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Review

The ecology of emerging neurotropic viruses

Kevin J Olival[1] and Peter Daszak[2]

[1]*Department of Ecology, Evolution and Environmental Biology & Center for Environmental Research and Conservation, Columbia University, New York, New York, USA;* [2]*Consortium for Conservation Medicine, 460 w. 34 st., New York, NY 10001, USA*

The authors review common themes in the ecology of emerging viruses that cause neurological disease. Three issues emerge. First, 49% of emerging viruses are characterized by encephalitis or serious neurological clinical symptoms. Second, all of these viruses are driven to emerge by ecological, environmental, or human demographic changes, some of which are poorly understood. Finally, the control of these viruses would be enhanced by collaborative multidisciplinary research into these drivers of emergence. The authors highlight this review with a case study of Nipah virus, which emerged in Malaysia due largely to shifts in livestock production and alterations to reservoir host habitat. Collaboration between virologists, ecologists, disease modelers and wildlife biologists has been instrumental in retracing the factors involved in this virus's emergence. *Journal of NeuroVirology* (2005) **11,** 441–446.

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Introduction

Emerging infectious diseases (EIDs) are diseases that have recently expanded in geographic range, moved into new host species or populations, that have recently changed in pathology, incidence, or are caused by pathogens that have recently evolved (Lederberg *et al*, 1992; Morse, 1993; Smolinski *et al*, 2003). These diseases receive widespread attention in the scientific literature and popular press, but efforts to understand how they emerge and to predict and prevent their emergence are relatively poorly developed (Murphy, 1998). In this review, we highlight the developing field of emerging disease ecology and discuss how this approach can be used to understand emerging neurotropic viruses.

Emerging neurotropic viruses

Emerging viruses of the nervous system are on the rise (Johnson, 2003; Solomon, 2003a); however, the proportion of EIDs that are neurotropic has not been previously quantified. To determine the number of emerging viruses that are associated with encephalitis and severe neurological symptoms, we reanalyzed a list of all 1415 pathogens of humans (Taylor *et al*, 2001). Of these, 77 viruses can be classed as emerging using previously published criteria (Lederberg *et al*, 1992; Morse, 1993; Smolinski *et al*, 2003). Published data on clinical features were reviewed for each of the 77 viruses to determine whether or not neurological clinical symptoms were described. We grouped each of the 77 viral taxa into one of four categories (0, 1, 2, or 3) depending on the presence and frequency of reported neurological symptoms (Table 1).

Our analysis suggests that nearly half (49%) of all emerging viruses are characterized by encephalitis or serious neurological clinical symptoms. Consistent with the trend for all EIDs, 75% (58/77) of emerging viruses (Taylor *et al*, 2001) are zoonotic; and 80% (24/30) of our category 1 neurotropic viruses are zoonotic. These data suggest two crucial issues. First, that emerging viruses are an important issue for neurological medicine and second, that that understanding the process of zoonotic disease emergence will be key to dealing with emerging neurotropic viruses.

Ecological approaches to disease emergence

A common feature of almost all emerging diseases are the ecological, environmental or demographic factors that drive their emergence (Morse, 1993). These

Address correspondence to Peter Daszak, Consortium for Conservation Medicine, 460 w. 34 st., New York, NY 10001, USA. E-mail: Daszak@conservationmedicine.org

Table 1 Emerging viruses categorized by neurological clinical features

Category	*Severe neurological symptoms or encephalitis*	*Number of viruses*	*% of total emerging viruses*	*Examples of viruses for each category*	*Example references*
0	None	34	44%	Rotaviruses; Mayaro virus; Sin Nombre virus	(Kapikian and Chanock, 1995; Tesh *et al*, 1999; Vapalahti *et al*, 2003)
1	Commonly reported—primarily neurotropic viruses	30	39%	Nipah virus; West Nile virus; Australian bat Lyssavirus	(Chua *et al*, 2000; Lanciotti *et al*, 1999; Mackenzie and Field, 2004)
2*	Rarely or occasionally reported	8	10%	Hepatitis E; Influenza A; Oropouche virus	(Kejariwal *et al*, 2001; McCullers *et al*, 1999; Moreli *et al*, 2002)
3**	Unknown	5	7%	Wesselsbron; Salehabad; Sabio virus	(Traore-Lamizana *et al*, 2001); n/a

*This *excludes* cases where the vaccine not the natural etiological agent was responsible for neurological symptoms (e.g., hepatitis B vaccine).
**It should be noted that most of the unknown viruses are in the families *Flaviviridae* and *Bunyaviridae*, and thus may likely have neurotropic properties based on the etiology of more well-known, closely related viruses.

include changes to land use that alter transmission between wildlife reservoirs and humans, changes to weather patterns that alter vector abundance, or migration of people due to war or famine (Lederberg *et al*, 1992; Smolinski *et al*, 2003). A series of recent papers has focused on these ecological or environmental drivers to provide a new approach to understanding and ultimately predicting disease emergence (Daszak *et al*, 2001; Guernier *et al*, 2004; Taylor *et al*, 2001). In particular, database analysis of the 1415 pathogens known to affect humans has shown that 75% of emerging infectious diseases are zoonotic (Taylor *et al*, 2001). For zoonotic EIDs, emergence can be viewed as a three step process. First, environmental, ecological, or demographic factors alter host-pathogen dynamics in the reservoir host. This leads to increased transmission rates within or between populations or to the setting up of new routes of transmission from reservoir to human populations. This provides selective pressure on the wide biodiversity of pathogen types, strains, or species found in animal reservoirs, allowing the strains that are able to survive in the altered environment, or new host to emerge. By measuring and analyzing the ecological drivers, understanding how these alter pathogen dynamics in reservoir hosts, and measuring the background diversity of unknown potential zoonoses, it may be possible in the future to move towards prediction of unknown zoonotic EIDs.

A number of ongoing studies demonstrate this approach. For example, hunters and butchers of bushmeat are a high-risk group for the emergence of novel zoonoses. A focused study of bushmeat hunters in Cameroon has demonstrated for the first time, the presence of nonhuman primate retroviruses (simian foamy viruses) in bushmeat hunters (Wolfe *et al*, 2004). This study was designed around an informal predictive ecological approach, that proposed negative-stranded RNA viruses and retroviruses as the most likely group of new zoonotic agents, and increasing intensity of bushmeat hunting as an ecological and demographic change that is likely to allow these viruses to emerge in humans (Burke, 1998).

In most cases, the first step in this ecological approach is to identify and quantify the factors that drive emergence. In Table 2, we have given some examples of the ecological drivers associated with emerging neurotropic viruses. Thorough quantification of these factors, and a more detailed understanding of how they alter virus dynamics in animal reservoirs, and contact rates with people, will allow development of predictive approaches to emerging neurotropic viruses. At present, the lack of knowledge of the biodiversity of unidentified related viruses means that approaches can only be used successfully on single viruses (e.g., Nipah virus, see below), or groups of closely related, well-studied viruses (e.g., simian immunodeficiency viruses [SIVs]).

Ecology of Nipah virus emergence

Nipah virus (NiV), a neurotropic, zoonotic paramyxovirus was first identified during a swine and human outbreak in peninsular Malaysia in 1998 and 1999 (Chua *et al*, 2000). In pigs, the disease presented as primarily a respiratory syndrome. In humans, the virus was strongly neurotropic, causing encephalitis and ultimately killing 105/265 infected people who were mostly farmers and abattoir workers who came into close contact with infected pigs (Chua *et al*, 2000). NiV has serological, antigenic, phylogenetic, and natural history similarities to Hendra virus (HeV), an Australian paramyxovirus first described in 1994 that has since killed 16 horses and two humans (Field *et al*, 2001; Hooper *et al*, 2001; Murray *et al*, 1995; Wang *et al*, 2001). These similarities led to the classification of both viruses in a new genus, *Henipavirus*. Common features of NiV

Table 2 Examples of ecological factors causally associated with the emergence of neurotropic viruses

Virus (Family)	*Distribution*	*Animal hosts*	*Vectors*	*Dates of emergence*	*Associated ecological drivers*	*References*
Nipah (*Paramyxoviridae*)	Southeast Asia, South Asia subcontinent	Fruit bats; pigs; dogs, horses	None	1998–9 (Malaysia); 2001–4 (India, Bangladesh)	Livestock production and trade; fruit farming; deforestation; bat migration	(Chua *et al*, 2000; Chua *et al*, 2002a; Field *et al*, 2001; Kumar, 2003; Hsu *et al*, 2004)
Rift Valley fever (*Bunyaviridae*)	Africa, Arabian Peninsula	Domestic ruminants; rodents; possibly bats	Mosquitoes (*Culex*, *Aedes*, and *Anopheles* spp.); possibly sandflies	1930–1978 (sub-Sahara); 1978–1990s (N. Africa); 2000 to present (Arabia)	Increased rainfall; dam construction; increased vector abundance; livestock production and trade	(Bicout and Sabatier, 2004; Digoutte, 1999; Fagbo, 2002; Madani *et al*, 2003; Ringot *et al*, 2004; Shoemaker *et al*, 2002)
Toscana Sandfly fever (*Bunyaviridae*)	Italy, Southern Europe	Possibly gerbils	Sandflies (*Phlebotomus* spp.)	1971-present	Rural and suburban gardens; warming climatic conditions; increased vector abundance; human travel/tourism	(Braito *et al*, 1998; Dionisio *et al*, 2003; Mendoza-Montero *et al*, 1998; Valassina *et al*, 1998, 2003)
West Nile (*Flaviviridae*)	Middle East, Europe, and Asia; North and Central America	Many bird, mammal, and reptile species	Mosquitoes (especially *Culex* and *Aedes* spp.)	1999-present (Americas)	Drought; increased rainfall; increased vector abundance; bird migration; increased airplane travel	(Epstein and Defillipo, 2001; Kilpatrick *et al*, 2004; Lanciotti *et al*, 1999; Marra *et al*, 2004; Rappole and Hubalek, 2003)
Kyasanur Forest Disease (*Flaviviridae*)	India	Monkeys; shrews	Ixodid Ticks (*Haemaphysalis spinigera)*	1957 to present	Drought, deforestation; habitat encroachment; increased contact with monkeys	(Adhikari Prabha *et al*, 1993; Das, 2004; Samuel and Prabhu, 1996; Saxena, 1997; Sreenivasan *et al*, 1986)
Japanese encephalitis (*Flaviviridae*)	Southeast Asia, South Asia, China; Pacific Rim, Northern Australia	Pigs; birds; other vertebrates	Mosquitoes (especially *Culex* spp.)	1935 to present; 1998 (Australia)	Rice farming; increased rainfall; increased vector abundance; livestock production	(Bi *et al*, 2003; Endy and Nisalak, 2002; Mackenzie *et al*, 2002; Nga *et al*, 2004; Solomon, 2003b; Sunish and Reuben, 2002)

with HeV also allowed for more targeted wildlife surveillance during the initial outbreak investigation in Malaysia. Subsequent virus isolation studies have confirmed that fruit bats or flying foxes (*Pteropus* spp.) are the most important natural reservoir host for NiV and HeV (Chua *et al*, 2002b; Halpin *et al*, 1999). Pigs appear to act as amplifying hosts, and there were no confirmed cases of direct transmission between bats and humans in Malaysia. The mechanisms that drove these species jumping events remain unknown. However, molecular studies of NiV suggest that it is an "old" virus that has not undergone any recent evolutionary changes (Field *et al*, 2001; Wang *et al*, 2001), therefore as with most EIDs, ecological factors, not evolutionary change, likely played the major role (Schrag and Wiener, 1995).

In 2001, an international team of ecologists, wildlife biologists and virologists began a research project to understand what factors led to the emergence of Nipah virus. The ultimate goal of this research is to develop predictive models to predict risk of future outbreaks in southeast Asia (see http://www.henipavirus.org). Two leading hypotheses were generated and are currently being tested. (1) Changes in the number, density, or management of pigs produced in Malaysia allowed a virus that is commonly introduced into pigs to persist endemically and emerge in humans. (2) Broad-scale deforestation in Malaysia and Sumatra coupled with the large El Ñino Southern Oscillation (ENSO)-related drought during 1997 altered the migration routes and feeding behavior of *Pteropus* spp. reservoirs and led to the introduction of this virus into pigs for the first time (Chua *et al*, 2002a).

To test these hypotheses, we have set up collaborations between scientists that study wildlife migration and diseases (veterinarians and wildlife biologists), mathematical modelers of viral dynamics in populations, Malaysian government workers who manage pig production data, and virologists who test

samples for NiV and perform experimental infections with this BSL-4 agent. The route of introduction of virus into pigs appears to be related to practice of growing fruit trees adjacent to piggeries, which allows efficient use of pig waste as fertilizer for trees. At the index farm for Nipah virus, mango and rambutan orchards were present around the 30,000-head farm at the time of the outbreak, with some trees overhanging pig pens (Chua *et al*, 2002a). This is the most likely factor responsible for the proximate "spill over" event, as pigs were probably exposed to virus-laden urine and masticated fruit pellets dropped from feeding flying foxes (Chua *et al*, 2002a).

Using satellite telemetry, we have found that the primary reservoir for NiV, the large fruit bat *Pteropus vampyrus*, travels large distances and that migratory patterns are driven by seasonal food abundance (Spencer *et al*, 1991). Our approach now is to map the distribution of fruit bat colonies, the population changes over time at these colonies and changes in NiV serology. In addition, population genetics studies are underway to infer historical migratory patterns and corroborate satellite telemetry data on population connectivity and dispersal. This will allow us to develop a predictive model for the presence of NiV-infected bats in Malaysia over flowering and fruiting seasons. We have found colonies of fruit bats relatively close (within 50 km) to the index farm, suggesting that the virus was present in bats foraging at the index farm year-round. The timing of the first human cases of Nipah virus infection predates the large ENSO event of 1997, suggesting that the virus was introduced to pig populations previously. Importantly period over 18 months elapsed between the first case of NiV and the large outbreak in 1999 (Chua *et al*, 2000b), suggesting that the virus maintained itself in the pig population at the index farm. We have revisited the management data for this farm and developed a simple model to describe NiV dynamics. Our analyses show that it is highly likely that the size of the farm, and the way the pigs were intensively managed (i.e., high turnover of piglets) allowed the virus to persist (Pulliam *et al*, unpublished data). The large scale outbreak of NiV in 1999 was likely caused by changes in pig farm management following repeated deaths, i.e., selling of farms and transport of infected pigs to other farms in this region and in the South of Malaysia.

This study demonstrates how collaborative approaches to the ecology of emergence can allow a fuller understanding of how viruses emerge through complex biological systems. For NiV, this system involves three host groups: bats, pigs, and humans; environmental changes including climate and deforestation; and viral dynamics within migrating species and dense populations of terrestrial domestic animals. We aim to use the understanding of NiV emergence to predict risk of future virus emergence in Malaysia, but also future emergence of unknown, related viruses. To do this, we are collecting samples from fruit bat reservoirs across their Indo-Pacific range, and attempting to identify currently unknown Henipaviruses. We will then be able to predict high risk areas globally for the emergence of these viruses by identifying the convergence of reservoir hosts and expanding livestock production.

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