louisiana, October 2005. *MMWR*, **55**, 31-32 (www.cdc.gov/mmwr/preview/mmwrhtml/mm5502a2.htm accessed on 17 July 2008).
10. de Ville de Goyet C. 2000. Stop propagating disaster myths. *Lancet*, **356**, 762.
11. de Ville de Goyet C. 2004. Epidemics caused by dead bodies: a disaster myth that does not want to die. *Rev Panam Salud Publ*, **15**, 297-299.
12. Floret N., Viel J.-F., Mauny F., Hoen B. & Piarroux R. 2006. Negligible risk for epidemics after geophysical disasters. *Emerg Infect Dis*, **12**, 543-548.
13. Gagnon A.S., Smoyer-Tomic K.E. & Bush A.B. 2002. The El Nino southern oscillation and malaria epidemics in South America. *Int J Biometeorol*, **46**, 81-89.

14. Gaspar M., Leite F., Brumana L., Felix B. & Stella A.A. 2001. Epidemiology of meningococcal meningitis in Angola, 1994-2000. *Epidemiol Infect*, **127**, 421-424.
15. Kalashnikov I.A., Mezentsev V.M., Mkrtchan M.O., Grizhebovskii G.M. & Briukhanova G.D. 2003. Features of leptospirosis in the Krasnodar Territory [article in Russian]. *Zh Mikrobiol Epidemiol Immunobiol*, Nov-Dec, 68-71.
16. Karande S., Bhatt M., Kelkar A., Kulkarni M., De A. & Varaiya A. 2003. An observational study to detect leptospirosis in Mumbai, India, 2000. *Arch Dis Child*, **88**, 1070-1075.
17. Katsumata T., Hosea D., Wasito E.B., Kohno S., Hara K., Soeparto P. & Ranuh I.G. 1998. Cryptosporidiosis in Indonesia: a hospital-based study and a community-based survey. *Am J Trop Med Hyg*, **59**, 628-632.
18. Kondo H., Seo N., Yasuda T., Hasizume M., Koido Y., Ninomiya N. & Yamamoto Y. 2002. Post-flood infectious diseases in Mozambique. *Prehospital Disaster Med*, **17**, 126-133.
19. Lifson A.R. 1996. Mosquitoes, models, and dengue. *Lancet*, **347**, 1201-1202.
20. Marin M., Nguyen H.Q., Langidrik J.R., Edwards R., Briand K., Papania M.J., Seward J.F. & LeBaron C.W. 2006. Measles transmission and vaccine effectiveness during a large outbreak on a densely populated island: implications for vaccination policy. *Clin Infect Dis*, **42**, 315-319.
21. Marx M.A., Rodriguez C.V., Greenko J., Das D., Heffernan R., Karpati A.M., Mostashari F., Balter S., Layton M. & Weiss D. 2006. Diarrheal illness detected through syndromic surveillance after a massive power outage: New York City, August 2003. *Am J Public Health*, **96**, 547-553.
22. Morgan O. 2004. Infectious disease risks from dead bodies following natural disasters. *Rev Panam Salud Publ*, **15**, 307-311.
23. Noji E.K. (ed.) 1997. Public health consequences of disasters. Oxford University Press, New York, 468 pp.
24. Noji E.K. 2005. Public health in the aftermath of disasters. *BMJ*, **330**, 1379-1381.
25. Pan American Health Organization (PAHO) 2004. Management of dead bodies in disaster situations. PAHO disaster manuals and guidelines series, No. 5. PAHO, Washington, 180 pp (www.paho.org/English/DD/PED/DeadBodiesBook.pdf accessed on 17 July 2008).
26. Qadri F., Khan A.I., Faruque A.S.G., Begum Y.A., Chowdhury F., Nair G.B., Salam M.A., Sack D.A. & Svennerholm A.M. 2005. Enterotoxigenic *Escherichia coli* and *Vibrio cholerae* diarrhea, Bangladesh, 2004. *Emerg Infect Dis*, **11**, 1104-1107.
27. Sack R.B. & Siddique A.K. 1998. Corpses and the spread of cholera. *Lancet*, **352**, 1570.
28. Saenz R, Bissell RA, Paniagua F. 1995. Post-disaster malaria in Costa Rica. *Prehospital Disaster Med*, **10**, 154-160.
29. Schneider E., Hajjeh R.A., Spiegel R.A., Jibson R.W., Harp E.L., Marshall G.A., Gunn R.A., McNeil M.M., Pinner R.W, Baron R.C., Burger R.C., Hutwagner L.C., Crump C., Kaufman L. Reef S.E., Feldman G.M., Pappagianis D. & Werner S.B. 1997. A coccidiomycosis outbreak following the Northridge, Calif, earthquake. *JAMA*, **277**, 904-908.
30. Spiegel P.B. 2005. Differences in world responses to natural disasters and complex emergencies. *JAMA*, **293**,1915-1918.
31. Sur D. 2000. Severe cholera outbreak following floods in a northern district of West Bengal. *Indian J Med Res*, **112**, 178-182.
32. Surmieda M.R., Lopez J.M., Abad-Viola G., Miranda M.E., Abellanosa I.P., Sadang R.A., Magboo F.P., Zacarias N.S., Magpantay R.L., White F.M., M.-L.G. Pascual, E.A. Tayag, F.C. Diza, M.-C.R. Roces, M.M. Dayrit & M.E. White 1992. Surveillance in evacuation camps after the eruption of Mt Pinatubo, Philippines. *MMWR CDC Surveill Summ*, **41**, 9-12 (erratum: *MMWR CDC Surveill Summ*, **41**, 963).
33. United Nations Cultural Scientific and Cultural Organization (Unesco) 2006. About natural disasters. Unesco, Paris (www.unesco.org/science/disaster/about_disaster.shtml accessed on 17 July 2008).
34. Vanasco N.B., Fusco S., Zanuttini J.C., Manattini S., Dalla Fontana M.L., Prez J., Cerrano D. & Sequeira M.D. 2002. Outbreak of human leptospirosis after a flood in Reconquista, Santa Fe, 1998 [article in Spanish]. *Rev Argent Microbiol*, **34**, 124-131.
35. Vollaard A.M., Ali S., van Asten H.A., Widjaja S., Visser L.G., Surjadi C. & van Dissel J.T. 2004. Risk factors for typhoid and paratyphoid fever in Jakarta, Indonesia. *JAMA*, **291**, 2607-2615.
36. Waring S.C., Reynolds K.M., D'Souza G. & Arafat R.R. 2002. Rapid assessment of household needs in the Houston area after tropical storm Allison. *Disaster Manag Resp*, Sep, 3-9.

37. World Health Organization 2005. Acute watery diarrhoea outbreak, Muzafarabad District. *WMMR*, **1**, 6 (www.who.int/hac/crises/international/pakistan_earthquake/sitrep/FINAL_WMMR_Pakistan_1_December_06122005.pdf accessed on 17 July 2008).
38. World Health Organization 2005. Epidemic-prone disease surveillance and response after the tsunami in Aceh Province, Indonesia. *Wkly Epidemiol Rec*, **80**, 160-164.
39. World Health Organization 2006. Acute jaundice syndrome. *WMMR*, **23**, 11-12 (www.who.int/hac/crises/international/pakistan_earthquake/sitrep/Pakistan_WMMR_VOL23_03052006.pdf accessed on 17 July 2008).
40. Yang H.Y., Hsu P.Y., Pan M.J., Wu M.S., Lee C.H., Yu C.C., Hung C.C. & Yang C.W. 2005. Clinical distinction and evaluation of leptospirosis in Taiwan – a case-control study. *J Nephrol*,**18**, 45-53.

