

population approximately every two years, following periods of waning immunity. Adults drive outbreaks in bats and appear to become susceptible to reinfection after  $\sim 7$  years. Phylogenetic analysis of N, P, and G gene regions from the same population over time showed >98% sequence homology. A divergent strain of NiV ( $\sim 80\%$  homology) was identified in eastern Bangladesh.

**Conclusion:** *Pteropus medius* is the reservoir for Nipah virus in Bangladesh and likely to be an ongoing source of human infection. NiV detection was not restricted to the Nipah Belt, suggesting spillover is possible anywhere in Bangladesh if a suitable strain and bat-human interface were present. Different strains in disparate locations and high homology in one location over time, suggests that there may be localized strains persistently circulating in bats. Human activities such as date palm sap harvesting, concurrent with viral circulation in local bat populations are likely to be the major driver of human outbreaks in Bangladesh. Viral dynamics in bats, which include years with no outbreaks, may explain years when no human NiV cases have been detected.

<http://dx.doi.org/10.1016/j.ijid.2016.11.056>

10.009

**Global correlates of emerging zoonoses: Anthropogenic, environmental, and biodiversity risk factors**



T. Allen<sup>a,\*</sup>, K. Murray<sup>b</sup>, C. Zambrana-Torrel<sup>c</sup>, S. Morse<sup>d</sup>, C. Rondinini<sup>e</sup>, V. Di Marco Lo Presti<sup>f</sup>, K. Olival<sup>g</sup>, P. Daszak<sup>g</sup>

<sup>a</sup> EcoHealth Alliance, Research Technology, New York/US

<sup>b</sup> Imperial College London, The Grantham Institute for Climate Change, London/UK

<sup>c</sup> EcoHealth Alliance.org, New York, NY/US

<sup>d</sup> Columbia University, New York, NY/US

<sup>e</sup> Sapienza Università di Roma, Global Mammal Assessment programme, Rome/IT

<sup>f</sup> Istituto Zooprofilattico Sperimentale of Sicily, /, Barcellona P.G. (Messina)/IT

<sup>g</sup> EcoHealth Alliance, New York, NY/US

**Purpose:** Human infectious diseases originating from wildlife represent a significant threat to global health, security and economic growth. Efforts to identify the geographic origins and underlying causes of disease emergence are essential to move interventions closer to the source, more effectively limiting subsequent impacts.

A previous study (Jones et al., 2008) used logistic regression to model the association between “EID events” and various factors, and found different distribution and driver associations for different categories of EID event.

We aim to better analyze the mechanistic underpinnings of disease emergence and address some methodological limitations of previous work. We focus on zoonotic EIDs and predictor datasets for specifically hypothesized mechanisms of emergence.

**Methods & Materials:** We used boosted regression trees to model associations between an updated set of zoonotic EID events and spatial predictors, selected for their relevance to a priori hypotheses about mechanisms of emergence. We included improved measures of mammal species richness, land use, land-use change and land cover. We constructed a novel measure of relative publication effort as a proxy for observation bias, and used a bootstrap resampling regime to account for spatial uncertainty in EID event data.

**Results:** Biodiversity, land cover and land use were the most important factors in predicting locations of disease emergence events, after accounting for observation bias and the baseline distribution of the human population. We found that disease emergence was more likely in areas of high mammal biodiversity and heavily forested areas. Weaker, but still important, factors included high levels of urbanization, and rapid land conversion to and from pasture.

**Conclusion:** The global distribution of zoonotic EID risk (EID ‘hotspots’) is concentrated in tropical regions where wildlife biodiversity is high, human populations dense and growing, and land use change is occurring rapidly. These regions are most likely to produce the next EID event, and therefore most valuable for surveillance in wildlife, livestock or people.

Directions for future research include: fitting and pooling separate models for different diseases; ‘ground truthing’ using data from wildlife to measure factors such as pathogen diversity and human contact with wildlife.

<http://dx.doi.org/10.1016/j.ijid.2016.11.057>

**10.010 Prevalence and risk factors of seropositivity to *C.burnetii* infection in dairy farms and dairy farmers, Chiang-Mai, Thailand 2015**



P. Doung-Ngern<sup>a,\*</sup>, P. Padungtod<sup>b</sup>, M. Emch<sup>c</sup>, D. Weber<sup>d</sup>, G. Kersh<sup>e</sup>, G. Koch<sup>f</sup>, S. Meshnick<sup>d</sup>

<sup>a</sup> Bureau of Epidemiology, Department of Disease Control, Mueang, NONTHABURI/TH

<sup>b</sup> Thai-MOPH - US.CDC, Global Disease Detection Regional Center, Nonthaburi/TH

<sup>c</sup> University of North Carolina Chapel Hill, Geography, Chapel Hill, NC/US

<sup>d</sup> University of North Carolina at Chapel Hill, Epidemiology, Chapel Hill/US

<sup>e</sup> Center for Disease Control and Prevention, USA, Rickettsial Zoonoses Branch, National Center for Emerging Zoonoses and Infectious Diseases, Atlanta/US

<sup>f</sup> University of North Carolina at Chapel Hill, Biostatistics, Chapel Hill/US

**Purpose:** A one year longitudinal study of Q fever among dairy farms and farmers was conducted in June 2015 in the areas of Chiang-Mai where Q fever was reported. This study was conducted by the collaboration between public health and animal health sectors. We reported a preliminary analysis of baseline information to describe the magnitude and factors associated with *C.burnetii* infection in this high risk population.

**Methods & Materials:** Two-stage random sampling of the farms and farmers was performed to identify cohort of dairy farms farmers. We conducted face to face interview with farmers, and collected blood for baseline assessment. Bulk tank milk samples from each farm were screened and specimens were collected from cows, other animals, and farm environment in the farms with milk positive. Farmer sera were tested using Indirect Immunofluorescence Assay (IFA). Milk and cow sera were tested using Enzyme-Linked Immunosorbent Assay. Vaginal swabs and environmental samples were tested using Polymerase Chain Reaction. Descriptive statistics and multivariate logistic regression were performed to describe baseline seroprevalence and factors associated with milk positive. This cohort of farms was followed up at 6 and 12 month after the baseline assessment.