

symposium

• AT THE ZOOLOGICAL SOCIETY OF LONDON • ORGANISED IN PARTNERSHIP WITH
THE ROYAL SOCIETY AS PART OF ITS 350TH ANNIVERSARY CELEBRATIONS IN 2010 •

DISEASE INVASION: IMPACTS ON BIODIVERSITY AND HUMAN HEALTH

Thursday 18 and Friday 19 November 2010

Organised by **Andrew Cunningham**
Peter Hudson FRS
Andrew Dobson

The Meeting Rooms
The Zoological Society of London
Regent's Park
London NW1 4RY

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images (from top): *Ixodes pacificus*, James Gathany, CDC; brain tissue showing amyloid plaques in a case of vCJD, Sherif Zaki, MD PhD, and Wun-Ju Shieh, MD PhD MPH, CDC; *Aedes aegypti*, James Gathany, CDC; Foot and mouth disease sign © 2001, Ben Gamble; graphical representation of influenza virion ultrastructure, Dan Higgins, CDC; face masks © iStockphoto.com/mammamaart; *Phlebotomus sp. Fly*, CDC/WHO



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LIVING CONSERVATION

9.00–9.15 **Welcome from Sir Patrick Bateson FRS**
*President, ZSL, and Emeritus Professor of Ethology, University of Cambridge;
former Biological Secretary and Vice-President of the Royal Society*

9.15–10.00 **Keynote address**
Wildlife disease: from the science of threats to the policy of mitigation
*Roy Anderson FRS, Department of Infectious Disease Epidemiology,
School of Public Health, Imperial College London*

Humans have acquired most of their infectious diseases from their close primate relatives, wildlife in general, and livestock in particular. Examples include HIV-1, SARS, West Nile virus and the recent pandemic of H1N1 where the viral strain contained genetic elements from bird and pig influenza viruses. It has been estimated that roughly 75% of the new diseases that have affected humans over the past 10 years have been caused by pathogens originating from mammals or birds, or from products of animal origin. Many of these diseases have had the potential to spread through human communities on a global basis and to become either pandemic or endemic.

Well known animal diseases that are transmitted to humans (i.e. zoonoses), such as rabies, brucellosis, leishmaniasis and echinococcosis, occur in many countries, especially in the developing world where they mostly affect the poor. However, zoonotic infections are also major problems in developed countries with intensive livestock production in densely populated regions.

The lecture will examine the recent history of emerging infectious diseases in human communities and the factors in our changing world that facilitate emergence and establishment. Ecological theory will be used to provide some general insights into the processes that promote emergence and persistence of zoonotic infections. Of the recent emerging infections of zoonotic origin, two will be examined in detail, namely SARS and H1N1. The lessons learnt from the global response to these two contrasting viral infections, and the mitigation strategies adopted, will be discussed.

10.00–10.30 **POSTER SESSION (TEA/COFFEE)**

SESSION I: DISEASE AS A THREATENING PROCESS

Chair: Hamish McCallum (Griffith University, Queensland, Australia)

10.30–11.00 **The eye of the finch: systems analysis of an emerging avian pathogen**

Andy Dobson¹, Erik Osnas¹, Dana Hawley², Andre Dhondt³, Keila Dhondt³, Wesley M. Hochachka³, Irby Lovette³, Evan Cooch³, Karel A. Schat³, Priscilla H. O'Connell³, Jessica L. Grodio³, Jonathan C. DeCoste³, Sarah States³, Paul Hurtado³, David Ley⁴, Ken Pollock⁴, Steve Greay⁵, Edan Tulman⁵ and Frasca Salvatore⁵

¹EEB, Princeton University; ²Virginia Tech University; ³Cornell University;

⁴North Carolina State University; ⁵University of Connecticut

Understanding the biology of disease emergence requires an understanding of disease dynamics at all scales from molecular to population and community levels. We very rarely get an opportunity to study emerging pathogens at all these scales as we are usually madly rushing to control the disease outbreak. Occasionally we get lucky and a disease outbreak occurs in a species and no one intervenes, if we monitor these epidemics we can obtain valuable information on the different stages of emergence. The arrival of mycoplasmal conjunctivitis (MG) in house finches in the Eastern US presented an important opportunity to monitor such an event. The Laboratory of Ornithology at Cornell University has monitored house finch populations since shortly after the pathogen emerged in the fall of 1993. It has now spread across the entire US and is working its way down the coast of California. In this talk we present results of work by a large team of ecologists, ornithologists, pathologists and veterinarians that have examined the dynamics of MG emergence from the scale of selection for the genomic base-pairs that code for attachment of the bacteria, through the immunology and pathology of interactions within the eyes of individually infected birds, to the population and community level consequences of pathogen emergence. The different scales of empirical study are connected by mathematical models of the interaction between host and pathogen; the whole provides important insights into the mechanisms that are far less frequently observed in other pathogen outbreaks.

11.00–11.30 **Host heterogeneity in the transmission of multi-host pathogens: a challenge and an opportunity for control**

A. Marm Kilpatrick, University of California, Santa Cruz, USA

Heterogeneity among hosts in infectiousness and contact rates can have large effects on the transmission and control of pathogens. In extreme cases a few individuals give rise to the majority of secondary infections, which have been termed super spreading events. For multi-host pathogens heterogeneity is the rule, and variation in host communities can exert strong influences on pathogen transmission. I will present data from several multi-host pathogens, including West Nile virus and Lyme disease, that quantify the impact of host heterogeneity on transmission. I will also show how details of the natural history of hosts, vectors, and pathogens enable invasion and intense transmission, and how we can take advantage of knowledge of the ecology of transmission to identify hotspots and even control disease. The recent emergence of many multi-host zoonotic pathogens highlights the importance of understanding and taking advantage of host heterogeneity to minimize impacts on wildlife and human health.

11.30–12.00 **Spillover, stuttering transmission, and the emergence of human monkeypox**

James O. Lloyd-Smith, University of California, Los Angeles, USA

Zoonoses are the predominant source of new human infectious diseases but there are remarkably few quantitative studies of the dynamics of zoonotic pathogens as they spill over from animals to humans and begin transmitting in the human population. Such analyses are crucial to understanding changing patterns of infection, projecting future public health impacts, and designing rational control policies that utilize limited resources to efficiently improve health outcomes. A conspicuous gap in current knowledge pertains to pathogens that exhibit limited transmission among humans—enough to cause a cluster of cases, but not enough to trigger an epidemic. Such ‘stuttering chains’ of transmission are red flags for emergence risk, since the barrier of human-to-human transmission has been breached, and establishment of endemic circulation is simply a matter of degree, or of environment. Stuttering chain events foreshadowed the emergence of SARS-Coronavirus, and have raised grave concerns for H5N1 influenza and Nipah virus, but have proven extremely difficult to study owing to their rare and transient nature.

This talk will address the challenge of modeling cross-species spillover and stuttering chains of weakly-transmitting zoonoses, focusing on the example of human monkeypox in the Democratic Republic of the Congo (DRC). Since the eradication of smallpox thirty years ago, an increasing proportion of the human population lacks immunological protection from smallpox or its vaccine, and as a consequence lacks cross-protective immunity to other orthopoxviruses. We analyze recent and historical epidemiological data to understand how declining population immunity to smallpox is enabling the emergence of human monkeypox in DRC. We project the rising burden of monkeypox infections and estimate the probability that monkeypox will cross the threshold enabling persistent circulation in the human population. Focusing on the contribution of human-to-human transmission against a background of zoonotic spillover, we use our model to dissect the possible drivers of the dramatic rise in monkeypox incidence detected in recent surveillance data. Our analysis reveals general principles about the dynamics of emerging zoonoses as transmissibility among humans increases, and about optimal targeting of surveillance and control activities.

12.00–12.30 **Genes, bugs and wildlife: the genetics of pathogen susceptibility**

Karina Acevedo-Whitehouse, Institute of Zoology, ZSL, Regent's Park, London NW1 4RY

The loss of genetic diversity, in particular that of functional genetic traits, decreases the evolutionary potential of a species or population by compromising their ability to respond to new challenges. Pathogens are one of such challenges, acting as evolutionary and demographic drivers of natural populations by affecting host survival and reproductive success. The strong coevolutionary relationship between pathogens and their hosts has helped shape and maintain genetic diversity, which in turn has influenced disease dynamics. In this light, it is pertinent and important to increase our understanding of the genetic basis of variations of the immune response of natural populations. Traditionally, such research has focussed on the major histocompatibility complex (MHC), a highly polymorphic genetic region involved in antigen-presentation that shows strong evidence of selection. However, the immune system is regulated by a large and extremely complex network of various genes, in addition to the MHC. This complexity poses diverse problems when attempting to identify or select candidate genes to study. One way that this can be partly circumvented is by focusing on neutral genetic variation. A number of studies have shown that aspects of fitness, including resistance to

pathogens, are significantly correlated with multilocus heterozygosity (known as Heterozygosity-Fitness Correlations, HFC). The basis for such associations is theorized to be due to (1) inbreeding depression—thought to affect homozygosity across the genome, increasing the chance of deleterious mutations being expressed, or (2) associative overdominance, where one or more of the genetic markers used to estimate heterozygosity is linked to a gene involved with the trait studied. As inbreeding depression is a rare event, it is likely that a large proportion of reported HFC are actually due to associative overdominance, thus potentially allowing the identification of key “immune” genes. Using this approach it has been possible to detect genetic regions likely to be driving variations in resistance to a number of infectious diseases that affect wildlife, including bovine tuberculosis in wild boar, hookworm infections in pinnipeds and herpesvirus-related cancer in sea lions. By focusing research on these putative targets we will be able to increase our understanding of these and other infectious conditions and enable a broader understanding of the levels at which natural selection can act on immunity. At a time when novel pathogens are increasingly emerging in natural populations, this approach may prove to be important to understanding disease dynamics and assessing epidemic risks.

12.30–13.30 **LUNCH**

SESSION II: ANTHROPOGENIC DRIVERS OF DISEASE EMERGENCE

Chair: Tony Sainsbury (Institute of Zoology, ZSL)

13.30–14.00 **The emergence and spread of finch trichomonosis**

Andrew A. Cunningham¹, Becki Lawson¹, Mike Toms² and Rob Robinson²

¹Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY, UK; ²British Trust for Ornithology, The Nunnery, Thetford, Norfolk IP24 2PU, UK

Trichomonosis is a disease of birds, caused by the protozoan parasite, *Trichomonas gallinae*, and characterised by necrotic ingluvitis. This disease has long been recognised in Columbiformes, Falconiformes and Strigiformes, as evidenced by the historic names of canker or roup (when affecting pigeons and doves) and frounce (when affecting birds of prey). It has until now, however, not been recognised in other avian orders. In 2005, trichomonosis was diagnosed as an emerging infectious disease in Passeriformes, when cases were detected in garden birds by the Garden Bird Health *initiative* (GBHi), a citizen science avian disease surveillance project in Great Britain. Morphological and molecular analyses confirmed the parasite as *Trichomonas gallinae*. In 2006, finch trichomonosis reached epidemic proportions throughout western and central England, with greenfinch *Carduelis chloris* and chaffinch *Fringilla coelebs*, two of the most common British garden bird species, being most affected. Losses were so great that, using data from the long-standing BTO/JNCC/RSPB Breeding Bird Survey (BBS), 2007 declines of up to 35% of breeding greenfinch populations and of up to 21% of breeding chaffinch populations were detected. There was a strong spatial association between the severity of declines in 2007 and the apparent incidence of the disease the previous year. Seasonal (summer/autumn) epidemic mortality continued in subsequent years, with an apparent eastwards and northwards spread in Great Britain. Again, greenfinch and chaffinch were

primarily affected, although a wide range of other passerines were found to be susceptible to the disease. In summer 2008, finch trichomonosis incidents were first diagnosed in southern Fennoscandia and the disease has since spread into other areas of continental Europe. Genetic comparison of British and Fennoscandian parasites found no sequence variation between regions examined, the ITS1/ 5.8S/ ITS2 region and part of the 16S rRNA gene, supporting an hypothesis of spread from Great Britain. We assessed bird migration as a mechanism of this spread using historical ring return data and identified migrating chaffinches from eastern England as the most likely vector of the parasite to Fennoscandia. Retrospective analyses of BBS and other citizen science data to further understand the initial emergence and spread of finch trichomonosis will be presented.

14.00–14.30 **Human-wildlife contact and emerging viral zoonoses from bats**

James Wood¹, Andrew Cunningham², Richard Suu-Ire³, Anthony R. Fooks⁴, Andy Alhassan⁵, David Hayman^{1,2,4}, Alison Peel^{1,2}, Kate Baker^{1,2}, Alexandra Kamins^{1,2}, Marcus Rowcliffe², Stephen Rossiter⁶, David Sargan⁷, Pablo Murcia¹, Paul Kellam⁸, Melissa Leach⁹ and Yaa Ntiamo-Baidu¹⁰

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⁹STEPS Centre, Institute of Development Studies at University of Sussex;

¹⁰University of Ghana, Legon

A number of diseases of humans have resulted from newly emerging pathogens of bats in the last 20 years. Some of these diseases have had a devastating impact on the communities affected, occasionally on a global scale. The list is not long, but includes some notable examples, including Severe Acute Respiratory Syndrome coronavirus (SARS CoV), Hendra virus (HeV) and Nipah virus (NiV). Research over the same time period has clarified that other significant diseases probably have their origins in bats, including the filoviruses Ebola and Marburg in Africa and rabies and other lyssaviruses.

In some cases, it appears that the virus infections have emerged in humans by direct spread from bats to humans or via simple fomite transmission, whereas in other cases the transfer has involved an intermediate amplifying host. The SARS epidemic spread probably from horseshoe bats to humans via palm civets that were being traded in Southern China; following human infection there was widespread human-to-human transmission until the disease was controlled. In all human outbreaks of HeV in Australia and the initial human outbreaks of NiV in Malaysia and Singapore, the infection spread from large *Pteropus* bats (so called “flying foxes”) to horses and to pigs and thence into humans. In subsequent outbreaks of NiV, particularly in Bangladesh, there has been direct bat-to-human transmission, perhaps through the consumption of fruit or palm sap with infected bat urine or saliva.

These few incidents have resulted in an understanding that bats may, similar to other mammalian groups, harbour substantial numbers of viruses (and other microbes) that are potential pathogens in humans.

While SARS appeared to be genuinely novel, it is not completely clear that sporadic transmission of other viral zoonoses, such as NiV, had never occurred before the 1990s. Certainly, filoviruses and rabies viruses have been infecting humans for some time.

Global changes are likely to act as a driver for further emergence of bat-associated pathogens. Ever-increasing encroachment of humans into forested and other regions, and habitat destruction, are likely to result in greater contact between humans and bats. Tourism and eco-tourism (particularly when caves housing large numbers of bats are promoted as interesting sites to visit) and economic activities (such as mining) may also play a role in increasing bat-human contact and hence disease transmission. Our studies are particularly focused in Ghana, where at least 100,000–200,000 fruit bats, which we know to be infected with a range of viruses, are eaten by humans.

14.30–15.00 **Highly pathogenic avian influenza H5N1: threats to biodiversity and human health**

Thijs Kuiken, Department of Virology, Erasmus Medical Center, Rotterdam, The Netherlands; and Artemis Research Institute for Wildlife Health in Europe, Utrecht, The Netherlands

Emerging pathogens, often from wildlife, threaten the health of wild animals themselves, domestic animals, and humans. An example of such a pathogen is highly pathogenic avian influenza virus (HPAIV) of the subtype H5N1. In recent years we have studied several unusual aspects of this virus.

First, spread of HPAIV among poultry flocks is traditionally thought to occur by transport of infected poultry, contaminated equipment, and people associated with the poultry industry. The spread of HPAIV H5N1, however, also has implicated wild birds. Our analysis of outbreaks of HPAIV H5N1 infection in waterbirds in Europe during the winter of 2005–2006 indicated that congregation of waterbirds along the 0 °C isotherm likely contributed to the spread and geographical distribution of these outbreaks. Experimental HPAIV H5N1 infections in six wild duck species showed that some species, particularly mallards, can potentially be long-distance vectors of HPAIV H5N1 and that others, particularly tufted ducks, are more likely to act as sentinels. Experimental HPAIV H5N1 infections in red knots indicated that birds in migratory condition may shed more HPAIV H5N1 and be more susceptible to severe disease, thus slowing yet not abrogating the geographical spread of HPAIV H5N1 by wild migratory birds.

Second, HPAIV H5N1 virus infection results in disease and mortality in multiple domestic and wild carnivore species that were not previously known to become ill from influenza A virus infection. Experimental HPAIV H5N1 infections in cats and foxes demonstrated that HPAIV H5N1 virus can productively infect carnivores, resulting in clinical disease or death. Virus replicated not only in the respiratory tract but also in multiple extra-respiratory tissues, and was associated with severe necrosis and inflammation. By direct inoculation of HPAIV H5N1 into the intestine, we confirmed the intestine as a portal of entry in cats and showed that the cell tropism of HPAIV H5N1 in cats infected via the intestine was different from that in cats infected via the trachea, targeting endothelial cells instead of parenchymal cells.

Third, HPAIV H5N1 is the first-known influenza virus to cause lower respiratory tract disease in humans. This contrasts with the idea that avian influenza viruses generally have little affinity for human respiratory tissues. We determined that HPAIV H5N1 virus attachment in the human respiratory tract is progressively more abundant towards the alveoli, where the virus attaches

predominantly to type II pneumocytes and alveolar macrophages. This attachment pattern fits with the limited pathology data on HPAIV H5N1 virus infection in humans, which show diffuse alveolar damage as the primary lesion and infection of type II pneumocytes.

Fourth, it has been speculated that the inefficiency of human-to-human HPAIV H5N1 transmission is determined in part by its lack of tropism for the human upper respiratory tract. We found that influenza viruses that are transmitted efficiently among humans attach abundantly to human upper respiratory tract, whereas inefficiently transmitted influenza viruses attach rarely. This suggests that the ability of an influenza virus to attach to human upper respiratory tract is a critical factor for efficient transmission in the human population. To determine the risk of HPAIV H5N1 evolving efficient transmission, we introduced all previously described amino acid substitutions and combinations thereof into a single genetic background and tested the receptor specificity of these 27 mutant viruses. At least three mutant viruses showed an attachment pattern to the human respiratory tract similar to that of the human H3N2 virus. These data show that HPAIV H5N1 potentially requires only a single amino acid substitution to acquire human receptor specificity, while at the same time remaining replication competent, thus suggesting that the pandemic threat posed by HPAIV H5N1 is far from diminished.

15.00–15.30 **The impact of climate change on disease emergence: lessons from the Arctic**

Susan J. Kutz, Bryanne Hoar and Alessandro Massolo

*Department of Ecosystem and Public Health, Faculty of Veterinary Medicine,
University of Calgary, Calgary, Alberta, Canada*

Climate change at northern latitudes is resulting in dramatic perturbations in the biotic and abiotic environment, including changes in the ecology of infectious diseases. Host-parasite interactions are highly sensitive to climate change and general predictions include reduced generation times and increased diversity, abundance and severity of pathogens in northern wildlife. During the past decade several examples of climate-linked parasite and/or disease emergence have been reported for arctic ungulates. The response of these host-parasite systems to climate change is complex, not uniform across taxa, and not always intuitive. For example, consistent with general climate change predictions, the generation time has decreased and the geographic range has expanded for *Umingmakstrongylus pallikuukensis*, the protostrongylid lungworm of muskoxen. Contrary to predictions, however, recent research on *Ostertagia gruehneri*, the Trichostrongylid abomasal nematode of caribou, suggests that generation times will be unaffected by climate change and increased temperatures could actually reduce availability of infective larvae at certain times of the year. We use these and other case studies of endemic and invasive northern parasites to explore the impacts of climate change on host-parasite interactions at high latitudes and discuss insights relevant to a broader understanding of parasite ecology.

15.30–16.00 **POSTER SESSION (TEA/COFFEE)**

SESSION III: MULTI-PATHOGEN IMPACTS ON DISEASE TRANSMISSION

Chair: Andrew Dobson (Princeton University, USA)

16.00–16.30 **Parasite species diversity: causes and consequences at multiple scales**

*Charles Mitchell, Department of Biology, University of North Carolina
at Chapel Hill, Chapel Hill, NC 27599-3280, USA.*

To test general theories of the causes and consequences of parasite biodiversity, I use plant pathogens as a model system. I will present recent results focused on two scales, biogeographic ranges and individual host organisms.

Many individual host organisms are simultaneously coinfecting by multiple parasite species. Further, most parasites infect multiple host species, so transmission between host species may influence coinfection rates, and coinfections may modulate between-species transmission. Theory from ecoimmunology predicts that hosts with quick-return physiologies/fast-living life-histories will function as parasite reservoirs. We tested this hypothesis experimentally at the individual host level, using an insect-vectoring plant virus. As predicted, quick-return hosts were more susceptible to infection, supported greater vector reproduction, and infected a greater fraction of foraging vectors. In order to mechanistically understand the causes and consequences of coinfection, we are currently extending this work to experimental multi-host, multi-pathogen communities in the field, coupled with mathematical models. We have also examined the scaling of parasite species richness, from individual hosts to a geographic region, by measuring the prevalence of four cereal and barley yellow dwarf viruses (C/BYDVs) along a 2000 km latitudinal gradient. C/BYDVs are insect-vectoring RNA viruses that cause one of the most prevalent of all plant diseases worldwide. The number of viruses in each infected host (alpha diversity) increased roughly two-fold across the latitudinal transect. This increase in coinfection corresponded with a decline in among-host pathogen turnover (beta diversity), suggesting that viruses in northern populations experience less transmission limitation than in southern populations.

At biogeographic scales, introduced species escape many pathogens and other enemies, raising three questions: (1) How quickly do introduced hosts accumulate pathogen species? (2) What factors control pathogen species richness? (3) Are these factors the same in the hosts' native and introduced ranges? We analyzed fungal and viral pathogen species richness on 124 plant species in both their native European range and introduced North American range. Hosts introduced 400 years ago supported six times more pathogens than those introduced 40 years ago. In hosts' native range, pathogen richness was greater on hosts occurring in more habitat types, with a history of agricultural use, and adapted to greater resource supplies. In hosts' introduced range, pathogen richness was correlated with host geographic range size, agricultural use, and time since introduction, but not any measured biological traits. Introduced species have accumulated pathogens at rates that are slow relative to most ecological processes, and contingent on geographic and historic circumstance.

16.30–17.00 **Effects of concurrent infection on host susceptibility and survival**

Mike Begon¹ and Sandra Telfer²

¹The University of Liverpool, UK; ²University of Aberdeen, UK

Interactions between parasites in natural populations have been studied only rarely. This is regrettable as it is likely that most hosts, including humans, at most periods, are infected with more than one parasite species, either simultaneously or sequentially. Is there a structure to these parasite communities, or are they just an assemblage of independent species? If there are patterns of coinfection, do they arise because infection by one parasite species affects susceptibility to others or because of inherent differences between hosts? And if parasites do affect one another, how powerful and important are these effects? We have collected a large data set on microparasites from individual field vole hosts, sampled frequently and over several years from natural populations. Inherent differences between hosts (their 'condition') do affect their probabilities of infection, but over and above this, we find that the parasites do indeed constitute an interactive network. We show that there are large positive and negative effects of other infections on the risk of being infected by any given pathogen and discuss possible mechanisms behind these effects: competition for host resources and indirect interactions via the host's immune system. Indeed, the effects are typically of greater magnitude, and explain more variation in infection risk, than the effects more commonly considered in disease studies, such as age, sex or season. There appear, moreover, to be comparable effects on the length and intensity of infection, and we discuss, too, the evidence for interactive effects between pathogens in their effects on host fitness (survival). The results highlight the dangers of the common practice of considering parasite species in isolation rather than looking at whole parasite communities. If we wish to know the risks to an individual of contracting one infection, we need to know what other infections it has. If an infection appears to be having an adverse effect on a host, that may be because of the other infections that accompany it.

17.00–17.30 **Discussion period**

17.30–18.45 **POSTER SESSION with cash bar**

18.45 **End of Day One**

19.00–21.00 **Symposium dinner for speakers and guests with tickets**

**SESSION IV: PATHOGEN DYNAMICS IN A MULTI-HOST SYSTEM
(INSIGHTS INTO MULTI-HOST DISEASE DYNAMICS)**

Chair: Tony Sainsbury (Institute of Zoology, ZSL)

9.00–9.30 **Genetic diversity of avian influenza viruses driven by fast-slow dynamics**
Pejman Rohani, University of Michigan, USA

Understanding the concurrent epidemiological and evolutionary dynamics of influenza viruses has become urgent. Recent research has proposed interesting explanations for the evolutionary mechanisms that underlie the distinct phylogenetic patterns observed in human influenza viruses.

The ecology and evolution of these RNA viruses in their avian reservoir hosts, however, remain enigmatic. Most importantly, the footprint of strong immune selection observed in human isolates is distinctly absent in bird communities, which some have proposed indicates evolutionary stasis in such systems. Here, we take advantage of genetic data on virus isolates from North American birds combined with an agent-based stochastic transmission model to explore potential responsible mechanisms for the contrasting phylodynamics of influenza viruses in human and avian hosts. Our analyses suggest the dual time scale of transmission dynamics in avian systems is key: rapidly amplifying fecal/oral transmission that acts on the time scale of the infectious period and an environmentally mediated transmission mode that operates over a much longer period. The inclusion of this indirect transmission pathway—much like the storage effect—leads to substantially greater coexistence and genetic diversity in avian influenza viruses. To test this hypothesis, we regress different phylogenetic metrics of tree structure against subtype-specific estimates of environmental durability. We find environmental persistence to be a strong predictor of genetic diversity. Environmentally acquired infections, while rare, shape the evolutionary structure of avian influenza viruses.

9.30–10.00 **Biodiversity and disease: food webs, parasites and invasions**
Kevin D. Lafferty, United States Geological Survey, Santa Barbara, California, USA

Parasites are dependent on free-living biodiversity and their life cycles often traverse food webs. Human activities alter food webs and biodiversity and this can have direct and indirect effects on parasites. For instance, invasive hosts can bring new parasites with them, but often leave most of their parasites behind, potentially releasing them from the control of natural enemies. Habitat degradation and climatic effects can impact parasite diversity if hosts are removed. Fishing can decrease parasitism in fished hosts or increase parasitism in lower trophic levels. In addition, parasites can influence properties of energy flow and structure in food webs. For example, parasites can increase rates of predation on their hosts and some parasites are able to divert the flow of energy across ecosystems. Parasites alter the topological structure of food webs in ways that may affect their stability. In particular, many parasites have complex life cycles and this makes them particularly sensitive to secondary extinctions. Because host diversity begets parasite diversity, parasites can act as positive indicators of ecological integrity.

10.00–10.30 **Heterogeneity in immune dynamics and disease severity**

Andrea L. Graham^{1,2}, Kenneth D. Bruce³ and Mark E. Viney⁴

¹Princeton University, USA; ²University of Edinburgh, UK;

³King's College London; ⁴University of Bristol, UK

Animals exhibit enormous immunological heterogeneity. Such heterogeneity can be caused by genetic and/or environmental variations, and is likely to shape the transmission potential, disease severity, and outcome of invading infections. We are interested in the extent to which susceptibility to acute disease invasions may be determined by the community of helminth parasites and microbes that are chronically present on mucosal and epithelial surfaces. These anatomical sites are immediately pertinent to the biology of emerging diseases because many air-borne, food-borne and water-borne infectious agents inhabit (or must transit into the body via) these surfaces. Furthermore, mucosa-dwelling helminths and microbes can have systemic immunological effects. In this talk we will explore the prevalence and the likely systemic consequences of helminth-microbe communities for incoming infections.

Helminths and microbes are among the most prevalent residents of human mucosa. For example, billions of people bear intestinal nematodes, and each human intestinal tract harbours over a kilogram of bacteria. Similarly, the lung is often a thoroughfare for migrating larval helminths and, when infected, can be carpeted by diverse microbes. Recent research suggests several reasons why this complex community might be critical to understanding the establishment and proliferation of subsequent invaders. First, helminths actively suppress immune responses, while microbes appear to entrain the host immune system to distinguish pathogens from commensals. Therefore, the mucosal community is arguably part of the immune phenotype of a vertebrate host. Second, intestinal community composition affects host nutrition in complex ways that can affect immune responsiveness. In particular, helminths can be associated with protein malnutrition, with profound consequences for the magnitude and type of immune responses a host can mount. Finally, helminths and microbes can directly facilitate or impede each other's development. For all of these reasons, it is essential to incorporate within-host community ecology in order to understand heterogeneities in the immune dynamics and disease severity exhibited by hosts.

Characterizing helminth-microbe communities and their impacts on their hosts is not an easy task. We contend, however, that interdisciplinary teams equipped with relevant, state-of-the-art tools are increasingly necessary and ready for the challenge.

10.30–11.00 **POSTER SESSION (TEA/COFFEE)**

SESSION V: CONTROL STRATEGIES: CURRENT AND FUTURE

Chair: James Wood (Cambridge Infectious Diseases Consortium, University of Cambridge, UK)

11.00–11.30 **PREDICT: Analyzing the drivers of emerging diseases to predict the origins of the next pandemic**

Peter Daszak, EcoHealth Alliance, USA

Work over the past two decades has shown that zoonotic disease emergence is essentially an ecological process in which anthropogenic factors drive the spillover and spread of wildlife microbes in human hosts. Because these ‘drivers’ of emergence are measurable, and to some extent their future trajectory is known, analyzing them provides a basis from which to predict the patterns of future zoonotic disease emergence. In this talk, I present an approach to identify the regions where the next emerging infectious diseases (EIDs) are most likely to emerge (EID ‘hotspots’) and our current efforts to refine our recently published work (Jones *et al.* *Nature* 2008). Using data on human travel and trade patterns we can take this strategy further by identifying the hotspot regions which are most likely to propagate the spread of a new EID. This research may provide a cost-effective strategy for pandemic prevention, by allowing us to allocate global resources to the regions where the next pandemic is most likely to originate. In an effort to design such an approach, the US Agency for International Development (USAID) has launched an Emerging Pandemic Threats program to prevent disease emergence in hotspot countries. I will describe our efforts under the ‘PREDICT’ project to target surveillance of wildlife populations within EID hotspots and identify new potential zoonoses before they emerge in people.

Reference

Jones, K. E., Patel, N., Levy, M. A., Storeygard, A., Balk, D., Gittleman, J. L. & Daszak, P. (2008) Global trends in human emerging infectious diseases. *Nature* **451**: 990–993.

11.30–12.00 **Surveillance technologies, strategies and practices**

*Sarah Cleaveland, Jo Halliday, Zac Mtema and Katie Hampson
Institute of Biodiversity, Animal Health and Comparative Medicine,
College of Medicine, Veterinary Medicine and Life Sciences,
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Good surveillance, which involves the systematic collection of health-related data and timely response to those data, is essential for the effective identification, tracking and control of outbreaks of infectious diseases. In an ideal world, for early warning of disease emergence events, we would want to identify predictive tools that allow us to detect and prevent cases ‘ahead of the epidemic curve’. However, we still have a very limited ability to predict when or where new diseases are likely to occur and, although we have some understanding of the drivers of disease emergence, it remains uncertain as to where, when and how surveillance activities should best be targeted. For surveillance of both novel and known diseases, designing perfect surveillance systems *de novo* is a luxury that we often cannot afford. In this presentation, we discuss approaches to zoonotic and wildlife disease surveillance, giving particular consideration to practical issues and the likely cost-effectiveness of different approaches.

We argue that for novel emerging zoonoses, ‘passive surveillance’ approaches, with a focus on strengthening of primary health systems (both human and animal) and improved communication

networks, are likely to provide the most cost-effective method for early detection of new diseases that have significant impacts on human and animal health. We suggest that surveillance systems are often designed without a clear understanding of how observers/reporters are motivated to report events, and without a clearly defined and meaningful response, either in the context of disease control or in terms of feedback to reporters. While surveillance investment has been dominated by diseases of concern to high-income countries, such as highly pathogenic H5N1, we argue that greater investment in surveillance of neglected zoonoses, which usually exert a more substantial human and/or livestock disease burden in local communities, will foster a culture of surveillance that will provide a more effective platform for detecting emerging diseases, while at the same time having the potential for significant health benefits. We also make the case that much emphasis has been placed on building laboratory capacity, and that while high-performance diagnostic tests remain the ideal for detecting infection and disease, information obtained from non-'gold-standard' techniques, including proxy measures, can provide extremely valuable information for many surveillance purposes.

Several key issues will be discussed: (1) understanding how to incentivise observers to report cases; (2) understanding how to link surveillance data with timely and effective responses; and (3) exploring how novel approaches, including communication technology, can contribute to improved efficiency and cost-effectiveness of surveillance.

12.00–12.30 Mechanisms and mitigation in multi-host carnivore communities

Dan Haydon, University of Glasgow, UK

I will discuss control strategies in the framework of a target population in which a need to control the impact of infectious disease has been identified, and a reservoir that serves as a source of infection. I will define a reservoir as one or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained and from which infection is transmitted to the defined target population. Existence of a reservoir is confirmed when infection within the target population cannot be sustained after all transmission between target and non-target populations has been eliminated. Pathogens will persist in populations larger than the critical community size (CCS), and these populations I term 'maintenance populations'. In complex systems, pathogen transmission between a number of non-maintenance populations could constitute a 'maintenance community'.

I will discuss the concept of CCS and its relevance to the control of infectious disease from the early work of Bartlett to the present day, and review features of host populations and pathogen life-histories that influence CCS and hence the propensity of host populations to become reservoirs. I will briefly discuss the difficulties of identifying reservoirs and present some examples of new approaches to understanding reservoir-target population dynamics.

Lastly, I will describe strategies for mitigating the impact of rabies in a high-profile target population—the Ethiopian wolf (*Canis simensis*): the problem of predicting when such interventions might be required, and when they are, the use of low-coverage vaccination strategies designed to prevent extinction-threatening population crashes, rather than the elimination of all sizeable outbreaks. However, the use of such strategies requires that we develop a better understanding of the determinants of population recovery.

12.30–13.30 LUNCH

SESSION VI: FUTURE PERSPECTIVES

Chair: Sarah Cleaveland (University of Glasgow, UK)

13.30–14.00 **Human viruses: emergence, host range and discovery**

M. E. J. Woolhouse, University of Edinburgh, UK

Currently there are 219 recognised virus species from 23 families that are known to infect humans. Roughly one quarter of these viruses are capable of causing major epidemics and/or persisting in human populations, but the remainder are zoonotic, with host ranges that include other mammal species and, less frequently, birds. Factors determining the ability of viruses to infect humans or be transmitted by humans remain poorly studied. The constraints may be either biological and/or ecological. As one example, there is evidence that the use of conserved proteins as receptors to gain entry to host cells is associated with a broad host range and a higher potential to jump into humans from other mammals. Numerous ecological drivers of viral emergence have been proposed but few of these have been studied with any rigour; further work is needed in this area.

In recent years viruses have contributed disproportionately to newly recognised human pathogen species. Following the first recognition of a human virus—yellow fever virus—in 1901 the rate of discovery accelerated in the mid-20th century but has remained remarkably steady at between three and four new species per year for the past 50 years. The shape of the discovery curve suggests that we are still very far from cataloguing the full diversity of human virus species. Over 30 putative human virus species are awaiting recognition by the International Committee for the Taxonomy of Viruses. None of these represent additional virus families, but there are three families lacking known examples of human viruses but which contain viruses that can infect other mammals. Overall, it is unclear to what extent the ‘pool’ of undiscovered viruses is populated by extant, unrecognised viruses in humans or extant, unrecognised viruses in non-human reservoirs that sporadically spill over into humans or the continual evolution of completely novel viruses.

A key aspect of the public health response to emerging viruses is effective surveillance systems enabling the rapid detection of outbreaks. A better understanding of the factors promoting virus emergence would allow more efficient, targeted surveillance programmes. However, the challenge to implement sustainable global surveillance is formidable and issues of reporting incentives, governance and funding urgently need to be addressed.

14.00–14.30 **Winning tropisms: linking pathogenesis to pathogens’ fitness**

Leslie A Reperant, Princeton University, USA

The tissue tropism of pathogens within individual hosts largely determines the clinical picture and severity of the disease resulting from infection, and is also intricately linked to their epidemiology, in particular to the route and efficiency of transmission between hosts. Yet, the impact of tissue tropism within individual hosts on pathogens’ potential to spread at the population level—an essential property of pandemic threats—is poorly understood. The tissue tropism of influenza A viruses in humans is partly determined by their receptor binding affinity. Influenza viruses’ receptor binding affinity also appears essential for their ability to be efficiently transmitted from human to human. Influenza viruses with human-like binding affinity, targeting receptors abundantly present in the upper regions of the respiratory tract, are efficiently

transmitted between humans. In contrast, influenza viruses with avian-like binding affinity, targeting receptors abundantly present in the lower regions of the respiratory tract, are inefficiently transmitted between humans. The impact of respiratory tissue tropism on the reproductive fitness of influenza viruses and their ability to spread at the population level is nevertheless difficult to assess empirically. We developed a mathematical model that for the first time links within-host pathogenesis processes in compartmentalized regions of the respiratory tract to influenza virus reproductive fitness at the population level. The model demonstrates that the tropisms of human-adapted influenza viruses optimize their reproductive fitness, and that besides receptor binding affinity, the level of immunity in the population plays a critical role in determining optimal tissue tropisms. Influenza viruses with human-like binding affinity have different optimal tissue tropisms in naive and partially immune populations, causing disease that differs in severity, as seen during pandemic and seasonal influenza. Lower fitness of viruses with avian-like or mixed binding affinity is associated with preferred tropism for lower regions of the respiratory tract. These viruses can cause severe if not fatal disease but are not efficiently transmitted between humans, as seen for highly pathogenic avian influenza viruses H5N1. Switching to human-like binding affinity can enhance the fitness of avian-like viruses. The proposed framework provides novel insights into the role tissue tropisms play in determining trade-offs between host pathogenesis and transmission efficiency in influenza virus evolution. It demonstrates the major impact pathogenesis at the host level has on a pathogen's potential to spread at the population level, and can be adapted to clarify the cross-scale epidemiological and evolutionary dynamics of other pathogens beyond the influenza virus.

14.30–15.00 **Disease and the dynamics of extinction**

Hamish McCallum, School of Environment, Nathan campus, Griffith University, Queensland 4111, Australia

In theory, infectious disease is certainly capable of driving populations to extinction. It is most likely to do so when a pathogen affects multiple host species, some of which are reservoir hosts, able to harbour infection with limited effect on their own population, or when the transmission rate of a host-specific pathogen depends weakly on the population size of the host. In both situations, extinction is possible because the force of infection remains high even as the affected species declines. Although infectious disease is infrequently cited as a driver of extinction in the IUCN Red List, blaming notorious extinction events from the palaeontological or historical record on infectious disease has recently become somewhat of a cottage industry. In most cases, insufficient evidence is available to verify or disprove the role of infectious disease. Two ongoing wildlife disease epizootics, of the amphibian chytrid fungus *Batrachochytrium dendrobatidis* (hereafter Bd) and of Tasmanian devil facial tumour disease (hereafter DFTD) allow us to investigate extinction-threatening epizootics as they progress through affected communities and populations. Bd is an apparently recently introduced disease that is causing widescale amphibian declines and extinctions, particularly in the Americas and Australia. Some species and populations within species appear highly susceptible, whereas others are relatively resistant. This is a classic case of a disease with reservoir species being able to cause extinction of highly susceptible sympatric populations. DFTD is an unusual infectious cancer, in which the cancer cells themselves are the infective agent. Cancer cells appear able to be transmitted between hosts by biting because genetic diversity in Tasmanian devil populations is so low that recipient devils do not see the cells from another individual as "non-self". Transmission and prevalence are maintained at very high levels even as populations decline dramatically, suggesting the likelihood of devil extinction from this entirely host-specific disease. In both cases, management strategies beyond isolation of uninfected

populations are likely to rely on identifying resistant host genotypes. The outcome of host-pathogen co-evolution will be important for the long-term survival of wild host populations of both diseases. Bd and DFTD, respectively, illustrate the importance of two anthropogenic factors that can be expected to predispose more species to disease-induced extinction in the future: human-mediated introduction of pathogens into naive populations, and loss of genetic diversity.

15.00–15.30 **POSTER SESSION (TEA/COFFEE)**

SESSION VII: PRESENT POLICIES AND FUTURE REQUIREMENTS

Chair: Andrew Cunningham (Institute of Zoology, ZSL)

15.30–16.15 **National and international policies to mitigate disease threats: what is possible and what is not**

Christopher Dye (World Health Organization, Switzerland)

I will outline the steps that take us from an identified disease threat to a policy for mitigation, highlighting some strategies for success and recipes for failure. With reference to human and zoonotic infections, I will consider how policy emerges from the definition of a threat and the search for solutions, and describe how policies are implemented. Some of the key determinants of what is (im)possible in making and enacting policy are: the perception of risk, the strength of the underlying science, the state of technology, the conflict between individual and public health, ownership and intellectual property, the choice of weaker (guidelines) and stronger (law) policy instruments, the level of public interest, political opportunity, institutional inertia, methods for enforcement, and who foots the bill. I will illustrate these points with reference to the control of cholera, extensively drug-resistant tuberculosis (XDR-TB), HIV/AIDS, polio, pandemic influenza, and rabies.

16.15–17.00 **Disease threats to humans and to biodiversity: what to do?**

*Robert M. May, (Professor Lord May of Oxford, OM AC FRS),
Zoology Department, Oxford University, Oxford OX1 3PS, UK*

Only in the past few decades has significant attention been directed to the role infectious diseases play in the geographical distribution and numerical abundance of plant and animal populations—including humans. It is as well that this subject has advanced rapidly, because the effects of increases in human numbers and in the ecological impact per person are exacerbating old threats from infectious diseases, and producing new ones. It is easier to enumerate these threats to ourselves and to biological diversity than to give confident recommendations about “what to do”. I will nevertheless make some attempt to answer the question I was asked to address.

17.00 **End of Symposium**

DISEASE INVASION: IMPACTS ON BIODIVERSITY AND HUMAN HEALTH 18 AND 19 NOVEMBER 2010 — SYMPOSIUM RESEARCH POSTERS PRESENTED

Assessing the effectiveness of the dog sterilization and rabies control program at IAAS, Rampur, Nepal

Basler, C.¹, Kaufman, G.¹, Singh, S.² and Shah, I.²

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Rabies continues to pose a serious threat to human, domestic animal, and wildlife health in many parts of the world. Dogs are the main reservoir for the rabies virus in South-east Asia. Recently, a dog sterilization and vaccination program has been implemented in the Chitwan district of Nepal. The program area includes urban centers, farmland, and communities that border Chitwan National Park. The control program could reduce the incidence of rabies cases in human, livestock, and wildlife populations. In order to determine the effectiveness of the program, 120 residents of four communities where the sterilization/vaccination program has been operating were surveyed and their responses were compared to 120 residents in four similar communities in which the program has not yet been introduced. A cross-sectional cluster sample survey was used in order to determine the community member's knowledge of rabies as well as their attitudes about the rabies control program and their willingness to use the services the program provides. The assessment found that 77% of respondent's dogs in the experimental communities had been vaccinated and 38% of their dogs had been sterilized. In comparison only 29% of respondent's dogs had been vaccinated in the control communities and 3% of the dogs had been sterilized. Despite the increase in vaccinated and sterilized dogs in the affected communities, more work is needed in order to truly reduce the threat of rabies to human and animal populations in the area.

Modelling the influence of habitat hot spots on the spread of Ebola in western lowland gorillas

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'Habitat hot spots', places regularly visited by large numbers of individuals or social groups, can substantially alter contact rates and hence disease propagation. However, few studies have addressed this question in wild animal populations. We developed a spatially-explicit model simulating contact patterns on a lattice in which animal groups visit hot spots at a rate inversely proportional to the distance between their home-range and the nearest hot spot. We first explored disease dynamics in a simple Susceptible-Infectious-Removed (SIR) model, where individuals could be infected either by other individuals within their group, by neighbouring groups, and when crossing other groups' home-ranges on their way to the hot spot. Then, we fitted this model to Capture-Mark-Recapture data collected in a large population of gorillas (*Gorilla gorilla gorilla*) visiting a forest clearing during an Ebola outbreak using approximate Bayesian computation. Our model predicts that hot spots can dramatically

increase the percolation probability of a disease, especially in poorly connected populations. Moreover, it reveals that hot spots can even strongly affect groups who never visit them because their home-ranges are too distant. Counter-intuitively, groups ranging far from the hot spot can become infected earlier and have a higher attack rate than those ranging close to the hot spot. Finally, fitting the model to our gorilla data allowed to successfully estimate its parameters, and to assess the influence of forest clearings on the spread of Ebola in Central Africa.

Coccidian and helminths control could help in viral disease management in European wild rabbit

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Wild animals are infected with several parasites simultaneously, thus co-infection must be one of the most important factors for understanding the epidemiology of wildlife diseases. The European wild rabbit is the key prey for several endangered predators in Europe. Several rabbit populations have dramatically declined due to both myxomatosis and rabbit haemorrhagic disease (RHD). Important research to investigate the optimal management of these diseases in the wild has been carried out; however, most of the studies have failed to consider the importance of co-infecting parasites on the epidemiology of these viral diseases. Taking advantage of an experimental restocking program in south-west Spain, we tested the effect of coccidian and helminth infections on seroconversion against both viral diseases in three artificially isolated populations of European wild rabbit. Following a 2-year field survey our results suggest that both coccidian and helminth loads influence the seasonal seroconversion rates against myxoma virus, with a less clear pattern for RHD. Higher seroconversion rates against myxoma virus occurred when populations experienced lower parasitic loads, while a lesser immune response occurred in highly parasitized populations (mainly by coccidian). These results have implications not only on viral disease epidemiology, but ultimately on wild disease management aimed to increase rabbit populations in areas where they are scarce and fundamental for endangered predator populations.

Identifying gaps in wildlife surveillance: testing the relative importance of contact versus relatedness in zoonotic disease emergence

Tiffany Bogich¹, Kevin Olival¹, Kate Jones² and Peter Daszak¹

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Emerging Infectious Diseases (EIDs) pose a significant burden on human health and global economies, and nearly 75% of EIDs originate in wildlife. Understanding broad patterns of zoonotic disease emergence is key to preventing the next EID and facilitating adaptive surveillance. The transmission of pathogens from wildlife hosts to humans is driven by a complex suite of factors including the ecology of the host species, opportunity for contact with humans, characteristics of the pathogen and the phylogenetic relatedness of the wildlife host to humans. We focus on the case of mammal hosts and their viruses, and construct a database of 937 mammal-virus associations from the literature and characterize contact opportunities for host species and pathogen traits for virus families. We test the effects of phylogenetic relatedness versus contact on whether or not a virus is shared between wildlife hosts and humans. Our results demonstrate that both the opportunity for contact and phylogenetic relatedness of hosts are significant factors in predicting whether or not a virus is shared between wildlife hosts and humans. Using this model, we can identify gaps in surveillance

where species share a lower than expected number of viruses with humans given a threshold level of contact and relatedness, in order to better target our surveillance strategy to wildlife species most likely to be the source of the next emerging infectious disease in humans.

On the distribution of henipaviruses in the Australasian region: does Nipah virus occur east of the Wallace Line?

Andrew C. Breed, Hume E. Field, Supaporn Wacharapluesadee, Indrawati Sendow and Joanne Meers. E-mail: a.c.breed@vla.defra.gsi.gov.uk

Hendra virus (HeV) and Nipah virus (NiV) are highly fatal viral zoonoses, for which bats of the genus *Pteropus* have been identified as the primary wildlife reservoir. It is not known whether the distributions of HeV and NiV are mutually exclusive or overlap, or indeed if other henipaviruses exist between the locations where HeV and NiV occur. The aim of this study was to test the hypothesis that NiV is restricted in distribution to west of the Wallace Line. Fruit bats were sampled from Australia, Papua New Guinea, East Timor and Indonesia (Sulawesi and Sumba) and were tested for the presence of antibodies to HeV and NiV with a subset being tested for the presence of HeV, NiV or henipavirus RNA by PCR. Evidence was found for the presence of NiV in both *Pteropus vampyrus* and *Rousettus amplexicaudatus* from East Timor. Serology and PCR also suggested the presence of a henipavirus that was neither HeV nor NiV in *Pteropus alecto* and *Acerodon celebensis* from Sulawesi, and in *P. alecto* from Sumba. These results suggest the presence of NiV in the fruit bat populations of East Timor on the eastern side of the Wallace Line and within 500km of Australia. This work contributes to the management of the risk posed by henipaviruses in fruit bat populations to domestic animal and public health in the Australasian region. It also highlights the need for further work to determine the distribution and diversity of viruses in the genus *Henipavirus* and their epidemiology in reservoir hosts.

Disease transmission in extreme environments: parasites infect reindeer during the Arctic winter

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There are two main parasite species of Svalbard reindeer: *Ostertagia gruehneri* and *Marshallagia marshalli*. Like most gut nematodes, *O. gruehneri* is transmitted during the summer and has been implicated as a significant factor in regulating population dynamics of Svalbard reindeer. In contrast, *M. marshalli* levels are low in summer and appear to accumulate during the cold winter months when Svalbard reindeer are vulnerable owing to starvation. Here, we report the results of an anti-helminthic experiment in which we test the hypothesis that *M. marshalli* is transmitted during the Arctic winter.

Predicting epidemic size using observable data: an indeterminate problem?

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Public health officials and conservation biologists would greatly benefit from being able to forecast the final size of an infectious disease outbreak. For moderate to low values of the basic reproductive number, R_0 , outbreaks generally either fadeout in the early stages or develop into a substantial outbreak. If it were possible to predict when fadeout was likely to occur, the need for implementation of costly precautionary control strategies could be

minimized. However, the predictability of even simple epidemic processes remains largely unexplored. Here we conduct an examination of simulated data from the early stages of a fatal disease outbreak and explore available observable information that might be useful for predicting final outbreak size. Specifically, would knowing the time of deaths for the first few cases allow us to predict the likelihood that an outbreak will be large or small? We predict that small outbreaks are more likely when intervals between death times are wide and that large outbreaks should occur when death times are clustered. Using two approaches, one based on trajectory matching, and the other on discriminant function analysis, we find that even in the best case scenario (with accurate and complete knowledge of epidemiological parameters and all death times), it is generally not possible to reliably predict the outcome of a stochastic, compartmental, Susceptible-Exposed-Infectious-Recovered process. We show that the inability to predict outcomes of SEIR models is not a function of the quality of the epidemiological data, but instead results from the indeterminacy of the process.

Fundamental patterns of cross-species disease transmission among domestic and wild mammals

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Infectious diseases pose a significant threat to both wild and domestic animal health with many parasites being shared across this interface. Domestic animals can act as reservoir hosts of disease for wild species, whereas wild hosts may themselves become reservoirs for pathogen spill-back into domestic stocks. The scope of the problem is widely recognised and highlighted by many well-documented examples (e.g. rabies and bovine TB) but general determinants of parasite host-range and drivers of cross-species transmission at the domestic-wildlife interface remain poorly understood.

Here, we use two large-scale host/parasite databases to quantify the phenomenon of pathogen sharing among wild and domesticated mammals, and to identify underlying ecological, genetic and geographical factors. Based on almost 1000 records of disease-causing organisms in domestic mammals, we examined which of these parasites have been reported in wild populations, using over 16,000 records across 348 wild carnivore and ungulate species. Our results show that the chance of cross-over into wild populations is highest for viruses and protozoa, particularly in carnivores. The degree of parasite sharing was lowest in the fungi (<10%) but otherwise remained remarkably similar across host and parasite groups (35–45%). Domestic host group generally predicted disease occurrence in the wild, but this was least true for bacteria and for wild carnivores.

These analyses provide a basis for understanding the mechanisms and for developing a predictive framework of pathogen spillover at the domestic-wild interface. Ongoing work focuses on biological predictors of parasite sharing and on the role of surveillance effort and geographical context.

Epidemiology of squirrelpox virus in the Eurasian red squirrel (*Sciurus vulgaris*) in the Merseyside area

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Merseyside remains one of the few strongholds of the Eurasian red squirrel (*Sciurus vulgaris*) in England. In response to the national red squirrel decline, line transect surveys monitoring squirrel abundance were instigated on Merseyside in 2003. Disease incidence was assessed during this period through convenience sampling and subsequent post mortem examination. Until 2007, small isolated outbreaks of squirrelpox (SqPx) were recorded. However, impacts on the population remained local. From 2007 to 2008 a large population decline of up to 80% was recorded. This coincided with increased submission of SqPx cases. With little evidence of other factors associated with the decline, it is proposed that SqPx was the major cause. Since December 2008, no further cases of SqPx have been identified; subsequently the population has increased. But had some of the survivors been infected? A post-epidemic serological survey to assess squirrelpox virus (SqPV) exposure of the remaining red squirrels showed 5% (5/93) had SqPV ELISA OD values that were significantly deviant from the normal distribution displayed by all other results (mean = 0.178 ± 0.047). These results are spatially related to local SqPx cases. Thus we propose these five individuals to be seropositive animals. This is the first account of clinically normal red squirrels that have shown they are capable of surviving exposure to SqPV in their natural environment. This study also highlights that a review of the current threshold value for the SqPV ELISA is required when applied to live red squirrels, especially when the period of exposure to sampling is prolonged.

Identifying risk in conservation introductions

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The International Union for the Conservation of Nature (IUCN) has initiated the revision and expansion of guideline documents on conservation translocations with an increased emphasis on movements of organisms out of their historic range. The impetus is the growing recognition that climate change has altered the context within which conservationists attempt to prevent extinctions. Techniques known variously as assisted colonization, assisted migration and managed relocation have to date only occasionally been implemented as a solution to climatic threats. However, this technique is being considered by a much broader range of conservation practitioners and policy-makers without the benefit of broad guidelines.

The IUCN Reintroductions Specialist Group and Invasive Species Specialist Group have come together to form the Task Force on Moving Plants and Animals for Conservation Purposes with the aim of developing guidelines for conservation introductions. Ahead of the launch at the World Conservation Congress 2012, we are embarking upon a process of consultation from a multi-disciplinary group of researchers and practitioners to develop authoritative guidelines. We require expertise that will allow us to recognize risk at all stages of the introduction process. This includes the risk of exposing translocated individuals into novel ecosystems thereby exposing them to disease, and vice versa. Communicating the uncertainty associated with disease invasion, disease spillover and other areas of risk will be key to the success of the guideline documents in informing and aiding conservation introductions in a changing environment.

Human-animal interfaces and emerging infectious diseases in South-east Asia

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South-east Asia is widely regarded as a hot spot for emerging infectious disease (EID) events, particularly for zoonotic pathogens with origins in wildlife. Reasons for this have been linked to mammalian diversity, human population densities and the trade in wild-caught animals. As human populations grow, people are encountering wildlife with increasing frequency and under new situations.

With highly pathogenic avian influenza (HPAI) H5N1 as an example, we explore the role of human activities in promoting interactions at the human-animal interface. Here we present some of our recent research investigating cultural practices involving the exploitation of animals, within Vietnam and Thailand, and consider the implications that these may have for the spread of zoonotic pathogens.

We have found that the human-animal interfaces exposed through the exploitation of birds for cultural practices present a range of opportunities for pathogen transmission, both between animals and from animal to human. The cultural exploitation customs within these two countries include a range of activities, such as merit release ceremonies, songbird and fighting cock contests. Many of these practices are gender-specific and accessible by all social and age classes. Exploitation activities vary between and within these two countries, and in addition to the direct transmission opportunities posed by the activities, the transportation of birds, combined with the mixing of birds of different origins, pose threats for the longer distance transportation of pathogens, particularly viruses such as HPAI.

Identifying the factors and quantifying the risks associated with the potential introduction of the zoonosis Crimean Congo haemorrhagic fever virus to the UK

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Crimean Congo haemorrhagic fever (CCHF) is a tick-borne zoonotic virus that is endemic to areas of south eastern Europe, eastern Asia and Africa. Its infection is asymptomatic in animal hosts but can cause severe illness in humans, with a case fatality rate of 30–80%. The main tick vector of the virus is *Hyalomma marginatum* and human outbreaks have occurred within the geographical range of this tick.

Research into the ecology of *H. marginatum* may allow us to predict where future outbreaks might occur. Currently *H. marginatum* is present in southern Europe and some predictions suggest the species may expand its range northwards with climate change. There have been reports of *H. marginatum* ticks being introduced into northern European countries including Germany and the UK, and the climate parameters are key to the survival and moult of the tick. Although *H. marginatum* is established in the Iberian Peninsula there have been no reported cases of CCHF in humans. This indicates that factors besides the presence of the tick vector are important in disease transmission.

CCHF is emerging in the Balkans and the risk of the virus entering western Europe, including the UK, through a number of routes should be considered. Adult *H. marginatum* actively seek out and feed on large mammals including horses and for this reason the potential for horses to carry ticks into the UK is the focus of the study here. Horses are imported into the UK for sporting activities from several countries where *H. marginatum* is established and CCHFV is endemic in some of these regions. With regard to tick-borne transmission of CCHFV, factors such as a UK endemic tick (e.g. *Ixodes ricinus*) becoming a vector, or *H. marginatum*

extending its geographical range to include the UK need to be considered. Although the climate in the UK is currently unsuitable for *H. marginatum*, suitability may increase with climate change. Habitat suitability modelling will be used to delimit areas of the UK that are at greatest risk of both establishment of *H. marginatum* and of subsequent cases of CCHF under present and future scenarios.

A within-host network of human coinfection

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Coinfection by multiple parasites affects human health, parasite dynamics and the efficacy of infectious disease prevention and treatment. The capacity for different parasites (viruses, bacteria, fungi, protozoa, helminths) to interact is poorly understood. Interspecific interactions between coinfecting parasites could occur in many ways, either directly or indirectly with the host's immune system or bodily resources. We aimed to summarise connected resources, parasites and immune system components in coinfecting humans using a network approach.

The published literature contains thousands of records of coinfections in humans, associated immune responses, as well as parasite resource requirements. We used over 250 publications on human coinfection from 2009 to build an evidence-based *parasite-immune system-resource* network. We recorded the identity of coinfecting parasites, immune system components, host resources and the reported relationships between them. The network represents the potential for parasites to interact, based on observation and theory found in recent coinfection literature.

Results show the great taxonomic variety of coinfecting parasites, with particular involvement of viruses. Some parasites were reported in more coinfections, most notably HIV, suggesting that recent disease invasion and induced immunodeficiency may facilitate many parasite interactions.

The network can also be used to generate numerous hypotheses for modelling work and suggestions for future observational and experimental research. The use of networks and other research tools to understand parasite interactions within coinfecting hosts will help predict the potential for and consequences of disease invasions, as well as improve infectious disease interventions.

The environmental epidemiology and dynamics of enteric bacteria in blue tits

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The composition and dynamics of avian enteric bacteria are determined by both host-specific and environmental factors, yet these are poorly understood. Owing to their economic importance, the traditional focus of research on gastrointestinal bacterial communities in birds has concerned poultry and their associated pathogens. However, in recent years the increased economic value associated with feeding wild birds, in combination with well-documented disease outbreaks, such as mycoplasmosis and salmonellosis, have led to renewed empirical interest in common garden birds as potential vectors of disease.

Whilst bacterial pathogens play an important role in causing host mortality, there is a paucity of data concerning the dynamics of the gut microbial communities of wild birds. Previous work on a population of breeding blue tits (*Cyanistes caeruleus*) has shown that the composition and species richness of the gut bacterial community influences host fitness. For example, low bacterial abundance was associated with female blue tits laying larger egg clutches later in the breeding season, whilst the annual survival of adults declined with increasing bacterial species richness.

Using the same blue tit population in this study, a cross-fostering experiment was performed in conjunction with molecular methods (PCR and TGGE), to examine the relative contributions of genetic and environmental effects to variation in bacterial gut communities. We also briefly describe how longitudinal sampling of these PIT-tagged birds will allow us to identify temporal variation within gut community dynamics. These studies will help improve our understanding of the stability of the microbial communities and their impacts on general avian health.

Determining the risk of tick-borne diseases to the UK

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The Medical Entomology and Zoonoses Ecology (MEZE) Group within Microbial Risk Assessment, Health Protection Agency (HPA), assess the risk posed by various zoonotic and vector-related issues specific to public health in the UK. This presentation aims to introduce the activities of MEZE and details some of the approaches taken to better understand UK tick populations now and in the future, the impact of weather and climate on hosts and pathogens and the impact of humans and our policies. The main topics to be covered include: (1) a collaborative project with the National Institute for Public Health and the Environment (RIVM) which led to the first detection of *Rickettsia* spp. in British *Ixodes ricinus* and *Dermacentor reticulatus* ticks; (2) a collaborative project with the Veterinary Laboratories Agency to determine the risk of introduction of exotic tick species on companion animals; and (3) the findings from 5 years of HPA tick surveillance data including the detection of two non-native tick species (*Hyalomma marginatum* and *Amblyomma* species), the expansion of previously geographically restricted *D. reticulatus* and ground data on the spread of *I. ricinus* in south-west England. The results of HPA tick surveillance demonstrate that it is possible to run a cost-effective nationwide surveillance programme to successfully monitor endemic tick species, identify subtle changes in their distribution and detect the arrival and presence of exotic species, as well as providing a repository of specimens for future pathogen analysis.

Co-infection consequences in wild mice

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Although most infectious disease studies adopt a single-host-single-parasite framework, hosts in natural populations are frequently co-infected by multiple parasite species. The question of whether and how co-infesting parasites may interact, and what the consequences of such interactions might be for parasite transmission and host fitness, has attracted much attention. However, most existing studies of parasite interactions in wild populations have been purely observational. Inference from observational data on this question has many difficulties and, as with traditional community ecology, the only reliable way of detecting and quantifying

interspecific interactions is through manipulative experiments. We performed a large-scale field medication experiment in populations of wood mice (*Apodemus sylvaticus*) to test for the presence of within-host parasite interactions, and their effects on host fitness. Individuals received either a sustained or transient program of treatment with the anti-nematode drug Ivermectin, or were given a control treatment. Subsequent longitudinal monitoring of marked individuals allowed the effect of nematode suppression on the remaining within-host parasite communities to be measured over time, as well as the consequences of treatment for host survival. Here we present early results from this study and discuss their implications for our understanding of how interspecific parasite interactions affect disease dynamics and host health.

Don't cry wolf

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In the information age and global outreach, the scientific research agenda is perhaps becoming more driven by socio-political or institutional vested interest than in response to the actual reality on the ground. In the health sector generally, there has been a massive increase in spending on infectious and so-called emerging diseases some of which is, undoubtedly, highly justified but some, perhaps, is in reaction to more speculative science. Given the threat of disease to the long-term survival of species, a case is being made, in this symposium, for greater investment from conservation funds. This poster paper gives some real examples, which both promote and question this development. Wildlife examples to illustrate these points include; morbilliviruses, highly pathogenic avian influenza viruses, EBOLA virus, Henipah virus, rabies virus, chytridiomycosis and anthrax. Rational suggestions are made on which areas of wildlife health warrant support from the biodiversity and conservation community in order to safeguard species.

Characterization of *Mycoplasma* spp. and *Salmonella* spp. in griffon vultures and wild tortoises housed in a wildlife recovery centre

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Wildlife recovery centres are essential infrastructure for the care, management, breeding and conservation of many wildlife species and the correlated biodiversity. However, they also have the potential to serve as a reservoir of different pathogens, as animals originating from different localities often are kept together, housed in captive and/or crowded conditions, sometimes for long recovery times. We selectively screened the birds of prey and the tortoise populations at the wildlife centre Centro Fauna Bonassai in Sardinia, Italy, by taking tracheal and cloacal swabs and by applying molecular and immunoblotting techniques, apart from standard microbiological isolation methods. Two novel *Mycoplasma* spp. were isolated from the upper respiratory tract of four Eurasian griffon vultures (*Gyps fulvus*) and phylogenetically classified within the *Mycoplasma* taxonomy at the group and cluster levels. We detected the pathogenic *Mycoplasma agassizii* in all tortoise species (*Testudo graeca*, *Testudo marginata* and *Testudo hermanni*) recovered in the centre, and also *Salmonella enterica* (serotypes Abony, Posdam, Granlo) with relevant zoonotic implications in *Testudo marginata* individuals. Different levels of pathogenicity and prevalence of these strains have important implications for the management and reintroduction of vultures and tortoises. Local endangered populations of these species might be seriously threatened by emerging infectious diseases, while the isolation of *Salmonella* strains which are also pathogenic in humans represents a public health concern.

Introduced Siberian chipmunks (*Tamias sibiricus barberi*) harbour more diverse *Borrelia burgdorferi* sensu lato genospecies than native bank voles

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Little attention has been given in the scientific literature as to how introduced species may act as a new host for native infectious agents and modify the epidemiology of a disease. In this study, we investigated whether an introduced species, the Siberian chipmunk (*Tamias sibiricus barberi*), was a potentially new reservoir host for *Borrelia burgdorferi* sensu lato causative agents of Lyme disease. First, we ascertained whether chipmunks were infected by all of the *Borrelia* genospecies associated with rodents and available in their source of infection, questing nymphs. Second, we determined whether the prevalence and diversity of *Borrelia* in chipmunks was similar to that of a native reservoir rodent, the bank vole (*Myodes glareolus*). Between 2006 and 2008 we trapped 335 chipmunks and 671 voles, and dragged 743 questing nymphs in a suburban forest in France. We searched for *B. burgdorferi* sl in ear biopsies taken from the rodents and in nymphs using PCR and RFLP. Chipmunks were infected by the three genospecies that were present in questing nymphs and that infect rodents (*B. burgdorferi* sensu stricto, *B. afzelii*, and *B. garinii*). In contrast, voles hosted only *B. afzelii*. Furthermore, chipmunks were more infected (35%) than voles (16%). These results may be explained by the higher exposure of chipmunks, because they harbour more ticks, or by their higher tolerance to *Borrelia*. If chipmunks are competent reservoir hosts for *Borrelia*, they may spillback *Borrelia* to native communities and eventually may increase the risk of Lyme disease transmission to humans.

ENHanCE project – ERA NET Health and Climate in Europe

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Considered a major threat to human health and well-being via possible effects on infectious diseases in both humans and animals, the potential impact of climate change on total disease burden is unknown. While some diseases may spread with climate change, others might recede; it is possible that the most important diseases in health or economic terms could be resilient, making any impact on total disease burden of minor importance.

To enable policy makers to assess the impact of climate change on total disease burden, the following are required: expansion of the knowledge base of climate impacts on diseases; development of robust methodologies to project the impact of climate change on the future of diseases; data on pathogens to be applied to methodologies. A recently funded international project tackles these issues by assessing how a subset of diseases might respond to climate change in Western Europe.

To investigate our disease future, the ENHanCE project addresses a series of questions. What pathogens affect or threaten the region? Which of these are most important? What are their

known climate drivers? These questions will be qualitatively examined by developing a database containing detail on pathogens, using a framework of questions to prioritise for human health, animal health and welfare, and economic impact, and distinguishing climate- and environmental-linkages to pathogens with automated search mechanisms of bibliographic software.

Once a shortlist of high-impact, climate-sensitive pathogens is obtained, ENHanCE asks how climate might change in the region using state-of-the-art climate-change forecasts. Both outputs will thereafter feed into work on selected pathogens, asking how they might respond to climate change. Modelling methodology will be developed using quantitative approaches, with outputs including estimations of future distribution, incidence or severity.

As non-climate drivers may also change our disease future, these will be considered based on discussion with stakeholders and decision makers. The results will be integrated with climate-driven outputs to derive a realistic assessment of our true disease future.

Spatial distribution of questing deer ticks *Ixodes ricinus* (Acari: Ixodidae) along woodland rides and glades in Wiltshire, UK – Implications for woodland management and public exposure

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This study aimed firstly to assess whether ecological and environmental factors could determine hot spots for questing deer ticks (*Ixodes ricinus*) in lowland woodland, and secondly whether both bracken management and ride (i.e. margins of woodland tracks) management has, or may have, an impact on reducing numbers of questing *I. ricinus*. The possible effects of woodland management strategies and public exposure are discussed, with reference made to nature conservation guidance on ride management and current management strategies at the study site in south Wiltshire. The occurrence and abundance of *I. ricinus* nymphs and adults appear predictable within a lowland woodland ride system. Certain climatic, vegetation and topographical variables appear to predict hot spots of questing nymph activity along woodland rides. Hot spots of activity were significantly positively associated with south and west-facing rides, and negatively associated with east-facing rides. North-facing rides appear to favour absence of questing nymphs. Vegetation-related variables, such as increased mat depth, reduced ride penetrability, occurrence of bracken and bramble, presence of large animal tracks and favourable sward height, all appear to favour a moist microclimate and increase host potential, thus promoting tick survival. Bracken management using Asulox herbicide (in combination with swiping/collecting) in woodland glades appears to reduce the depth of bracken litter/mat and consequently reduce numbers of questing nymphs, compared to sites where bracken was swiped/collected but not treated. In conclusion, several recommendations related to ride management, seasonal mowing, use of bracken herbicide and mat management are discussed in relation to tick control. Advice is also suggested to woodland visitors to reduce public exposure to ticks.

Assessing the possible implications of wetland expansion and management on British mosquitoes

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The expansion of existing wetlands, their creation from arable land, and the development of new salt-marsh to alleviate coastal erosion are important issues in the UK, as the environment sector adapts to the possible impacts of climate change and continues to meet its goals in providing increased wetland habitat for wildlife, and an outdoor space for human well-being. Concerns have been raised over the potential impacts that such initiatives might have on British mosquitoes and the possible future transmission of infectious diseases. This paper details ongoing Health Protection Agency activities at the Great Fen, Cambridgeshire, in relation to impacts of wetland management/expansion on British mosquitoes and considers possible environmentally friendly mitigating strategies for the key British mosquito species of various wetland habitats. Developing this evidence-base is a crucial element in preparing for the emergence of mosquito-borne disease in the UK and in aiding policy makers in their assessments of the risks and impacts associated with wetland expansion on mosquito nuisance and disease risk. It is also important in ensuring that biodiversity gain and habitat restoration can advance without inadvertently elevating the risks from disease vectors.

Effect of hemoparasites *Trypanosoma cruzi*, *T. evansi* (Kinetoplastida: Trypanosomatidae) and microfilariae sp. on health of brown-nosed coatis from the Pantanal wetlands, Brazil

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Trypanosoma cruzi and *T. evansi* are protozoans that cause Chagas disease in humans and "Mal de Caderas" disease in horses, respectively. Coatis (Procyonidae: *Nasua nasua*) have been suggested as important reservoirs for these parasites in the Brazilian Pantanal. Here we examined the relative importance of high parasitemias by *T. cruzi* and *T. evansi*, and abundance of microfilariae on the health of coatis captured in the Pantanal from 2006 to 2009 (N=70). We used coati body condition and hematological parameters as response variables in linear models that were compared using AIC. Body condition decreased in coatis with high parasitemias of *T. evansi*, especially during the reproductive season. Total red blood cell counts and packed cell volume decreased in males with high *T. evansi* parasitemias, but females showed a more variable response. Monocytes decreased in individuals with high parasitemias by *T. cruzi*, while eosinophils decreased mainly in males with high parasitemias by *T. evansi*. Total white blood cell counts and neutrophils decreased in males with high parasitemias for both *Trypanosoma* species during the reproductive season, whereas females showed variable responses to infection. Females therefore apparently handle infection better than males. In addition, an overall decrease in health occurred during the breeding season, when coatis are under reproductive stress. High abundances of microfilariae in the bloodstream were also associated with decreased coati health, but its effect size was comparatively small. This study shows the potential of hematological parameters along with body condition as indicators of hemoparasite impacts on health of wild hosts.

Fragmentation of host populations and its effect on parasite dynamics in a natural island system

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Ecological theory predicts that parasites may be unable to persist in small, isolated host populations. Empirical examples for this prediction exist in human notifiable diseases, such as measles, but are largely absent for wildlife systems. Determining the limits to parasite persistence in natural systems has obvious implications for disease eradication. At the same time, it can inform our understanding of how disease dynamics may be altered in wildlife populations that become small and fragmented. On the one hand, these populations may benefit from dealing with a reduced set of parasite species. On the other hand, parasites may become more prevalent in isolated populations as hosts are more crowded or have a reduced ability to deal with infection due to genetic inbreeding effects. Here, we present results of a 2-year study monitoring the distribution and abundance of three main parasite groups and their two woodland rodent hosts within a naturally fragmented island system. The results indicate that most parasites remained detectable even in very small populations of less than 30 hosts. However, one gastro-intestinal helminth, *Capillaria*, appears to be absent from bank voles on islands. *Ixodes* ticks were more prevalent on islands compared to mainland sites, whereas the reverse was true for fleas. We found that despite some island populations being extremely small, there is limited evidence that host abundance restricts parasite distribution. However, infection prevalence in some parasite species appears to be affected by host population size and isolation.

Does stress influence micro- and macroparasite burdens in wild chacma baboons (*Papio ursinus*)?

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The need to understand parasitological dynamics and host susceptibility has become ever greater with the increasing awareness of emerging infectious diseases and the potential impact of zoonotic transfer on human health. One area of recent interest has been the role of stress as a factor in parasitic infections. This study investigated the role of stress in micro- and macroparasite infections in a wild primate population.

141 faecal samples from 31 habituated female chacma baboons (*Papio ursinus*) in Namibia were analysed for gastro-intestinal parasites using the formol-ether sedimentation technique. Five different measures of parasite load were recorded per sample: parasite taxon richness, the occurrence of two potentially pathogenic parasites *Balantidium coli* and *Streptopharagus pigmentatus*, and the relative abundance of *B. coli* and eggs-per-gram of *S. pigmentatus* where present.

The five measures of parasite load were analysed in relation to matching sample data on individual stress faecal cortisol metabolites, age, body condition, and the time of day and month of sample collection, using general linear mixed-models (GLMMs).

The results indicated that seasonality and individual body condition may be important factors influencing host parasite loads. Stress, however, as measured by faecal cortisol metabolites, does not appear to be associated with higher parasite loads, indicating that the role of cortisol may be minimal or that its interactions with parasites are more complex.

Sex, hybrid vigour and inbreeding avoidance in a fish pathogen

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Not all pathogens co-evolve with their hosts. Increasingly, we are aware that disease invasions may result from successful host switches, an evolutionary strategy that appears the norm in an important group of fish pathogens, the gyrodactylid monogeneans. These ubiquitous ectoparasites of teleost fish reportedly employ both asexual and sexual reproduction, allowing them to maximise their ability to colonise available niche space rapidly, but also maintaining genetic diversity for increased transmission abilities, virulence and infectivity. In *Gyrodactylus* spp., sexual recombination may further enhance the chance of successful host switches and in doing so may cause disease outbreaks in previously uninfected, susceptible fish hosts. Here, we crossed three divergent strains of *Gyrodactylus turnbulli* and compared the fitness of pure with mixed-strain parasite populations to examine heterosis. Using microsatellite markers to unambiguously demonstrate the occurrence of sexual recombination in gyrodactylids, we showed that mixed parasite populations on individual guppies (*Poecilia reticulata*) reached larger infrapopulations than pure parasite populations. There was also evidence of inbreeding avoidance, such that parasites infecting a fish already infected by a parasite of the same strain were slower to attach compared to those infecting a fish previously infected with a different strain. In light of the increasing global transport of food and ornamental fish reared in aquaculture facilities potential problems associated with heterosis and inbreeding in ubiquitous pathogens is discussed.

Current but not past microparasite infections drive helminth burdens in feral cats

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Most free-living hosts are simultaneously infected by several species of parasites that are gradually acquired. However, interactions between parasites in natural populations have been poorly studied, and a key question that remains is whether or not past infections by a type of parasite affect susceptibility to others. In this work we aim to address the following question: could microparasite infection history influence macroparasite burden in a medium-sized mammal? We explored the interactions between a mixture of past (three viruses: Feline Leukaemia virus, Parvovirus and Coronavirus, three bacteria: *Chlamydophila* sp., *Leptospira interrogans* and *Ehrlichia* sp. and one Protozoan: *Leishmania infantum*), and current (one virus: Feline immunodeficiency virus (FIV) and one protozoan: *Toxoplasma gondii*) microparasite infections and current helminth burdens (estimated after necropsy) using the Partial Least Square regression (PLSr) method in a sample of 11 young (< 1 year old) and 48 adult wild-caught feral cats from Mallorca (Balearic Islands, Spain). According to the Th1/Th2 trade-off, we expected that helminth burdens should be higher in individuals that were exposed in the past to many microparasite species. Our results showed that most of the helminth species covaried positively, with adult cats infected by *Toxoplasma* and/or FIV (both current infections) having the higher helminth burdens. Thus, in disagreement to our prediction, current infections were more important than past infections for explaining the current helminth burdens. Our research indicates that future research should consider the importance of parasite communities rather than parasites species in isolation.

Epidemiology of digenean biliary parasites in the Eurasian otter *Lutra lutra*

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Otters *Lutra lutra* can act as host to a range of parasitic fauna capable of infecting multiple host species. Investigation of the variables influencing the distribution, prevalence and intensity of such generalist parasites is important for public and veterinary health, particularly when the parasite has newly arrived in an ecosystem. The Cardiff University Otter Project routinely post mortems road-killed otters from England and Wales. Two digenean species were recovered from the bile and bile ducts, *Pseudamphistomum truncatum* (12.5% prevalence) and *Metorchis albidus* (6.5% prevalence) of 477 screened gall bladders. Both species are thought to have only recently been introduced to the UK and there is no record for either prior to 2000. The spatial distribution of these parasites was examined using a modified Ripley's K statistic, $K[i](r)$, with Ripley's isotropic edge correction to assess for clustering. *Pseudamphistomum truncatum* infections cluster and are over-represented in the southwest of England and South Wales whereas *M. albidus* infections appear randomly distributed. At present, there is no detectable increase in prevalence with year for either parasite. These parasites cause significant pathological damage to the biliary system of their host and therefore it is necessary to monitor their distribution to protect both wild and domestic piscivores against these generalist pathogens. The presence of these parasites in the UK now provides an ideal opportunity to understand how a pathogen can spread across a novel habitat.

Parasitic diversity of wild carnivores and considerations on their conservation

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A pilot study was performed in order to collect data to update information on the parasitic diversity of mesocarnivores in southern Portugal, and to establish relationships between parasite dispersal, carnivore conservation and human health. We studied the following roadkilled carnivore hosts: foxes (*Vulpes vulpes*), mongoose (*Herpestes ichneumon*), stone martens (*Martes foina*), common genet (*Genetta genetta*) and Eurasian badgers (*Meles meles*). A sample of hunted foxes was also included. We collected geo-reference information for all animals, which was used for spatial analysis (QGIS/GRASS). Thorough necropsies were performed and macroparasites collected, identified and preserved. A common genet parasite, *Ancylostoma martinezi*, was recorded in Portugal for the first time.

Statistical species richness of parasites and its correlations with human and environmental factors were determined. Human population density, water drainage, soil use, and minimum distance to head council city proved no significant statistical relation with the infection status of foxes. Results obtained on zoonotic parasites present in wild animals enhance the necessity of multidisciplinary work between conservation biology and medical sciences.