



Welcome to the

**2009 Conference on the  
Ecology and Evolution of  
Infectious Diseases**

***“Infectious Disease Dynamics in Multi-host Multi-  
pathogen Communities”***



Hosted by the  
**University of Georgia**

**Athens, GA**

**20-23 May, 2009**

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## About Our Sponsors



The President's Venture Fund was established to assist with significant funding opportunities that are brought to the President's attention by the Provost with the support of a department head and dean. This fund is supported by the Arch Foundation for the University of Georgia, an organization focused on securing the private financial resources that will help continue the rise in academic quality at UGA. It is the primary goal and mission of the Arch Foundation to build and sustain an endowment that will help make UGA one of America's very best public universities.

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Since 2006, the National Science Foundation has supported the annual EEID workshops by providing administrative support, travel and tuition support for students, and support for the workshop instructors (NSF DEB 0722115: Training Workshops on the Ecology and Evolution of Infectious Diseases).

### **2009 EEID Local Steering Committee:**

Sonia Altizer, Assoc. Professor, Odum School of Ecology, University of Georgia

Pejman Rohani, Professor, Odum School of Ecology, University of Georgia

Julie Rushmore, PhD student, Odum School of Ecology and College of Veterinary  
Medicine

Nanda Dórea, DVM, MS student, Veterinary and Biomedical Sciences and Poultry  
Diagnostic and Research Center

**Table of Contents**

About our sponsors..... i

Meeting schedule..... 1

Abstracts

    Talks..... 7

    Posters..... 16

    Field trips..... 40

Venue

    Classic Center..... 41

Dining

    Lunch in Athens..... 42

    Dinner in Athens..... 43

Maps

*Athens area with hotels and conference venue*..... 46

*UGA Campus map*..... 47

List of conference participants ..... 48

## 2009 EEID Meeting Schedule

### Wednesday, May 20: Registration and Welcome Reception

**6:30pm** Registration opens, Classic Center Firehall

**7:00-** **Welcome Reception, Classic Center Firehall**

**10:30pm** Complimentary beverages and cocktail buffet provided by Center Stage Catering

*Parking is available at the Classic Center each day at no charge to participants*

### Thursday, May 21: Symposium at Classic Center, Grand Hall

**7:45am** Registration opens

**8:15am** Continental breakfast served in Grand Hall lobby

**8:30am** Welcome and opening remarks: John Gittleman, Dean, Odum School of Ecology; Sonia Altizer, conference co-organizer

#### Session 1: Dynamics of multi-host pathogens

*Moderator: Pejman Rohani, UGA Odum School of Ecology*

- 9:00 ***Hierarchies of selection drive the evolution of host range in pathogens and parasites*** Mike Antolin Colorado State Univ.
- 9:40 ***Dynamics of multihost pathogens in Serengeti carnivores*** Meggan Craft Univ. of Glasgow
- 10:00-10:20 ***Location, location, location: context is the driver of infection by a vector-borne generalist grass pathogen group*** Elizabeth Borer Oregon State Univ
- 10:20 ***BREAK: Complimentary refreshments served in Grand Hall Lobby***
- 10:40 ***Spillover dynamics of monkeypox in humans*** James Lloyd-Smith UCLA
- 11:00 ***Bacteria-phage coevolution in the phyllosphere*** Britt Koskella Oxford Univ.
- 11:20 ***Reversal of fortune: human pathogens killing coral in the Florida Keys*** Jim Porter Univ. of Georgia
- 11:40 Synthesis session *led by Amy Pedersen, Marm Kilpatrick, Meghan Duffy*

**12:00pm-1:30pm** Lunch (on your own)

#### Session 2: Co-infections and dynamics of multi-pathogen interactions

*Moderator: John Drake, UGA Odum School of Ecology*

- 1:30 ***The transmission dynamics of influenza*** Sunetra Gupta Oxford Univ.
- 2:10 ***Exotic orboviruses in the United States: the invasion of occupied habitat*** Dave Stallknecht Univ. of Georgia

- 2:30 ***How well can immune systems multi-task, and how well should they?*** Andrea Graham Univ. of Edinburgh
- 2:50 ***BREAK: Complimentary refreshments served in Grand Hall Lobby***
- 3:10 ***Virulence tradeoffs in a vertebrate virus*** Andrew Wargo Univ. of Washington
- 3:30 ***The clever hitchhiker: rapid fixation of internal segments of avian influenza viruses*** Rubing Chen Penn State Univ.
- 3:50 ***Immunoepidemiology of a gastrointestinal nematode in single and co-infections*** Lisa Murphy Univ. of Glasgow
- 4:10 ***Damned if you do, damned if you don't: non-target pathogen effects on viral resistance transgenes in wild gourds*** Matt Ferrari Penn State Univ.
- 4:30 *Synthesis session led by Helen Wearing and Kim Pepin*

**6:30pm-** Dinner (on your own, downtown Athens)

## **Friday, May 22: Symposium at Classic Center, Grand Hall**

**8:15am** Continental breakfast served in Grand Hall lobby

**8:30am** Announcements and updates (*Posters can be set up starting at 8:00am*)

### **Session 3: Dynamics of pathogens with multiple transmission modes and other heterogeneities**

*Moderator: Erin Lipp, College of Public Health, Environmental Health Sciences*

- 9:00 ***Insect outbreaks and heterogeneity in resistance to pathogens*** Greg Dwyer Univ. of Chicago
- 9:40 ***Human social contact patterns and the spread of infection*** Ken Eames London School of Hygiene and Tropical Medicine
- 10:00 ***The aspergillus-sea fan epizootic: modelling a moving target and the evolution of resistance*** Drew Harvell Cornell Univ.
- 10:20 ***BREAK: Complimentary refreshments served in Grand Hall Lobby***
- 10:40 ***Urban habituation, ecological connectivity and epidemic dampening: the emergence of Hendra virus from flying foxes*** Raina Plowright Penn State Univ.
- 11:00 ***Environmental transmission and the ecology of avian influenza viruses*** Pej Rohani Univ. of Georgia
- 11:20 ***Cholera in Bengal: multiple transmission*** Aaron King Univ. of Michigan

*modes, multiple infection outcomes*

11:40 Synthesis session led by Olivier Restif and Katie Hampson

12:00pm-1:30pm Lunch (on your own)

**Session 4: Consequences for pathogen emergence and control**

Moderator: Sonia Altizer, Odum School of Ecology

1:30 *Antigenic drift of influenza viruses* Derek Smith Univ. of Cambridge

2:10 *Adaptation of zoonotic pathogens to human transmission* Angela McLean Univ. of Oxford

2:30 *Canine influenza and canine parvovirus as models of viral host switching* Colin Parrish Cornell Univ.

2:50 **BREAK:** Complimentary refreshments served in Grand Hall Lobby

**Overview of poster presentations** (1 min PPT slides) Moderator: Sonia Altizer

3:30 Slides will be presented in the order shown for the poster titles on p. 16 of the program

**Poster session: Grand Hall South 1**

5:00-7:00pm (Please remove posters at 7:00pm, prior to dinner)

**Dinner/Social; Empire Room, Foundry Park Building**

7:00-9:30pm (Walk to the back side of the Classic Center and across Foundry Street)  
Food and drinks provided by Center Stage Catering; Live music provided by *Grogus*

**Saturday, May 23: Hike/Field Trip**

9:00 am Buses depart from back parking lot of the Classic Center Building (near the 1<sup>st</sup> level of the parking deck) for Tallulah Gorge or Panther Creek Trail. (More information provided in the hike section of the program)

4:00pm Buses return to Classic Center

6:00pm **Group discussion** on North Lawn of UGA Campus, in front of the Chapel, (near intersection of Broad and Jackson St. and the famous UGA arch; see campus map on last page of program). Discussion to focus on emerging themes in EEID and plans for the 2010 conference

**Dinner/Social; Transmetropolitan Pizza** 145 E Clayton St, Athens

7:00-9:30pm Upper level reserved for EEID conference.

## SPEAKER ABSTRACTS

*In alphabetical order by last name*

### **Hierarchies of Selection Drive the Evolution of Host Range in Pathogens and Parasites**

Mike Antolin, Colorado State University

The problem of disease emergence falls in parallel with the evolution of host range. Host ranges are determined by opportunities for selection arising at multiple levels, from the geographic ranges of both pathogens (parasites) and their hosts at the broadest scale, to relative genomic flexibility of both at the finest scale. Here I lay out a framework for understanding opportunities for selection on host range, illustrated by the case of plague in North America. The bacterium that causes plague (*Yersinia pestis*) is thought of as a highly virulent pathogen with a broad host range. In reality, the case of plague in prairie dogs points to local spread of clones that persist within single host species along with their fleas that act as vectors.

### **Location, Location, Location: Context is The Driver of Infection by a Vector-Borne Generalist Grass Pathogen Group**

Elizabeth T. Borer, Oregon State University

Co-authors: Eric W. Seabloom, Charles E. Mitchell, Alison G. Power

Prevalence of many pathogens is increasingly being linked to host context, including the host and non-host community and abiotic environment. Recent large-scale observations of barley and cereal yellow dwarf viruses (B/CYDV), a group of generalist, vector-borne grass pathogens, suggest that infection differs among host species and increases with soil nitrate. However, like many vectored-pathogens, the relative importance of host identity, location, and community context in determining infection risk remain unclear from observational data. We applied a factorial combination of nitrogen and phosphorus fertilizer at hierarchically nested spatial scales ( $10^5$  to 1 m) to examine drivers of infection. We planted six common Pacific Coast annual and perennial grass species in two blocks at five West Coast grassland sites (>5,000 hosts). Hosts were individually tracked and assayed for B/CYDV. Infection did not differ among host species or phylogenetic groups; fertilization increased host size and cover of high-quality hosts but did not alter prevalence. Prevalence varied spatially, with greatest variation among blocks, suggesting factors such as community context as drivers of infection. Infection was unaffected by host richness but increased with cover of long-lived hosts. Overall, meso-scale variation ( $10^3$  m) is a stronger covariate of B/CYDV infection than host identity or community context.

### **The clever hitchhiker: rapid fixation of internal segments of avian influenza viruses**

Rubing Chen and Edward C. Holmes, Penn State University

Contrasting evolution patterns have been found between HA/NA/NS, which related to the host immune reactions, and the five internal segments (PB2/PB1/PA/NP/M) of avian influenza viruses. Comparing to the deep root age and multi-subtype phylogeny in the first group of segments, the internal segments only have approximately 10 times shallower genetic diversities and form a single gene pool. However, it is not clear what evolutionary processes are responsible for these different patterns of genetic diversity. To explain the

discrepant evolution pattern of the two groups of segments, we estimated the divergence time of each segment of AIVs sampled globally using a Bayesian MCMC coalescent method. Strikingly, the internal segments of currently circulating AIV strains only diverged in approximately 100 (98-129) years ago, with the highest 95% HPD 170 years ago. More interestingly, similar divergence time is observed in the individual subtypes of HA/NA/NS segments. We therefore propose that the genetic structure of AIV, and particularly the differing divergence times of the segments, is the result of a combination of occasional selective sweeps in the HA and NA, with transient genetic linkage to the internal gene segments.

### **Dynamics of multihost pathogens in Serengeti carnivores**

Meggan Craft, University of Glasgow

Co-authors: Sarah Cleaveland, Andrew P Dobson, Eblate Ernest, Katie Hampson, Magai Kaare, Tiziana Lembo, Craig Packer & Dominic Travis

We studied the viral transmission dynamics of rabies and canine distemper (CDV) in the Serengeti Ecosystem, Tanzania. Our study included an extensive vaccination program for 30,000-40,000 dogs/year, as well as field observations, molecular genetic analysis, and modeling. Genetic data show that a single rabies virus variant circulates among a range of species, with no evidence of species-specific virus-host associations. Within-species transmission was more frequently inferred from high-resolution epidemiological data than between-species transmission. The balance of evidence suggests that the reservoir of rabies in the Serengeti ecosystem is a complex multi-host community where domestic dogs are the only population essential for persistence, though other carnivores could contribute to the reservoir as non-maintenance populations. Despite vaccination of domestic dogs against CDV, the virus continued to be detected in wild carnivores, either because of a higher  $R_0$  in this disease or a more complex reservoir system involving wild carnivores as well as domestic dogs. Modeling suggests that multiple hosts were involved in the 1994 CDV outbreak in Serengeti lions. In addition, virulence of CDV in lions appears to depend on the intensity of co-infection with *Babesia*, a tick-borne parasite that reaches high levels of parasitemia during droughts from tick infestations of starving ungulates.

### **Insect outbreaks and heterogeneity in resistance to pathogens**

Greg Dwyer, University of Chicago

Boom-bust population fluctuations in herbivorous insects are often driven by baculoviruses, fatal pathogens transmitted as insects feed. Baculoviruses can be easily used in field experiments, and work in my lab therefore uses experiments to understand sources of heterogeneity in susceptibility in gypsy moths. In classical insect-pathogen models, some level of heterogeneity in susceptibility is required to avoid unstable cycles, as in many host-parasitoid models. Sufficiently high levels of heterogeneity, however, lead to unrealistic stable population dynamics, yet our data for the gypsy moth suggest that heterogeneity in susceptibility is indeed high enough to prevent outbreaks. For the gypsy moth, standard models are thus clearly incorrect. The incorrect assumption appears to be that the distribution of susceptibility is constant. Additional experiments have shown that this distribution can change either because of natural selection, or because of induction of plant defensive compounds. Experiments with half-sib groups have shown that susceptibility is heritable, and experiments before and after epidemics have shown that susceptibility declines after epidemics. Meanwhile, models that allow for evolution of host resistance can reproduce outbreaks even for high levels of heterogeneity. Similarly, induction of defensive tannin compounds because of insect defoliation reduces heterogeneity in susceptibility, and models incorporating this latter mechanism also produce realistic cycles for high baseline levels of heterogeneity. In both of these cases, however, a basic difficulty is that the

models describe susceptibility in terms of phenomenological distributions. We are therefore currently extending our models to allow for variation in susceptibility at the individual level.

### **Human social contact patterns and the spread of infection**

Ken Eames, London School of Hygiene and Tropical Medicine

It is often difficult to determine precisely what is meant by an epidemiologically risky contact. For many infections, the proximity, duration, and intensity of an encounter necessary for transmission are unknown. Therefore, it is not obvious which specific questions a contact survey ought to ask. Here we describe the results of a detailed survey of human contact patterns and show that very different levels of heterogeneities emerge depending on which factors we consider: the difference between physical and conversational contacts, and the importance of the social setting of a contact are discussed. We use these data to parameterise a weighted network model of infection spread, and show that pessimism about these different heterogeneities may be misplaced. We briefly discuss ongoing data collection projects designed to help make the link between contact patterns and the spread of influenza.

### **Damned if you do, damned if you don't: non-target pathogen effects on viral resistance transgenes in wild gourds**

Matthew Ferrari, Penn State University

There is increasing concern about the escape of transgenes that convey resistance to natural enemies from cultivated crops to wild species. Establishment and spread of a resistance gene in wild populations depends on both the fitness costs associated with resistance and the ecological context that mediates those costs. We study the introgression of a viral resistance transgene from cultivated squash to their wild con-specific, *Cucurbita pepo* ssp *texana* in the presence of a non-target pathogen. *C. pepo* is affected by two major classes of vector-transmitted pathogens: viruses transmitted by generalist aphids and the bacterium, *Erwinia tracheiphila*, transmitted by specialist cucumber beetles. The beetle vectors of *Erwinia* selectively forage on large plants, resulting in an increased mortality risk due to bacterial infection. Viral infection, though rarely fatal, results in reduced growth and fecundity, which reduces visitation by the vectors of *Erwinia*. Thus, in the presence of both pathogens, virus resistant transgenics have an increased risk of a fatal bacterial infection. From common garden experiments with mixed fields of wild-type *C. pepo* and transgenic hybrids, we show that selective foraging by vectors results in a counter-intuitive cost to viral resistance in the context of the full pathogen community.

### **How well can immune systems multi-task, and how well should they?**

Andrea Graham, University of Edinburgh and Princeton University

Mammalian hosts faced with multiple parasites, whether in series or in parallel, often benefit or suffer from co-infection, depending upon the extent to which the parasites share anatomical compartments, antigenic signatures, or sensitivity to immune effector mechanisms. This suggests that the immune system doesn't always respond specifically to each parasite. Given that mammalian immune systems presumably evolved in the presence of a web of parasites, why do they not multi-task perfectly? I propose three overlapping possibilities. First, immune multi-tasking may be required only when resource competition among parasites is minimal. Natural selection for greater multi-tasking ability may thus have been buffered by other mechanisms of parasite population control. Second, there are limits to the resolution at which both immunological signals and parasite antigens can be

distinguished. Thus, even if perfect specificity were desirable, it may be unachievable. Third, blurred immunological 'vision' may even be optimal, as a bet-hedging strategy given unpredictable exposure to parasites. Natural selection may therefore have favored immune systems of intermediate response specificity. I will illustrate these points with examples from helminth-malaria co-infections of mice. Deeper understanding of how well immune systems can and should multi-task may allow better prediction of the dynamics of multi-parasite interactions.

### **The transmission dynamics of influenza**

Sunetra Gupta, Oxford University

Co-authors: Mario Recker, Oliver G Pybus, Sean Nee

It is commonly believed that influenza epidemics arise through the incremental accumulation of viral mutations, culminating in a novel antigenic type that is able to escape host immunity. Successive epidemic strains therefore become increasingly antigenically distant from a founding strain. Here, we present an alternative explanation where, because of functional constraints on the defining epitopes, the virus population is characterized by a limited set of antigenic types, all of which may be continuously generated by mutation from preexisting strains and other processes. Under these circumstances, influenza outbreaks arise as a consequence of host immune selection in a manner that is independent of the mode and tempo of viral mutation. By contrast with existing paradigms, antigenic distance between epidemic strains does not necessarily accumulate with time in our model, and it is the changing profile of host population immunity that creates the conditions for the emergence of the next influenza strain rather than the mutational capabilities of the virus.

### **The Aspergillus-Seafan Epizootic: Modelling a Moving Target and the Evolution of Resistance**

Drew Harvell, Cornell University

Co-authors: Steve Ellner, John Bruno, Kiho Kim

Although coral disease has taken a large toll on populations, some species, like seafans, showed impressive resilience in the face of a massive epizootic. Caribbean seafans passed through a massive 7 year epizootic, marked by high prevalence of infection, high mortality rates and almost complete reproductive failure of infected fans. Monitoring of marked fans revealed that the host-pathogen interaction is a moving target, characterized by an early highly prevalent, virulent phase, and a later period of endemicity and recovery. Population structure just after the epizootic peak in 1997 showed a paucity of recruits compared to six years later. One hypothesis for the absence of recruits is reproductive suppression observed in infected fans, which made up to 70% of the peak epizootic population. The combination of failed recruitment and high adult mortality created strong selection for the evolution of resistance. A prominent hypothesis for decline of the epizootic, that hosts became resistant under such intense selection, is supported by modeling results. We examined whether host evolution could proceed quickly enough to explain the observed decrease in prevalence from an average of 30- 50% in 1997 to <10% by 2003. The model predicts that the initial size dependent infection risk will decline over time as large fans become preferentially infected and large susceptibles are more rapidly weeded out. Thus, host evolution could explain both the decline of the epizootic and the change from strongly-size dependent to weakly size-dependent infection risk as the epizootic progressed.

### **Cholera in Bengal: multiple transmission modes, multiple infection outcomes**

Aaron A. King, University of Michigan

In endemic regions, the human-*Vibrio cholerae* system is complex. Infections are initiated via contact with an environmental reservoir as well as through more direct contact. Environmental drivers play important modulatory roles in the reservoir, in transmission, and in herd immunity at a range of timescales from seasonal to interannual. The outcomes of infections are far from stereotypical, with many mild infections for every recorded case. Finally, the nature and importance of protective immunity to cholera are incompletely understood. In this talk, we will shed light on these issues using competing mechanistic models confronted with recent and historical time-series records of cholera incidence and mortality.

### **Bacteria-phage coevolution in the phyllosphere**

Britt Koskella, University of Oxford

Co-Authors: Angus Buckling, University of Oxford and John Thompson, University of California, Santa Cruz

Recent examination of host-parasite interactions has shown that coevolution occurs in relatively short time periods and is strongly influenced by the spatial structure of populations. This relationship between patterns of coevolution and spatial heterogeneity is particularly relevant given the increasing frequency of species dispersal via human movement. The tritrophic interaction between plants, bacteria, and bacteriophage is ideal for establishing the scale and magnitude of coevolutionary events; bacteriophage influence both the motility and population size of bacteria, and thus alter the impact of bacteria on the host plant. A recent increase in the spread of the bacterial pathogen *Pseudomonas syringae* pathovar *aesculi* across Horse chestnut trees (*Aesculus* species) in the UK has left the population at serious risk. One promising control option is phage therapy, which utilizes naturally-occurring, virulent phage isolates to suppress the reproduction and spread of bacteria. We examined parasite local adaptation across multiple spatial scales using bacteria and phage isolated from Horse chestnut trees across Oxfordshire. The results show that phage are strongly locally adapted at the scale of individual trees, but not across leaves within a tree. These results indicate that bacteria-phage coevolution is occurring despite the complexity of bacterial dispersal and population dynamics within the phyllosphere.

### **Zoonotic spillover and the dynamics of human monkeypox**

James Lloyd-Smith, University of California, Los Angeles

Spillover transmission from animal reservoirs to human populations plays a central role in the ecology of zoonotic infections, but has been largely neglected in the study of zoonotic dynamics. I will present analysis of the spillover dynamics of human monkeypox, a zoonotic infection prevalent throughout central and west Africa. Monkeypox is also a prototype emerging zoonosis, with concern centering on its potential to fill the niche left vacant by smallpox eradication. By considering the degree of human-to-human transmissibility - and how it changes as smallpox vaccination coverage declines - it is possible to identify the contribution of zoonotic spillover to total incidence and to extract the temporal profile of spillover transmission. Analysis of historical and contemporary data sets yields insights into the distribution of risk in the human population and the future epidemic potential of monkeypox. These methods will be applicable to a broad class of multi-host pathogens exhibiting limited transmission in the secondary host species.

## ***Adaptation of zoonotic pathogens to human transmission***

Angela McLean, Oxford University

Novel emerging infections are almost all of zoonotic origin. Many zoonoses cross the species barrier into humans but never transmit from one human to another. Others transmit very occasionally or even often enough to cause short chains of transmission. Only rarely are zoonoses efficient enough at transmitting between people that they are able to cause epidemics of novel emerging infections, but when they do the consequences can be devastating. Investigating the processes by which novel infections of humans adapt to their hosts is an important part of understanding the process of disease emergence. It is not just a question of within-host evolution because pathogens must adapt to a series of different hosts in a chain of transmission and not all hosts will exert the same selection pressures. Models of the process must therefore encompass both within-host evolution and between-host transmission. Motivated by the ongoing evolution of HIV and the putative evolution of pandemic influenza I will present mathematical models of the process of adaptation to human transmission. For HIV the data pose questions about the long-term outcome of viral adaptation to immune selection pressures. We can use simple models to understand how within-host processes will govern long-term epidemiology. For influenza we use our models to address questions about how to detect dangerous emergence events in which viruses acquire the ability to transmit efficiently between people.

### **Immunoepidemiology of a gastrointestinal nematode in single and co-infections**

Lisa Murphy and Isabella Cattadori, University of Glasgow and Penn State University

Immuno-epidemiology is a powerful tool for understanding the mechanisms affecting host-parasite interactions in individuals and for explaining the consequences of such interactions at the population level. Our studies into the dynamics of nematode infection in a free living rabbit population sampled over a 30 year period suggested that rabbits develop a strong acquired immune response to *Trichostrongylus retortaeformis* infection. Using experimental infections we have now examined this host-parasite interaction in more detail allowing us to clearly identify the immune-mediated mechanisms of regulation and confirm our epidemiological results. Over the course of the infection we have recorded immunological (antibody response and cytokines), parasitological (biometry, fecundity, intensity), haematological and histological data. Our results confirm that rabbits develop both a strong systemic and localised immune response to *T. retortaeformis* with parasite-specific IgA and IgG antibody production peaking around 25 days post infection. By around 60 days the infection is cleared. Despite this rapid response however the nematode is able to mature and shed eggs before this immune response is fully established. These findings provide valuable information allowing us to develop a more accurate model of single nematode infection in wild rabbit populations.

### **Canine influenza and canine parvovirus as models of viral host switching**

Colin Parrish, *Baker Institute, College of Veterinary Medicine, Cornell University*

*Co-authors:* Karin Hoelzer, Edward Holmes

We are studying two models where an animal virus has shifted its host range. One is a parvovirus (feline panleukopenia virus (FPV)) which mutated to form canine parvovirus (CPV), and the other is the H3N8 canine influenza virus (CIV) which arose as a variant of the equine influenza virus. Both of these host-shifting viruses arose from viruses of other hosts which crossed the host range barrier and established epidemic infections in dogs. We are examining the evolutionary events that were associated with the emergence of each of

these viruses, and are correlating the dynamic changes found in the sequences (in both populations and individual infected animals) with studies of the virus functions. For the CPV event we are examining the specific roles of the receptor binding and changes in capsid-receptor affinity, and are correlating those to changes in the antigenic structure of the capsids. For the CIV event we are examining the variation in the virus and correlating changes with the known antigenic sites of the viruses, and are also beginning to examine the interactions of the viruses with the specific sialic acids of dog and horse tissues.

### **Urban habituation, ecological connectivity and epidemic dampening: the emergence of Hendra virus from flying foxes (*Pteropus spp.*)**

Raina Plowright, Penn State, Center for Infectious Disease Dynamics and Consortium for Conservation Medicine

Co-authors: Patrick Foley, Hume Field, Andy Dobson, Janet Foley, Peggy Eby, Peter Daszak

Anthropogenic environmental change is often implicated in the emergence of new zoonoses from wildlife, however there is little mechanistic understanding of these causal links. Here we model the transmission dynamics of a recently-emerging zoonotic paramyxovirus, Hendra virus (HeV), in its endemic host, Australian *Pteropus* bats (fruit bats or flying foxes). HeV is a Biosecurity Level 4 (BSL-4) pathogen, with a high case fatality rate in humans and horses. With a model parameterized from field and laboratory data, we show that the recent emergence of HeV may be explained by urban habituation and decreased migration—two widely observed changes in flying fox ecology that result from anthropogenic transformation of bat habitat in Australia. Urban habituation increases the number of flying foxes in close contact with human and domestic animal populations, and in addition, our models suggest that decreased bat migratory behavior leads to a decline in population immunity, giving rise to more intense outbreaks after local viral reintroduction. Hence, flying fox population connectivity may actually dampen the intensity of outbreaks and reduce zoonotic events. Our findings provide the first detailed mechanistic explanation of the sporadic temporal pattern of HeV emergence, and of the urban/peri-urban distribution of the eleven known HeV outbreaks in horses and people.

### **Reversal of Fortune: Human Pathogens Killing Coral in the Florida Keys**

James W. Porter (UGA, School of Ecology) and Erin K. Lipp (UGA, School of Public Health)

The movement of disease from wildlife populations into human populations is well documented. We report here on a reverse infection of the threatened elkhorn coral species, *Acropora palmata*, by the human enterobacterium, *Serratia marcescens*. Previous application of Koch's Postulates had identified this bacterium as the causal agent of white-pox disease, which has decimated coral populations in the Florida Keys and elsewhere around the Caribbean. Our current analysis demonstrates that the specific strain that kills coral comes from humans, not from other potential vertebrate vectors, such as local populations of sea gulls, key deer, cats, or fish. Advanced waste water treatment is effective at removing this bacterium. Localities in the Florida Keys that have upgraded their wastewater treatment plants to AWT standards are currently without white-pox; localities without AWT facilities continue to have active white-pox outbreaks.

## **Environmental transmission and the ecology of avian influenza viruses**

Pejman Rohani, University of Georgia

Understanding the transmission dynamics and persistence of avian influenza viruses (AIVs) in the wild is an important scientific and public health challenge because this system represents both a reservoir for recombination and a source of novel, potentially human-pathogenic strains. The current perspective locates all important transmission events on the nearly-direct fecal/oral bird-to-bird pathway. In this talk, I review overlooked evidence, based on which I propose that an environmental virus reservoir gives rise to indirect transmission. This transmission mode could play an important epidemiological role. Using a stochastic model, I will examine how neglecting environmentally generated transmission chains could underestimate the explosiveness and duration of AIV epidemics in addition to its long-term persistence. This phenomenon is shown to have potentially important pathogen invasion implications: the non-negligible probability of outbreak even when direct transmission is controlled, the long-term epidemic potential of previously infected sites, and the role of environmental heterogeneity in risk. Finally, I will speculate on the implications of environmental transmission for spread among networks of hosts, virus evolution and subtype diversity.

## **Antigenic drift of influenza viruses**

Derek Smith, Cambridge University

Thirty plus years of global influenza virus surveillance, in multiple species, provides a remarkable dataset for the study of influenza virus evolution. Because the purpose of much of this surveillance is vaccine strain selection, these data have been analyzed antigenically as well as genetically. I will describe the antigenic evolution of influenza A(H3N2) viruses from the influenza pandemic in 1968 to present, including examples of the influenza vaccine strain selection process. This evolution of human influenza viruses will be contrasted with studies in pigs and birds, to show the importance of the immunity in the host population on the evolution of the virus.

## **Exotic Orbiviruses in the United States: The Invasion of an Occupied Habitat**

David E. Stallknecht and Andrew B. Allison

Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia

Since 2004, we have detected four exotic orbiviruses in white tailed deer (*Odocoileus virginianus*) in the United States: bluetongue virus (BTV) serotypes 1, 3, and 12, and epizootic hemorrhagic diseases virus (EHDV) serotype 6. Although these introductions mirror recent BTV and EHDV introductions into Europe and the Mediterranean region, the United States situation is unique in that numerous indigenous BTV and EHDV are already present; indigenous viruses include BTV-2, -10, -11, -13, and -17 as well as EHDV-1, and -2. As multi-host and vector-borne viruses, the successful colonization of a new region by BTV and EHDV may be dictated by multiple adaptive events. Host related barriers not only relate to efficient replication within individual (and possibly new) hosts but also in minimizing the effects of acquired immunity within host populations. Invading viruses also have to contend with new vector species, and in the case of BTV, there is some evidence of adaptation to new *Culicoides* vectors. Of all of the exotic orbiviruses detected in the US to date, EHDV-6 has been detected most frequently over the widest geographic area. Although we do not know the source of the prototype introduced EHDV-6, genetic analyses of these viruses indicate that these US strains are a product of reassortment between EHDV-2 and EHDV-6. While these viruses are antigenically unique, they possess many of

the conserved genes associated with indigenous EHDV-2 including the viral protein (VP7) that is associated with cell surface attachment and infection in *Culicoides* vectors.

### **Virulence tradeoffs in a vertebrate virus**

Andrew Wargo, University of Washington

Co-authors: Gael Kurath and Ben Kerr

Pathogens can interact with each other on many levels. On one of the most basic levels, different genotypes of a particular pathogen species can interact in their host, much in the same way that different parasite species might interact. We are interested in how multiple genotypes of the pathogen, infectious hematopoietic necrosis virus (IHNV), that differ in virulence, interact in their natural host, rainbow trout. We were curious to see how virulence might tradeoff with viral fitness traits such as entry into the host, replication, competition, and transmission; and what impact this might have for virulence evolution. We conducted *in vivo* experiments to measure these IHNV fitness parameters by infecting fish with a low virulent genotype, a high virulent genotype, or a mixed infection of the two virus genotypes. We then measured in host viral load and transmission through the course of infection for the individual genotypes. Our results suggest that the more virulent genotype of the virus had greater overall fitness and out competed the less virulent genotype primarily due to higher in host replication. We also found that other fitness traits such as entry and shedding, might tradeoff with virulence.

## POSTER ABSTRACTS

*In alphabetical order by last name*

### **#1 Ecological and evolutionary determinants of disease distribution in a natural host-pathogen system**

Jessica L. Abbate

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The ability to predict range limits for pathogens and parasites offers an important tool for targeting disease prevention and control strategies. In building these models, it is important to understand the relative importance of both ecological and evolutionary factors that have been shown to be important for species range distributions in general, along with components dictating disease spread and persistence. Though the importance of the environment on host-pathogen interactions has been well-characterized in agriculture, there have been very few studies incorporating its contribution to disease in natural systems. Changes in transmission dynamics and resistance structure across ecological gradients may act to further restrict the distribution of ecologically suitable habitats. This research will investigate what factors determine the restriction of another smut disease to high-elevation populations within the natural range of the host, *Silene vulgaris*. It is possible that the hosts have separate evolutionary histories and only occupy contiguous habitats more recently, that the lower-elevation hosts are more resistant to disease, or that altitudinal conditions are impacting pathogen transmission, infectivity, or resistance costs. Patterns of host population structure, variation in resistance, pathogen infectivity, disease response to environmental conditions, and ecological niche modeling are all being employed to evaluate these competing hypotheses.

### **#2 Environmental Investigation of a Case of Murine Typhus in Los Angeles.**

Kyle F. Abramowicz<sup>1,2\*</sup>, Michael P. Rood<sup>3</sup>, Marina E. Ereemeeva<sup>1</sup>

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Classic murine typhus, due to *Rickettsia typhi*, is endemic in the continental U.S. in areas of Texas and southern California. We conducted an environmental investigation of a murine typhus case occurring in an urban area of Los Angeles. Four *Rattus norvegicus* heavily infested with *Xenopsylla cheopis* (average 32.5 fleas per animal, range 20-42) were trapped and fleas, blood and tissues were collected. DNAs from all specimens were tested for *R. typhi* and *R. felis* using a Taqman assay targeting the rickettsial citrate synthase gene. Although rickettsiemia was not detected, DNA of *R. felis* was detected in at least one tissue from each rat. Tissues from 3 rats were also positive for *R. typhi* DNA. *Rickettsia typhi* and *R. felis* DNA was detected in fleas collected from each animal with average minimal infection rates of 10% and 32.3%, respectively. This suggests that while *R. typhi* circulates in urban Los Angeles, *R. felis* may be more widespread in flea and rodent populations. Some strains may be more infectious than previously thought for rats and, possibly, humans.

### **#3 Persistence of canine distemper virus in the Greater Yellowstone Ecosystem's carnivore community**

Emily AlMBERG, University of Minnesota

Co-authors: Paul C. Cross (USGS & Montana State University)  
Douglas W. Smith (National Park Service)

Canine distemper virus (CDV) causes acute, highly immunizing infections among its wide range of carnivore hosts. Repeated outbreaks of CDV among Yellowstone National Park's (YNP) wolves (*Canis lupus*), coyotes (*Canis latrans*), and cougars (*Puma concolor*) prompted questions as to how, where, and at what scale CDV might be persisting in the regional carnivore community. Using several stochastic, spatially-explicit susceptible-exposed-infectious-recovered (SEIR) simulation models, we determined that (1) current wolf populations in the Greater Yellowstone Ecosystem (the larger ecosystem surrounding YNP) are too small to support endemic CDV, (2) that under the assumption that coyotes are the primary reservoir host, there would need to be between 5,000 - 10,000 packs of coyotes, or between 50,000 and 100,000 individuals, to have a 50% probability of pathogen persistence over 10 years, and (3) the inclusion of a second host species, capable of inter-species transmission, can greatly increase the probability of long-term CDV persistence, particularly at relatively small spatial scales. Furthermore, spatial connectivity and demographic turnover within simulated host populations affected both local epidemic dynamics, such as the length and variation in inter-epidemic periods, and pathogen persistence. Given the small group sizes of carnivores and their annual reproductive pulses, CDV probably requires multi-host transmission for long-term persistence.

#### **#4 Manipulative Experiments Exploring Transmission of a Zoonotic Pathogen in its Host Population**

Karoun H. Bagamian, Emory University

Co-authors: Richard J. Douglass, James N. Mills

Studies of the relationship between zoonotic pathogens and their reservoir host populations have yielded conflicting data regarding the effects of infection on the health of the host, as well as the relationship between host population density and disease prevalence. Previous investigations of these questions have utilized theoretical models and/or descriptive studies. In the summers of 2007 and 2008, we tested these two questions by running large scale manipulative field experiments in outdoor enclosures using the deer mouse (*Peromyscus maniculatus*)-Sin Nombre hantavirus host-parasite system. We constructed six 0.10 hectare enclosures in Butte, Montana. One infected and a predetermined number of uninfected adult wild deer mice were released into each enclosure to create "high" or "low" density populations. Mice were then trapped weekly or bi-weekly to monitor their infection status and individual characteristics. We will present preliminary results on effects of host population density on frequency of transmission events and other relevant variables between our high and low density populations, and any effects of SNV infection on measures of general health (weight gain, reproductive status, etc.) in our experimental population.

#### **#5 Lemur disease ecology: Linking health, ecosystem viability and conservation in Madagascar**

Meredith Barrett<sup>1</sup>; Randall E. Junge<sup>2</sup>

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Anthropogenic effects on ecosystems have reached unprecedented levels, with potentially disastrous consequences for global environmental, wildlife and human health. Human encroachment into wildlife habitat leads to degradation and fragmentation, and increases

contact among wildlife, humans and domestic animals. With Madagascar's rampant rate of human population growth and deforestation, the widespread presence of domestic and invasive species, and its evolutionary isolation, it can be viewed as a potential location for future disease risk. Consistent baseline health monitoring of lemur populations provides an effective tool for evaluating health and preparing for future disease occurrences. Limited, disconnected surveys of lemur health have been conducted, yet there remains a need for more extensive, country-wide evaluations that also assess diseases of invasive species, domestic animals and humans. Our study investigates how spatial, climatic, anthropogenic and conservation factors affect the occurrence of infection throughout Madagascar. We have evaluated body condition, endo- and ectoparasite richness and prevalence, and viral serology data from 528 lemurs within 15 reserves. Currently we are incorporating these data with habitat characteristics and evaluations of the human pressures affecting each site. While still conducting the analyses, we predict that lemurs in smaller reserves that lie within 5 kilometers of human settlements suffer higher parasite prevalence, richness, frequency of multiple infections, and reduced body condition.

## **#6 Effects of Global Change on the Prevalence of a Seagrass Pathogen**

Gabriela Blohm, Lindsey Albertson and J. Emmett Duffy

The abiotic environment of many biological communities has changed rapidly during the last century and pathogen outbreaks have often occurred alongside such changes. Coastal ecosystems are of particular interest because they are important for local economies and are experiencing changes in temperature, nutrient levels and community structure. Additionally, reports of a 'catastrophic' seagrass wasting disease date as far back as 1933 and have increased nearly two-fold during the past 20 years. Seagrasses provide a number of economic and ecological services, thus their conservation is important. To identify the ecological factors that best explain these outbreaks in the temperate seagrass *Zostera marina*, we manipulated temperature, nutrient levels and grazer density in a fully factorial mesocosm experiment. We monitored seagrass primary production, senescence and necrosis due to *Labyrinthula zosterae*, the reported causative agent of wasting disease in *Z. marina*. Previous correlational field studies suggest that increased temperature will lead to more frequent disease outbreaks. We found that nutrient enrichment had a larger effect on disease prevalence than increased temperature and herbivore damage. Our results suggest that changes in nutrient availability in seagrass ecosystems provide an ecological advantage to microbial pathogens. Reducing the frequency of wasting disease outbreaks in seagrass ecosystems will require a decrease in nutrient inputs to these ecosystems.

## **#7 Opposing seasonal changes in adaptive and innate immunity in African Buffalo (*Syncerus caffer*)**

Brianna Beechler<sup>1</sup>, Austin Bell<sup>1</sup>, Vanessa Ezenwa<sup>2</sup>, Anna Jolles<sup>1</sup>

1. Oregon State University, College of Veterinary Medicine and Department of Environmental Sciences, Corvallis OR
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We investigated patterns of adaptive and innate immunity in free-ranging African buffalo in relation to season, host traits (age, reproductive status, body condition) and infection profiles (bovine tuberculosis (BTB), gastrointestinal helminths, coccidia, ticks). We used an in-vitro assay measuring bactericidal killing ability of whole blood to assess innate immunity; and cytokine (IFN $\gamma$ ) release in response to in-vitro stimulation with a novel antigen (pokeweed mitogen) as an index for adaptive immunity. Innate and adaptive immunity showed contrasting associations with host traits and infections: Bactericidal activity of blood was negatively associated with tick burden, pregnancy and lactation; while

cytokine responses were more pronounced in older animals and those with heavier worm burdens. Neither innate nor adaptive immune responses showed any association with host body condition, TB infection or coccidia. Finally, we tested whether tradeoffs between adaptive and innate immunity are detectable in our study population. Our data revealed a weak negative association between innate and adaptive immunity within individual hosts; and a robust seasonal pattern at the population level, with innate immunity up-regulated, but adaptive immunity down-regulated in the dry season as compared to the wet season. This is consistent with the notion that animals under resource restriction may invest in less costly immune defenses such as complement activity rather than metabolically expensive adaptive responses.

## **#8 Herd Immunity Acquired Indirectly From Interactions Between the Ecology of Infectious Diseases, Demography, and Economics**

Matthew H Bonds (Harvard School of Public Health) and Pejman Rohani (UGA)

Patterns of morbidity and mortality around the globe are determined by interactions between infectious diseases and systematic human socioeconomic processes. The most obvious of these patterns is that the greatest burdens of infectious diseases are found among the poor who lack the basic resources for disease prevention and treatment. Yet, it is becoming increasingly clear that many infectious diseases are themselves causes of poverty due to their effects on labor productivity. A subtle phenomenon that receives little attention in the epidemiology literature and is especially important for poor communities is the role of the birth rate as an important direct cause of high disease burdens. Because of their high rates of transmission and life-long immunity, the persistence of many child diseases such as measles, rely on high rates of reproduction as their source of susceptible individuals. Thus, there are significant direct health benefits of lower fertility rates, which are further enhanced by interactions with economic processes. Indeed, fertility, poverty and disease all interact with each other in important and predictable ways that can be built into traditional disease ecology models. We present such a model here that provides insights into the long-term effect of policy interventions. For example, because of indirect income effects, herd immunity may be acquired with lower vaccine coverage than previously thought. It can also be acquired simply through reductions in the birth rate. Our model thus provides a disease ecology framework for the analysis of demographic transitions.

## **#9 When is enough sampling enough? Population dynamics and zoonotic infectious disease: the case of deer mice, *Peromyscus maniculatus*, and Sin Nombre virus**

Scott Carver, Montana Tech of the University of Montana

Field biologists face decisions about the optimization of sampling strategies which minimize effort to benefit. In general, "more sampling effort", where possible, is considered better. Few studies however, examine "what is the smallest amount of sampling that is sufficient for our purpose?" We use a longitudinal study of deer mouse populations and prevalence of antibody to Sin Nombre virus (SNV, Bunyaviridae: Hantavirus; the agent of hantavirus pulmonary syndrome in humans) to assess how a reduction in sampling frequency influences the ability to document population demographics of deer mice and dynamics of SNV infection. Data were collected monthly (since 1994) at three trapping grids near Cascade Montana. We examined how sampling every two months, quarterly, bi-annually and annually were related to the average and variance of deer mouse abundance and dynamics of SNV (number of antibody-positive deer mice and antibody prevalence). We also examined how this reduction in sampling frequency influenced detection of annual extremes in deer mice and SNV antibody (highs and lows). Results from less frequent sampling were

correlated with average and variance in deer mouse abundance and dynamics of SNV antibody. Reduced sampling frequency provided conservatively similar estimates of average deer mouse abundance, but progressively overestimated the average number of deer mice with antibody and the average antibody prevalence. Reduced sampling frequency also resulted in detecting fewer annual extremes of deer mouse abundance and infection. Our study indicates that average deer mouse population abundance over time can be followed with reasonable accuracy while sampling less frequently than monthly. Sampling effort necessary to capture temporal dynamics of SNV infection however, differ from effort necessary to capture demographic patterns in deer mouse abundance. Findings in this study may be more widely applicable to sampling strategies for other hosts and their pathogens, and be helpful to ecologists, wildlife biologists and public health officials dealing with emerging infectious diseases.

## **#10 Evolutionary Dynamics of Class II avian Paramyxoviruses Type 1 (PMV-1)**

Yee Ling Chong<sup>1</sup>, Abinash Padhi<sup>1</sup> and Mary Poss<sup>1,2</sup>

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The Paramyxovirus type 1 (PMV-1), a non-segmented single-stranded negative sense RNA virus, causes diseases in respiratory, gastrointestinal tract and/or the nervous system in wide range of avian species. This study was undertaken to understand the evolutionary dynamics of class II PMV-1 that are maintained in both wild and domestic bird populations. There are two distinct classes of PMV-1 (class I and II) with class II further categorized into nine distinct genotypes. Taking all existing full length genomes, our ML-based phylogenomic analyses revealed seven distinct genotypes that are clustered into two major lineages. While genotypes I and II, which comprised all the vaccine strains formed a unique cluster, the remaining genotypes which comprise the more recent isolates appeared to be monophyletic. We demonstrate phylogenetic evidence for single and multiple recombinant events from multi-avian host PMV-1 isolates. Interestingly, the regions of mosaic structures driven by recombination breakpoints were clustered at hotspot regions between NP, P and M genes. We also indentify potential inter-lineage parental strains and parental vaccine strains which could be involved in the recombination. This result was further supported by phylogeny-based incongruence test. Bayesian estimates of the evolutionary rates of each genomic region of class II PMV-1 are within the range of 0.72 - 4.5 × 10<sup>-3</sup>/site/year, which are consistent with the high rates of evolution of other previously reported negative ssRNA viruses. The divergence of all the six genomic regions of class II PMV-1 was consistent and began around late-1800 to early-1900. Bayesian skyline analyses showed a gradual increase in effective population size, in terms of relative genetic diversity till the year 1990 followed by an abrupt decline thereafter.

## **#11 General patterns of covariance among host physiological traits predict key epidemiological traits**

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We hypothesize that key epidemiological traits, namely host susceptibility, competence, and tolerance to infection, are causally determined by general patterns of covariance among host physiological traits. In plant and animal communities, host phenotypic variation

represents trade-offs among traits that may influence host-vector-pathogen interactions. For example, 'fast-living' animal phenotypes may invest less in immunological defenses compared to 'slow-living' phenotypes. Similarly, 'quick-return' (QR) plant phenotypes likely invest less in resistance to vector attack and more in tolerating damage compared to 'slow-return' (SR) phenotypes. Despite these patterns, direct tests linking host phenotypic variation to epidemiological traits are lacking. We therefore predicted that our plant species would vary along a QR-SR phenotypic continuum and that host susceptibility, competence, and tolerance would be greater in QR phenotypes. In greenhouse experiments, we quantified phenotypes of six California grass species and used aphid vectors to experimentally inoculate hosts with barley yellow dwarf virus (PAV). We show that QR phenotypes supported greater vector populations, were more likely to become infected when inoculated (i.e., were more susceptible), and produced more infected vectors (i.e., were more competent). Moreover, QR phenotypes were more tolerant of infection. These results suggest that host physiological phenotype may predict key epidemiological traits and therefore disease risk, transmission, and prevalence may be greater in QR dominated communities.

## **#12 Mycoplasmal Conjunctivitis in House Finches: Spatio-temporal Pathogen Evolution**

André Dhondt, Wesley M. Hochachka, Irby Lovette, Karel A. Schat, Evan Cooch, Andy Dobson, Erik Osnas, Dana Hawley, David Ley

As part of our study of spatial and temporal variation in disease prevalence, we are examining the role that pathogen evolution has played in apparent differences in prevalence and virulence from eastern to western North America.

## **#13 Filter-Feeding Bivalves Can Remove Avian Influenza Virus from Water and Reduce Infectivity**

Christina Faust, University of Georgia

Co-authors: David Stallknecht, David Swayne, and Justin Brown

Avian influenza (AI) viruses are transmitted within wild aquatic bird populations through an indirect fecal-oral route involving fecal-contaminated water. In this study, the influence of filter-feeding bivalves, *Corbicula fluminea*, on the infectivity of AI virus in water was examined. A single clam was placed into a flask (n=48) with distilled water inoculated 1:100 with a low pathogenic AI virus (A/Mallard/MN/190/99 (H3N8)). Water samples were collected prior to inoculation and at time points up to 96-hr post inoculation (PI) for titration in chicken embryo fibroblasts. Viral titers in water with clams were significantly lower at 24- and 48-hr PI compared to viral-infected water without clams (n=9); however, the onset and rate of viral titer reduction varied between individual trials. To determine if AI virus that was removed from the water by clams remained infective 18 wood ducks (*Aix sponsa*) were divided into six test groups and intranasally or orally inoculated with a variety of treatments of clam supernatant, whole clams, and water exposed to A/whooper swan/Mongolia/244/05 (H5N1). None of the wood ducks inoculated with H5N1 HPAI-infected water that was filtered by clams or that were inoculated or fed tissue from these clams exhibited morbidity or mortality. All of the wood ducks exposed to H5N1 HPAI-infected water control flasks (without clams) and the original inoculum died. These results indicate that filter-feeding bivalves can remove and reduce the infectivity of AI viruses in water, and demonstrate the need to examine biotic, in addition to abiotic, environmental factors that can potentially influence AI virus transmission.

## **#14 Beyond the dilution effect: Determinants of *Trypanosoma cruzi* vector infection index in a deforested landscape"**

Nicole Gottdenker<sup>1</sup>, Jose Calzada<sup>2</sup>, Anamaría Santamaría<sup>2</sup>, Azael Saldaña<sup>2</sup>, Humberto Membache<sup>2</sup>, and C.R. Carroll<sup>1</sup>

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In Panama, deforestation is associated with increased vector abundance and vector infection prevalence with the multi-host parasite *Trypanosoma cruzi*, agent of Chagas disease in humans. This study evaluates two potential mechanisms by which deforestation may lead to increased vector infection prevalence with *T. cruzi*: a change in reservoir host community structure and vector infection with a congeneric parasite, *Trypanosoma rangeli*. Vector infection with *T. cruzi* and *T. rangeli* were determined by a duplex polymerase chain reaction assay. Species composition of reservoir hosts was indirectly determined by amplification and sequencing of a vertebrate-specific 12SrRNA gene from vector blood meals. Vector infection prevalence and vector blood meal composition were compared across areas with varying degrees of anthropogenic disturbance. There was no significant relationship between host species diversity and *T. cruzi* vector infection prevalence. However, host species composition differed in protected forest compared to deforested areas. As *T. rangeli* vector infection prevalence decreased in deforested areas, *T. cruzi* infection increased. Complex interactions between vector and host co-infection with *T. cruzi* and *T. rangeli*, such as immune-mediated competition, and differences in reservoir host community composition may account for the landscape-related changes in *T. cruzi* vector infection and potential human disease risk.

## **#15 Testosterone drives parasite exposure and transmission potential in wild mice**

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Male-biased infection is a common phenomenon in vertebrate-parasite systems and male-biased transmission has been experimentally demonstrated. One mechanism that is hypothesized to create male-biased transmission is the immunosuppressive effect of testosterone because it increases susceptibility to infection. Testosterone also influences host behavior and, consequently, may increase exposure to parasites. To test how testosterone could increase exposure and transmission, we undertook a longitudinal mark-recapture study where we experimentally elevated testosterone levels in wild male rodents. Individuals in control populations reduced the average number of contacts over the treatment period in accordance with seasonal behavior, while populations with experimentally elevated testosterone levels maintained the number of contacts between hosts. As a result, the transmission potential was higher in testosterone treated populations compared to controls. Our results indicated that males with high testosterone levels alter the population level contacts, producing different social networks and increasing transmission potential compared to those where testosterone is at background levels.

## **#16 Why do antibody responses induced by helminths and malaria cross-react?**

Karen Grocock, University of Edinburgh

Co-authors: Dr. Tracey Lamb, Prof. Jean Langhorne, Prof Judi Allen, Dr Dave Shuker and Dr Andrea Graham

Do cross-reactive antibodies induced by distinct pathogens result from a constrained ability of the immune system to discriminate antigens, or are cross-reactive responses a deliberate bet-hedging strategy by hosts? If the antigenic distance between two taxonomically distant parasites is small, the immune system may not perceive them as different. Similarly, constraints such as lymphocyte number may prevent the production of a specific antibody for every antigen. To determine the relative strength of cross-reactive versus antigen-specific responses in mice co-infected with nematodes and malaria, we used ELISA to calculate antibody titre from a serial dilution of serum. We also examined whether cross-reactive responses were targeted toward carbohydrate or protein moieties by treating the parasite antigens with periodate, thus disrupting carbohydrate epitopes. Neither periodate treatment nor titration completely eliminated cross-reactive responses, suggesting that the antigenic distance between malaria and helminths may actually be small. However, the co-infected animals were slightly protected against malaria infection. Thus, cross-reactive responses, as described in this and other co-infection systems, raise the interesting question of whether production of cross-reactive antibodies might provide broad-spectrum protection for a host faced with infection by an unpredictable range of parasites.

### **#17 Molecular Characterization of Flu-Receptor – Hemagglutinin Interactions. Computational Prediction of HA Specificity.**

Jodi A. Hadden, University of Georgia

Co-authors: B. Lachele Foley, Loretta Yang, John P. Nolan, S. Mark Tompkins and Robert J. Woods

Hemagglutinin (HA) mediates attachment to and entry of influenza virus into host cells by binding to sialic acid receptors at the cell surface. Human influenza viruses preferentially bind to sialic acid linked to galactose by  $\alpha$ -(2-6) linkages; the main type found on the epithelial cells of the human upper respiratory tract. Avian viruses tend to bind to  $\alpha$ -(2-3) linkages that are found predominantly on avian intestinal epithelium [1]. All influenza A viruses that have infected mammals emerged as some point from avian species [2]. Changes in the amino acid sequence of HA can alter the sialic acid specificity of influenza viruses, with the change of one or two amino acids [3] being sufficient to change the receptor binding specificity and affect interspecies transmission barriers.

We report computational simulations using the GLYCAM force field [4] of human and avian receptor – HA complexes, based on structural data for the human 1934 H1 influenza strain [3]. The theoretical methods correctly identify H1 HA as selective for human  $\alpha$ -(2-6) linkages and provide insight into the origin of the affinity differences, but also indicate limitations of current simulation methods. In conjunction with computational methods, microparticle-based flow cytometry assays will be developed to quantitatively analyze influenza virus receptor specificity.

### **#18 Impact of forest canopy disturbance on mosquito vector and vertebrate host communities in southwestern Virginia.**

M. Camille Harris DVM, MS and Dana M. Hawley PhD

*Department of Biological Sciences, Virginia Tech, Blacksburg, VA*

La Crosse encephalitis virus (LAC) is maintained through a natural transmission cycle between mosquitoes and small mammals in hardwood forests. Forest canopy disturbance (FCD; i.e., silviculture and deforestation) has been shown to impact the dynamics of mosquito-borne malaria parasites. Its influence on arboviruses, however, is unknown. The

goal of our research is to assess the influence of FCD on LAC dynamics. Here, we present FCD impacts on the mosquito vector and vertebrate host communities. 45% (5/11) of collected mosquito species are known arboviral vectors. General linear models revealed significant FCD effects for *Cx. pipiens/restuans* ( $p=0.0001$ ), *Ae. triseriatus* ( $p=0.0006$ ) and *Oc. japonicus* ( $p=0.0085$ ). The former was most abundant on shelterwood and the latter two on undisturbed forest sites. Of the most abundant avian species, two showed a significant preference for undisturbed forest: *Contopus virens* ( $p=0.0037$ ) and *Sitta carolinensis* ( $p=0.027$ ). Small sample size prevented analysis of mammal species richness across the sites.

### **#19 The influence of host and pathogen genetics on Mycoplasmal conjunctivitis virulence in House Finches (*Carpodacus mexicanus*)**

Dana Hawley (Virginia Tech), David Ley (NC State University), Andy Dobson (Princeton University), and Andre Dhondt (Cornell University).

Understanding the ecological and evolutionary factors that mediate pathogen virulence in natural systems is complicated by potential contributions of host, pathogen, and the environment. Here we take advantage of previously documented geographic variation in house finch (*Carpodacus mexicanus*) infection with the bacterial pathogen *Mycoplasma gallisepticum* in order to experimentally test whether host and/or pathogen traits contribute to lower observed virulence (e.g. pathogen-induced morbidity) in the western, native range of the host. We used a two-way factorial common garden experiment to simultaneously test effects of both host and pathogen genetics on virulence. We exposed equal numbers of wild-caught eastern (introduced) and western (native) house finches to strains of *M. gallisepticum* isolated in the eastern (1994) versus western range (2006). We detected no influence of host origin (native vs introduced house finches) on pathogen virulence, but the 2006 western strain of *M. gallisepticum* was significantly less virulent in both eastern and western hosts than the eastern strain, which was collected in 1994 during the initial stages of the epidemic. Our results indicate that pathogen rather than host variation underlie the lower virulence of Mycoplasmal conjunctivitis in the native range of the host. These results suggest that *M. gallisepticum* has evolved lower virulence, as measured by host pathology, as it spread from the eastern to western range of the host.

### **#20 Molecular Epidemiology of Feline Immunodeficiency Virus in the Domestic Cat**

Jessica Hayward (Cornell University) and Allen G Rodrigo

A large-scale epidemiological study of Feline Immunodeficiency Virus has been undertaken in populations of the domestic cat (*Felis catus*) in New Zealand. An FIV prevalence of 19 % ( $n = 416$ ) was established from lymph node samples of feral cats. Phylogenetic analysis of envelope gene sequences shows that known FIV subtypes A and C circulate in New Zealand cat populations. In addition, we have identified sequences of unknown subtype in all three amplified regions of gag, pol and env. Evidence of intragenic recombination has been found in all three gene fragments, with the highest levels in the env gene. Complex intergenic recombination has also been identified in several infected hosts. Additionally, we observed the genetic diversity between env sequences from different tissues of infected individuals. In general, low intrahost genetic diversity was found but the first case of FIV compartmentalization in a domestic cat was also documented. Finally, the evolutionary rate of FIV from serial samples from three companion cats was estimated using a Bayesian coalescent method. The rate of 3-6 % per decade is about twice as fast as that documented for FIV in the cougar, *Puma concolor*. This research represents the largest phylogenetic-based studies on naturally-occurring FIV infection in domestic cat populations.

**#21 Drug-induced release of drug resistant malaria parasites: competition at varying ratios of drug resistant and drug sensitive clones**

Silvie Huijben, Penn State University

Co-authors: Brian Chan and Andrew Read

Resistance against most antimalarial drugs is widespread nowadays. Large amounts of funding have recently become available for the continuous development of new drugs to replace the failing ones. However, extending the useful lifespan of the drug is cheaper and would furthermore reduce morbidity and mortality as a direct result of failing drugs. To achieve this, it is important to gain a better understanding of the evolution of drug resistant parasites. Using a rodent malaria model, we studied the competitive interactions between drug-resistant and drug-sensitive parasites at a range of frequencies: from rare mutant to equal abundance. We found that the resistant parasites were progressively more suppressed when in increasing minority. Drug treatment caused a competitive release of resistant parasites, which was greatest for the most suppressed parasites. This experiment illustrates the importance of resistant parasites at low frequencies in an infection, since these parasites can explain much of the dynamics observed in patients following treatment. This study furthermore highlights the role of drug treatment in the up selection of resistant parasites.

**#22 Modeling the dynamics of Epstein-Barr virus infection**

Giao Huynh and Fred Adler, University of Utah

Epstein-Barr virus (EBV) is one of the most widespread human viruses, infecting over 90% of humans worldwide and persisting for the lifetime of the person. Most people infected with EBV are asymptomatic, but the virus has been associated with many diseases and cancers including infectious mononucleosis (IM), Hodgkin's lymphoma (HL), and nasopharyngeal carcinoma (NPC). Within an infected host, EBV targets two major cell types, B cells and epithelial cells, and virus emerging from one cell type preferentially infect the other. We propose a mathematical model to describe the regulation of EBV infection within a host. We show that the ability of EBV to switch between cell types helps it to maintain the infection and high level of virus shedding even in the face of the host immune response. The model implies that subtle differences between hosts in their antibody response may play an important role in the development of infectious mononucleosis.

**#23 Field data elucidating the dynamics of coronavirus vaccination in a host population.**

Mark W. Jackwood, University of Georgia.

In this study we examined the dynamics of a live avian coronavirus vaccine in commercial chicken flocks. We will report on the coverage of the vaccine in the population, the relative amount of vaccine virus in individuals, and the clearance or persistence of the vaccine in the flock. In addition, we examined the role immunity in the population plays regarding persistence of some avian coronavirus types and the potential for viral molecular evolution leading to new strains of the virus.

**#24 Wildlife-livestock conflict: the risk of pathogen transmission from bison to cattle outside Yellowstone National Park**

A. Marm Kilpatrick, UC Santa Cruz

Co-authors: Colin M. Gillin, Peter Daszak

Interactions between wildlife and domestic livestock have created conflict for centuries because of pathogen transmission, competition for space and food, and predation. However, the transmission of pathogens from wildlife to domestic animals has recently gained prominence, including H5N1 avian influenza from wild ducks to poultry, bovine tuberculosis from badgers to cattle, and brucellosis from elk and bison to cattle. The risk of transmission of *Brucella abortus* (the causative agent of brucellosis) from bison (*Bison bison*) to cattle around Yellowstone National Park (YNP) is a hotly debated topic and an important conservation issue. Here we use a model to integrate epidemiological and ecological data to assess the spatio-temporal relative risk of transmission of *Brucella* from bison to cattle outside YNP under different scenarios. Our risk assessment shows that relative risk is spatially and temporally heterogeneous with local hotspots, shows a highly skewed distribution with predominantly low risk, and is strongly dependent on climate and the abundance of bison. We outline two strategies for managing this risk, and highlight the consequences of the current adaptive management plan. Our results provide a detailed quantitative assessment of risk that offers several advantages over projections of numbers of bison leaving YNP. They suggest that risk could be effectively managed with lower costs, but that land use issues and the larger question of bison population management and movement outside the park might hinder the prospect of solutions that will please all stakeholders. More broadly, our work provides a model framework for quantifying the risk of wildlife-livestock pathogen transmission to guide management actions.

## **#25 MHC Heterozygosity: Can one survive without the tribe?**

Kelly E. Lane, University of Notre Dame

Co-authors: Concerta Holley, Megan Ericson, Agustin Fuentes, and Hope Hollocher

The role of the major histocompatibility complex (MHC) in response to parasitic infection is well documented, with pathogen pressure maintaining population level allelic diversity within this region of the genome. However, the level of heterozygosity within an individual has not fully been examined and in response to individual abundance and diversity of parasites. Here, we compare the individual genetic heterozygosity of 24 wild, long-tailed macaques (*Macaca fascicularis*) from 15 populations across the island of Bali, Indonesia, to the parasite species richness, abundance, and diversity of the gastrointestinal parasites harbored by these macaques. More specifically, we compare the heterozygosity of 12 unrelated heavily infected macaques to 12 individuals with no infections found. As populations, these two groups had very little between group genetic differences, but as individuals, heterozygosity varied significantly. We found that those individuals with the greatest levels of heterozygosity had significantly greater abundance, richness, and diversity of parasites. Population level heterozygosity may serve to reduce overall parasite exposure to the individual within that population. However, once a parasite has successfully been introduced into a host population, genetic variability, even in the MHC region of the genome, may not function to reduce the individual's parasitic burden.

## **#26 Models with natural immune-boosting help explain pertussis dynamics and changes in age-specific incidence during the vaccination era**

Jennie Lavine, Penn State University

Using an SIR model involving loss of immunity and immune-boosting upon reexposure, we review and attempt to explain three changes in patterns of pertussis infections since vaccination began. (1) The change in age distribution of infection, (2) the change from apparently stochastically driven 2-5 year cycles with peak cases in the summer, to deterministically driven 3-4 year cycles with both summer and winter peaks in the vaccine era, and (3) the overall increase in incidence despite consistently high vaccine coverage.

We hypothesize that these patterns can be explained by a combination of significant levels of natural immune boosting in the pre-vaccine era, age-specific contact patterns, and a high proportion of subclinical secondary cases. To address these issues we analyze a set of nested models that incorporate all of the proposed mechanisms and we compare them to a detailed 19-year age-structured data set from Massachusetts. Age distributions are calculated analytically from probabilistic models and the dynamics and ages are generated using a suite of age-structured ODEs.

## **#27 Emphasizing parasitic manipulation in vector-borne diseases**

Thierry Lefevre, Emory University

Infectious vector-borne diseases present one of the most pressing issues facing public health systems. In response, there has been progress in understanding the transmission of vector-borne diseases, and the prospects for the success of their control depend, in part, on the basic reproductive number,  $R_0$ . Generally, methods of estimating this fundamental epidemiological parameter is based on uninfected hosts characteristics, and assume that uninfected and infected insect vectors and vertebrate hosts are similar. However, evidence suggests that this assumption may not be valid and the influence of parasitism on host phenotype is now well documented. Many vector-borne parasites have indeed been shown to alter phenotypic traits of their insect vectors and vertebrate hosts in a way that increases contact between them and hence increases the probability of parasite transmission. These changes, include, the feeding behavior, survival and immune system of the vector, as well as attraction, defensive behavior, blood characteristics and immune system of the vertebrate host. Based on  $R_0$ , additional changes, such as, vertebrate host choice by infected vectors or parasite development duration in the vector are expected. Introducing the parasitic manipulation concept into vector-borne diseases highlights fruitful avenues not only for fundamental research, but also for developing strategies for disease control.

## **#28 Emerging and Evolving: Infectious Hematopoietic Necrosis Virus in Western Washington Salmon**

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There has been a recent emergence of a deadly fish virus in the rivers of the Olympic Peninsula in Washington State. The new virus, referred to as the M-D strain of infectious hematopoietic necrosis virus (IHNV), is significantly because it is highly lethal to steelhead trout. The M-D strain of IHNV is believed to have originated in the upper Columbia River watershed in the late 1970s. Since then the M-D strain has continued to evolve and spread down the Columbia River watershed. The emergence of M-D IHNV into the geographically separate watersheds in the Olympic Peninsula constitutes a dangerous risk to genetically distinct stocks of steelhead trout. In order to protect steelhead in the region, the origin and risks of M-D IHNV must be assessed so that the fisheries co-managers, including tribal, state, and federal agencies, can establish better policy for viral control. Therefore, to understand how the virus is moving between watersheds and what threat epidemics of M-D IHNV pose to wild animals in affected watersheds, epidemiological and biological studies will be conducted using data from all co-managed agencies along with data on relevant ecological, physical and anthropogenic factors.

## **#29 Influence of two vector species on arbovirus transmission dynamics**

Cynthia Lord, University of Florida

Many mosquito-borne arboviruses have more than one vector, which may overlap in space and time and interact differently with vertebrate hosts. The presence of multiple vectors at one location over time will influence the epidemiology of the system, and affect the design of intervention strategies to protect particular hosts. A simulation model developed for West Nile and St. Louis encephalitis viruses and *Culex nigripalpus* was expanded to consider two vector species. The model was used to examine the consequences of different combinations of vector abundance patterns on the transmission dynamics of the virus. The abundance pattern based on *Cx. nigripalpus* dominated the system and was a key factor in generating epidemics in the wild bird population. The presence of two vectors often resulted in multiple peaks of transmission. A species which is active in the winter could enable virus persistence until another vector became active. When the virus was introduced into the system affected the number of epidemic peaks and when the first peak occurred. The seasonal abundance pattern of a vector species will affect its role in transmission cycles. Future work will vary aspects of vector competence in conjunction with seasonal abundance patterns.

### **#30 Decelerating spread of West Nile virus due to percolation in a heterogeneous, urban landscape**

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Emergence of vector-borne diseases in specifically urban environments is a leading public health problem throughout the world. Standard models predict that pathogens spread as waves with constant or increasing speed, even in urban environments. In contrast, we provide evidence that West Nile virus showed significant decelerations in 6 out of the 8 years studied at its North American epicenter in New York City, using unprecedented data on WNV-positive mosquitoes and birds collected by the The New York City Department of Health and Mental Hygiene (NYC-DoHMH). We reproduce a similar decreasing speed with a spatially explicit theoretical model of West Nile for high levels of habitat heterogeneity, in the vicinity of a critical point. Geostatistical analysis reveal New York to be on the edge of such criticality, resulting in the decelerating waves detected. Local dispersal dominates the spread of WNV in NYC, and we are able to identify the presumptive origin of the epizootics in 6 out of the 9 years studied. Comparison of control strategies suggest that targeting susceptible sites in the immediate vicinity of infected locations can efficiently prevent pathogen spread to remote susceptible areas. To our knowledge, our study provides the first evidence of decelerating waves of infection due to environmental heterogeneity.

### **#31 The ecological niche and geographic distribution of plague in North America: Shaped by mammal reservoirs or other factors?**

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The enzootic cycle of plague is one of near maintenance, where infection does not lead to widespread mortality, while epizootic cycles lead to local extirpations of hosts. We hypothesize that the ecological niche of *Yersinia pestis* is independent of the ecological niches of host reservoirs, and that enzootic cycles are maintained by mammal species where their ranges overlap with the range of plague. To test this hypothesis, we use

Ecological Niche Models (ENMs) and randomization tests to assess niche identity (NI) and niche similarity (NS). Initial findings show that NI tests reject identity ( $P < 0.001$ ) between plague-infected and overall range for *Taxidea taxus*, *Spermophilus* spp., and *Cynomys* spp. NS tests were less conclusive: plague occurrences of *T. taxus* and *Cynomys* were not statistically similar to museum data ( $P < 0.05$ ), but *Spermophilus* comparisons failed to reject the null hypothesis. Only one test comparing plague-infected ecology with overall occurrences suggested non-similarity, whereas the remaining comparisons failed to reject the null hypothesis of non-similarity. We tentatively conclude that plague niches are independent of the ecology of reservoir species, and that the distribution of plague in North America is not shaped by mammalian host ecology.

### **#32 Modeling landscape-scale pathogen spillover between domesticated and wild hosts: Asian soybean rust and kudzu**

Charles Mitchell, University of North Carolina

Co-authors: Anna Fabiszewski and James Umbanhowar

Many emerging pathogens infect both domesticated and wild hosts. This raises the question: does managing negative impacts of disease on a focal host population (whether domesticated, endangered, or pest) require management of only the domesticated host, only the wild host, or both? To answer this, we developed a spatially implicit model of a pathogen transmitted by airborne spores between two host species restricted to two different landscape patch types. We parameterized the model for Asian soybean rust, a disease that emerged in the U.S. in 2004, caused by a fungus that also infects the invasive species kudzu. Our model predicts that epidemics are driven by the host species that is more abundant in the landscape. In managed landscapes, this will generally be the domesticated host. However, many pathogens survive overwinter on wild hosts, which provide the source of initial inoculum at the start of the growing season. Our model predicts that very low local densities of infected wild hosts are sufficient to initiate epidemics in the domesticated host. Therefore, managing epidemics by reducing wild host local density may not be feasible. In contrast, managing to reduce pathogen infection of a domesticated host can reduce disease impacts on wild host populations.

### **#33 The Potential Effects of Climate Change on Sleeping Sickness Epidemics**

Sean Moore, Oregon State University

Sourya Shrestha, University of Michigan

Kyle Tomlinson, University of Wageningen, NL

Holly Vuong, Rutgers University

John Hargrove, South African Centre for Epidemiological Modelling and Analysis

An estimated 70,000 cases of Human African Trypanosomiasis (HAT, commonly known as sleeping sickness) occur each year, and 60 million people are currently estimated to be at risk of infection in sub-Saharan Africa. Caused by the parasitic protozoan *Trypanosoma brucei*, the disease is transmitted between humans and animals by tsetse flies. Because the distribution of tsetse flies in Africa is strongly correlated with temperature and other climatic variables, trypanosomiasis was recently identified as one of the twelve wildlife or zoonotic diseases most likely to spread due to predicted climate changes during the 21st century. To examine the potential impacts of projected warming on trypanosomiasis epidemiology we constructed a model of disease transmission dynamics that incorporates the effect of temperature on several epidemiological parameters including tsetse feeding, mortality, and the parasite's incubation period. The model predicts the temperature range over which HAT epidemics are capable of occurring, and successfully identifies the range of climatic

conditions in areas where HAT has historically been, or currently is, present. The model does a better job of predicting disease absence than presence due to the existence of other factors such as human population density or disease control efforts that can limit disease transmission even when suitable climatic conditions exist. While certain regions where the disease is currently endemic will likely become too warm for the continued persistence of tsetse flies, and therefore trypanosomiasis, we predict that several regions in Southern Africa where tsetse flies are currently absent will likely become suitable climatically for HAT epidemics by the end of this century. In addition, in a significant portion of Central and West Africa where trypanosomiasis cases only occur sporadically, warmer temperatures may lead to more frequent disease outbreaks.

### **#34 Transmission Dynamics and Underreporting of Kala-azar in the Indian State of Bihar**

Anuj Mubayi, The University of Texas at Arlington

Kala-azar (or Indian Visceral Leishmaniasis) is a vector borne infectious disease affecting primarily poor communities in tropical and subtropical areas of the world. Bihar, a state in India, has one of the highest prevalence and mortality levels of Kala-azar but the magnitude of the problem is difficult to assess because most of the cases are handled by private health providers who are not required to report them. We study the impact of underreporting using district level reported incidence data from the state of Bihar. We derive expressions for, and compute estimates of Kala-azar's reproduction numbers as well as levels of underreporting for 21 districts of Bihar. The average reproduction number estimates for the state of Bihar range from 1.1 (2003) to 4.3 (2005) with some districts' estimates supporting values less than one in the two years. It is estimated that the proportion of underreported cases declined from about 88% in 2003 to about 73% in 2005. However, our estimates show that at least 5 districts had still over 90% levels of underreporting in both years. Estimated underreporting is adjusted to reported incidence data and high-risk districts are identified. Four out of eight (in 2003) and three out of nine (in 2005) districts are miss-identified as high-risk by reported data. Total of seven (in 2003) and five (in 2005) districts are not even there in the list of high-risk districts according to reported incidence suggesting significantly different targeting of resources.

### **#35 Arbovirus Divergence in Response to a Novel Vertebrate Host**

Valerie A. O'Brien and Charles R. Brown, University of Tulsa

Many arboviruses may persist for extended periods in a single host-vector enzootic system. However, arboviruses can retain characteristics required to infect additional species of hosts or vectors, and when novel hosts are introduced could undergo rapid evolution and potential emergence. Buggy Creek virus (BCRV; *Togaviridae*, *Alphavirus*) is an ecologically unusual arbovirus vectored by the swallow bug (*Oeciacus vicarius*), a hematophagous ectoparasite historically of the cliff swallow (*Petrochelidon pyrrhonota*) and more recently of the introduced house sparrow (*Passer domesticus*). BCRV exists in two genetic lineages that occur sympatrically. One lineage is more associated with house sparrows and apparently better adapted to birds as amplifying hosts, while the other may be more associated with bugs. As part of a study of BCRV population dynamics, we sampled adult and nestling cliff swallows and house sparrows living in cliff swallow colonies in Nebraska for BCRV. House sparrows were 8.8 times more likely than cliff swallows to be BCRV-positive by RT-PCR and BCRV-infected sparrow nestlings frequently died. The variation in prevalence and pathology of BCRV in introduced and natural vertebrate hosts may be driving the evolution of separate lineages, and the system offers an opportunity to study virus divergence resulting from changing ecological conditions.

### **#36 Canine influenza and canine parvovirus as models of viral host switching**

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We are studying two models where an animal virus has shifted its host range. One is a parvovirus (feline panleukopenia virus (FPV)) which mutated to form canine parvovirus (CPV), and the other is the H3N8 canine influenza virus (CIV) which arose as a variant of the equine influenza virus. Both of these host-shifting viruses arose from viruses of other hosts which crossed the host range barrier and established epidemic infections in dogs. We are examining the evolutionary events that were associated with the emergence of each of these viruses, and are correlating the dynamic changes found in the sequences (in both populations and individual infected animals) with studies of the virus functions. For the CPV event we are examining the specific roles of the receptor binding and changes in capsid-receptor affinity, and are correlating those to changes in the antigenic structure of the capsids. For the CIV event we are examining the variation in the virus and correlating changes with the known antigenic sites of the viruses, and are also beginning to examine the interactions of the viruses with the specific sialic acids of dog and horse tissues.

### **#37 Infection dynamics of *Bordetella bronchiseptica* in a free-living population**

Ash Pathak, Penn State University

*Bordetella bronchiseptica* is a gram-negative bacterium infecting a wide range of mammals. It transmits rapidly and effectively in laboratory, domestic and livestock conditions, but relatively little is known regarding its transmission dynamics in free-living populations. We investigated this in a natural population of rabbits using an ELISA developed in-house to detect antibodies specific to the bacterium. Annual seroprevalence of *B. bronchiseptica* is high and appears to increase with increasing host age, but declining in older animals. In addition, seroprevalence increases with seasonal progression with highest seroprevalence in the winter months and lowest in spring, coinciding with the start of the breeding season. It exhibits an annual cyclical pattern of infection suggesting frequent reinfection and lack of protective immunity. Breeding females may serve to transmit the infection to their naïve litters as 48% of un-weaned kittens had seroconverted by 2 months of age.

### **#38 Ecology of infectious pathogens in wild and domestic African carnivores**

Katherine Prager, UC Davis

Co-authors: Linda Munson, Patricia Conrad, Edward Dubovi, Jonna Mazet, Andrea Packham, Charles Rupprecht, Rosie Woodroffe

A key issue in understanding the dynamics of multi-host pathogens in an ecosystem is the identification of the pathogen reservoir. In Africa, domestic dogs (*Canis familiaris*) have traditionally been thought to be the reservoir of canid pathogens; however, other wild carnivores, such as black-backed jackals (*Canis mesomelas*) and spotted hyenas (*Crocuta crocuta*) may play an important role in transmission dynamics and possibly as reservoirs. From 2006 to 2009 serum samples were collected from the Ewaso ecosystem of Kenya from each of three different carnivore species: black-backed jackals (67), spotted hyenas (68) and domestic dogs (65). These samples were collected from areas with high (5 dogs/km<sup>2</sup>) and from areas with low domestic dog densities (0.07 dogs/km<sup>2</sup>). Samples have been / will be tested for antibodies against the following pathogens: canine distemper virus, canine parvovirus, canine coronavirus, rabies virus and *Neospora caninum*. Once serologic analyses

are completed, mixed model logistic regression will be used to determine whether there exists a difference in seroprevalence of antibodies against the different pathogens in jackals and hyenas living in areas with low versus high dog densities. Serologic analysis of dog samples will confirm that these animals have been exposed to the pathogens of interest.

**#39 Conceptualizing and quantifying parasite spread through connected landscapes: Modeling diffusion of human schistosomes in China using molecular genetics and environmental models**

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Schistosomiasis has re-emerged in mountainous agricultural environments that had previously attained transmission control in central Sichuan Province, PRC. While it is clear that transmission in this region exhibits a strong response to environmental change, the underlying mechanisms shaping this relationship are unclear. In particular, little is known about how environmental change influences parasite spread. The relative importance of the transport of intermediate hosts or *S. japonicum* larvae through waterways in relation to other potential modes of schistosome spread, such as human and domestic animal movement, is unknown. Here, we illustrate how we combine multilocus genotyping of parasites and snails with environmental modeling to identify the diffusive pathways along which parasites are transported into new and existing locales. Fully Bayesian assignment techniques are outlined, and the delineation of hydrological and overland diffusion corridors using a functional connectivity algorithm is illustrated. The ultimate aim is to determine how anthropogenic change can modify diffusion parameters, thereby influencing transmission.

**#40 Anything to declare? The dynamics of pathogen-mediated invasion.**

Olivier Restif, University of Cambridge

Parasites and pathogens play a major role in the structure and stability of ecological communities. In particular, generalist pathogens have the potential to interfere with the competitive relations between their hosts. Thus, an invasive species may benefit from being introduced with a pathogen into a habitat occupied by a related host species naïve to that pathogen. I will address the following question: What characteristics of the pathogen, in relation to both host species, are most likely to make the invasion successful? Previous approaches, based on steady state analysis of models describing homogeneous host populations, have ignored the possibility of stochastic extinction of either host or pathogen. I consider the introduction of an invasive host with a generalist pathogen into a spatially-structured habitat occupied by a pathogen-free resident host. The success of invasion is assessed by recording the probability of extinction (and time to extinction) of each host species in different scenarios. As expected, more virulent pathogens result in more disruptive dynamics. However, whether this is beneficial to the invader depends on other factors such as spatial structure. This flexible framework can be applied to a number of ecological systems.

#### **#41 Individual and combined effects of multiple pathogens (*Achlya flagellata*, *Ribeiroia*, and *Batrachochytrium dendrobatidis*) on Pacific treefrogs (*Pseudacris regilla*)**

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In nature, hosts encounter multiple pathogens whose combined effects on individual hosts may be synergistic, additive, or antagonistic. Interactive effects of pathogens on individual hosts may ultimately influence host populations. Although several pathogens are implicated in amphibian population declines, interactions between pathogens have received very little attention as possible contributing factors. Using Pacific treefrog (*Pseudacris regilla*) larvae as hosts, we used a multifactorial experiment to test for interactions among three amphibian pathogens: the trematode *Ribeiroia*, the fungus *Batrachochytrium dendrobatidis* (Bd), and the oomycete *Achlya flagellata*. Exposure to *Ribeiroia* cercariae caused elevated mortality, increased frequency of limb deformities, and delayed development in larval treefrogs. In contrast, exposure to Bd accelerated treefrog development, though no Bd infections were found. Thus, exposure to Bd caused sublethal but biologically significant effects among apparently uninfected individuals. Larvae may have responded to the presence of Bd by increasing their rate of development. We found no effects of *Achlya* or strong evidence of interactions between pathogens, but the occurrence of such interactions may be context-dependent. Due to the high degree of context dependency in amphibian diseases, tests for possible between-pathogen interactions in a variety of amphibian host-pathogen systems and ecological scenarios are warranted.

#### **#42 Levels of inbreeding in protozoan parasites of the *Leishmania Viannia* subgenus**

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Leishmaniases, diseases affecting humans and animals caused by protozoan parasites belonging to the *Leishmania* genus, still pose considerable public health problems. It is suggested that *Leishmania* species present a basically clonal population structure associated to rare sexual recombination events. However, the markers used were little adapted and clonality was inferred from the analysis of linkage disequilibria that are far from ideal in that respect. The objective was to study the population structure and reproductive mode of two

*Leishmania* species. In total, 125 strains of *Leishmania braziliensis* and 170 of *Leishmania guyanensis* were genotyped on 12 microsatellite loci. The results obtained appear in contradiction with a simple clonal propagation: (i) strong homozygosities were found at each locus, associated to strong linkage disequilibria, advocated for a sexual and endogamous reproductive strategy, (ii) a significant part of the high heterozygote deficits observed is likely the consequence of a Wahlund effect, i.e. the coexistence of strongly differentiated genetic entities. The hypothesis is that there are different proportions of clonality, selfing and sexual recombination events in relation to species. It will be interesting to work on finer scale in order to circumscribe this Wahlund effect observed. The debate on clonality/sexuality of these parasites is far from being solved and deserves further investigations.

#### **#43 Impacts of the interaction between viral pathogens and mutualistic fungi on plant performance under elevated CO<sub>2</sub>**

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To fully understand how increases in atmospheric CO<sub>2</sub> concentrations will impact future ecosystem productivity and carbon storage requires that we understand how the strength and nature of multiple, co-occurring species interactions are altered. Specifically, since both microbial mutualists and plant pathogens can modulate plant-carbon cycles, the growth, fecundity and population dynamics of these two groups of widespread microbes may modify plant performance in response to elevated CO<sub>2</sub>. Using arbuscular mycorrhizal fungi (AMF) and barley yellow dwarf virus PAV (BYDV) we investigated microbial impacts on plant performance due under changes in atmospheric CO<sub>2</sub> and phosphorous addition. We grew the invasive species *Avena fatua* (wild oats) and *Bromus hordeaceus* (soft brome) in individual pots in replicated continuous flow chambers. We factorially manipulated atmospheric CO<sub>2</sub> (585 PPM), soil phosphorous, AMF association, and BYDV infection. To assess plant performance, we measured survivorship, above and belowground biomass, tiller number, BYDV infection, aphid reproduction, AMF colonization, seed production, and photosynthetic capacity. BYDV inoculation reduced *B. hordeaceus* photosynthetic capacity by 30% compared to mock inoculated plants. Additionally, rates of transpiration for *B. hordeaceus* were 34% lower when plants were both infected with BYDV and phosphorous addition while *A. fatua* rates of transpiration were lower when plants were infected with BYDV. Thus, effects of BYDV infection depended on phosphorus supply, but did not interact together, or with atmospheric CO<sub>2</sub> concentration.

#### **#44 Behavioral Determinants of Pathogen Transmission in African Apes**

Julie Rushmore<sup>\*1,2</sup>, Sonia Altizer<sup>1</sup>

<sup>1</sup>UGA ODUM SCHOOL OF ECOLOGY, <sup>2</sup>UGA COLLEGE OF VETERINARY MEDICINE

Infectious diseases are an important threat to the health and persistence of Africa's endangered apes. Currently, little is known about how social contact affects disease transmission among primates. Further, no studies have directly examined how sexually transmitted diseases (STDs), which potentially sterilize their hosts, impact wild ape population dynamics. To better understand the role of pathogens in the conservation of African great apes, I will examine 1) contact-based networks for pathogen transmission, 2) the prevalence of sexually transmitted diseases (STDs), and 3) behavioral defenses against STDs in wild Ugandan apes. I will conduct behavioral observations at Kibale Forest National

Park, in Uganda, which has a well-studied and well habituated population of over forty chimpanzees. Further, I will screen previously collected ape urine and blood samples for candidate STDs using genus-level PCR at the UGA College of Veterinary Medicine. An integrative understanding of how behavioral ecology and disease epidemiology affect pathogen transmission in apes will provide crucial information for developing effective management strategies to aid in protecting endangered apes in the event of a disease epidemic.

#### **#45 Effect of the TYLCC virus gene $\beta$ C1 on the facilitation of virus transmission by Whiteflies vectors.**

L. Salvaudon, Penn State University

Co-authors: C. M. De Moraes, J. Yang, N. Chua & M. C. Mescher

Many of the most ecologically and economically important diseases of plants are vectored by insects. A growing number of studies have demonstrated that some pathogens can effectively manipulate their host's chemistry to favor the attraction, establishment and/or dissemination of their insect vectors on infected plants. Until now, though, the mechanisms by which such manipulation occur stay unclear. The *Tomato Yellow Leaf Curl China virus* (TYLCCV) is transmitted by whiteflies and requires a prolonged feeding of those herbivores on host plants to be picked up and disseminated. This virus associates with a satellite carrying only one gene,  $\beta$ C1, which has been identified as responsible for the main morphological symptoms on infected plants (curling of the leaves). However, evidence also suggest that another function of this gene, or possibly its main one, might be to interact with the regulation of plant defenses against whiteflies herbivores through the Jasmonic acid pathway. We thus investigated the effect of this gene on different components of plant chemistry and attraction to whiteflies using transgenic lines of *Nicotiana benthamiana* and *Arabidopsis thaliana* uninfected by TYLCCV but expressing  $\beta$ C1.

#### **#46 Predator Presence Supresses Immune Function in Amphibian Larvae**

Sarah Seiter, University of North Carolina

Life-history theory predicts that energetically costly activities, such as growth, reproduction, or predator defenses should trade off against immunity. However, the effects of predator induced phenotypes immune system are relatively unexplored. I experimentally tested the effect of natural predators on the immune system of wood frog tadpoles (*Rana sylvatica*) by exposing half of the tadpoles to caged dragonfly larvae predators, and half to empty cages. I then administered a standard immunoassay, the phytohemagglutinin (PHA) assay to a randomly selected group of animals from each treatment. These results reveal that exposure to predators reduces the response to PHA in larval *R. sylvatica*. Furthermore, predator-exposed larvae lack the typical decline in immunocompetence during metamorphosis that is found in normal amphibian larvae and have a weaker response to PHA throughout their development. Thus, predators have an effect on both immunocompetence and developmental patterns of immunity. Generally, predator exposure may facilitate parasitic infection in amphibians by reducing immune function, and thereby render amphibian populations vulnerable to co-exploitation by both predators and parasites.

#### **#47 Effects of viral sampling strategy on coalescent-based reconstructions of epidemics**

J. Conrad Stack, Matthew Ferrari, Bryan Grenfell, Penn State University

With the rise in the collection of sequence data, the last few decades have seen significant effort to incorporate theoretical population genetics and epidemiology. Acute RNA viruses (ARV), such as measles and influenza, have been a major target of this effort in part due to their public health importance as causes of morbidity and mortality and in part due to their very high mutation rate. Much of the progress made thus far has come from the maturity of coalescent theory, the application of which allows researchers to infer population-level processes such as demographic history, migration, and recombination from patterns in ancestral gene trees (genealogies or, more commonly, phylogenies). Here we focus on inferring demographic history from simulated sequence data. Collecting samples only when convenient - and not based on a specific sampling design - can cause biases in demographic parameter estimation. These biases are less severe under simple demographic scenarios such as constant or exponential growth. However, the strong non-linearity inherent in recurrent epidemics (such as caused by ARVs) means that the timing of sampling can have a major impact on the coalescent reconstruction. We perform a simulation study to explore this effect. To tie the analysis to realistic epidemic dynamics, we explore the dynamics of neutral viral variation in a well-validated model for prevaccination-era measles dynamics. We found that sampling at the tail end of a major epidemic consistently provided better short-term (1-2 year) reconstructions of the epidemic curve. Our results also suggest that cutting up datasets that span long time periods based on some prior knowledge of the natural system can also provide better reconstructions.

#### **#48 Disease dynamics of a multi-host pathogen in an avian community.**

Sarah L. States, Wesley M. Hochachka, André A. Dhondt, Cornell University

Because many pathogens can infect multiple host species within a community, disease dynamics in a focal host species can be affected by the composition of the host community. We examine the extent to which variation of species' abundances in an avian host community may contribute to geographically-varying prevalence of a recently emerged wildlife pathogen. *Mycoplasma gallisepticum* is a pathogen novel to songbirds that has caused substantial mortality in one songbird species in eastern North America. This host, the house finch (*Carpodacus mexicanus*), is the primary songbird host species for *M. gallisepticum*, but other bird species act as alternate hosts. Laboratory experiments have demonstrated *M. gallisepticum* transmission among some of these species; however, still unknown is the real world impact on disease dynamics of variation in abundances of these hosts. We analyzed data from winter-long citizen science bird and disease surveys in the northeastern United States. We found that disease prevalence in house finches was affected by the abundances of American goldfinches and house finches, although the combinations of abundances that maximized disease prevalence were rare. Nevertheless, our results indicate that spatial variation in bird communities has the potential to cause geographic variation in disease prevalence in house finches.

#### **#49 The effect of climate on the distribution of the dengue fever vector, *Aedes aegypti*, in Ecuador**

Anna Maria Stewart and Mercy Borbor-Cordova, SUNY-ESF

Climate change is projected to increase the prevalence of mosquito-borne diseases by expanding the geographic range of mosquitoes. Dengue fever (DF), a virus transmitted to humans by the *Aedes aegypti* mosquito, is one of the most significant and rapidly spreading vector-borne viruses, yet few studies have empirically modeled the effect of climate on the distribution of *A. aegypti* on a fine scale. The objective of this exploratory analysis was to identify the climatic gradient space occupied by *A. aegypti* in Ecuador. Using GIS, raster grids of climate were developed from meteorological data from over 100 weather stations (1982 to 2005) and the historical presence or absence of *A. aegypti* was mapped based on

quarterly entomological surveys (2000 to 2005). Using FORTRAN, I determined the optimal climatic gradient space of *A. aegypti* in Ecuador by estimating the relative and absolute abundance of the mosquito along dual temperature and precipitation gradients. Preliminary results indicated that *A. aegypti* distribution in Ecuador was limited by temperature more than by precipitation. This critical research will aid in the development of a geographical model of disease transmission that will allow public health policy makers to anticipate and mitigate future DF epidemics.

### **#50 Defining the Evolutionary Pathway of CPV Adaptation in Dogs**

Karla M. Stucker<sup>1</sup>, Tyler D. Lillie<sup>1</sup>, Edward C. Holmes<sup>2</sup>, Colin R. Parrish<sup>1</sup>

<sup>1</sup>Cornell University, <sup>2</sup>The Pennsylvania State University

In order to predict, control, and ultimately prevent, viral emergence events, the molecular mechanisms that support a successful virus host range switch must be better understood. Canine parvovirus (CPV) is one of the few well-established models for studying viral emergence, and has been followed closely since it emerged as a new pathogen of dogs in the late 1970s. Today, a newer clade of viruses that includes CPV-2b has replaced the original variant, CPV-2, in nature suggesting that these newer variants are better adapted to the canine host. To help define the evolutionary pathway CPV has taken during its adaptation in dogs, a panel of 22 intermediate viruses has been constructed. Phylogenetic analysis of the CPV capsid gene, VP2, shows that this new clade of viruses is defined by 4 nonsynonymous changes in VP2. Each of these 4 sites has been mutated together, pairwise, and individually from the CPV-2 sequence to the CPV-2b sequence; similarly, the corresponding back mutations from CPV-2b to CPV-2 have been made. The relative fitness of these intermediate viruses is being inferred from measures for viability, infectivity, antigenicity and receptor binding, and will be tested directly in pairwise competition assays.

### **#51 *Ranavirus* prevalence in amphibians populations for an area with past virus-related mortality events in Great Smoky Mountains National Park**

Megan Todd-Thompson\* and Benjamin M. Fitzpatrick\*

\**Department of Ecology and Evolutionary Biology, University of Tennessee, Knoxville, TN*

One third of amphibian species are threatened with extinction. Disease is one of the main causes of amphibian declines. Viral infections, specifically viruses in the *Ranavirus* family (Iridoviridae), have been documented in several distinct groups of amphibians and have resulted in amphibian die-offs throughout North America. Most studies of *Ranavirus* in amphibian populations have focused on extreme mortality events, when casualties sometimes exceed 90%. How, or if, the virus is maintained in a population outside of die-off events is not well understood. This study focuses on the amphibians in the Cades Cove area of Great Smoky Mountains National Park because of the area's history of *Ranavirus*. I have not detected *Ranavirus* in any of the 77 salamander tissues I have assayed. This corresponds to a prevalence of 5% or less (with 95% confidence based on the binomial distribution). This result raises the question of whether the virus is now extirpated from Cades Cove, and what if any long-term ecological impacts have resulted from the die-offs recorded nearly ten years ago.

### **#52 Host culling as an adaptive management tool for chronic wasting disease in white-tailed deer: a modeling study**

Gideon Wasserberg, Erik E. Osnas, Robert E. Rolley, and Michael D. Samuel

Department of Forest and Wildlife Ecology, 207 Russell Laboratories, Wisconsin Cooperative Wildlife Research Unit, US Geological Survey, 204 Russell Laboratories, 1630 Linden Drive, University of Wisconsin, Madison, Wisconsin 53706, USA; and Bureau of Science Services, Wisconsin Department of Natural Resources, 2801 Progress Road, Madison, Wisconsin 53716-3339, USA

We developed a multi-state population matrix model to evaluate management issues on density- (DD) and frequency-dependent (FD) transmission, time since disease introduction, and deer culling on the demographics, epizootiology, and management of CWD. Both DD and FD models fit the Wisconsin data for a harvested white-tailed deer population, but FD was slightly better. Time since disease introduction was estimated as 36 (95% CI, 24–50) and 188 (41–>200) years for DD and FD transmission, respectively. Deer harvest using intermediate to high non-selective rates can be used to reduce uncertainty between DD and FD transmission and improve our prediction of long-term epidemic patterns and host population impacts. A higher harvest rate allows earlier detection of these differences, but substantially reduces deer abundance. Results showed that CWD has spread slowly within Wisconsin deer populations, and therefore, epidemics and disease management are expected to last for decades. Non-hunted deer populations can develop and sustain a high level of infection, generating a substantial risk of disease spread. In contrast, CWD prevalence remains lower in hunted deer populations, but at a higher prevalence the disease competes with recreational hunting to reduce deer abundance.

### **#53 Pneumococcal capsular polysaccharide structure predicts serotype prevalence**

Dan Weinberger, Harvard School of Public Health

There are 91 known serotypes of *Streptococcus pneumoniae*, each of which produces a unique surface polysaccharide. The nasopharyngeal carriage prevalence of particular serotypes is relatively stable worldwide, but the host and bacterial factors that maintain these patterns are poorly understood. Given the possibility of serotype-replacement following vaccination against seven clinically-important serotypes, it is increasingly important to understand these factors. We sought to identify novel correlates of carriage prevalence. Using in vitro experiments, we found that more prevalent serotypes tended to be more heavily encapsulated and were more resistant to killing by neutrophils. Additionally, we found that specific structural characteristics of the polysaccharides themselves correlated with degree of encapsulation, and polysaccharide structure was associated with serotype carriage prevalence in both vaccinated and unvaccinated populations. The results of this study suggest a novel model for differences in serotype prevalence in which serotypes producing polysaccharides with particular structural features are more heavily encapsulated. This, in turn, allows them to avoid neutrophil-mediated killing and persist in the nasopharynx for a longer duration and reach higher prevalence.

### **#54 Co-infection of *Silene-vulgaris* by sympatric species of anther-smut**

Christopher Winstead-Derlega and Jessie Abbate, University of Virginia

The basidiomycete, *Microbotryum spp.*, is a naturally occurring fungal pathogen of flowering plants in the Caryophyllaceae family. The genus contains many host-specific species, each causing similar symptoms of the pollinator-transmitted anther-smut disease. The pathogen sterilizes infected individuals by eliminating pollen production and inhibiting ovary formation. The plant-pathogen system has been used to study host shifts, disease incidence, and host sterilization. Two anther-smut species have been found in sympatry on naturally occurring *Silene-vulgaris* host populations. This is in contrast to other host species, which are typically only colonized by a single pathogen lineage. To understand the

maintenance of these two species in presumably the same niche space, we are testing the hypothesis of whether or not infection by these two species is independent, or whether they interfere or facilitate one another. In vivo lab experiments tested the effects of successive and simultaneous co-inoculation with the two fungal species on the probability of disease development in the host. Inoculation treatments included co-inoculation sequential inoculations for both species and single-species control inoculations. This is an experimental approach to the question of how sympatry of pathogen species is maintained in host populations. Presently, plants are flowering and data is concurrently being collected and analyzed.

## FIELD TRIPS/HIKE

**May 23, 2009**

Transportation and lunch will be provided for registered conference participants.

Buses will leave the Classic Center at **9am**, and return by **4pm**. The buses will depart from the back lot of the Classic Center near the 1<sup>st</sup> level of the parking deck. We expect to hike from 11:00-2:30pm, stopping mid-way for lunch. The following items are recommended for the hike: sturdy walking/hiking shoes, clothes for warm/hot weather, insect repellent, sunscreen, hat, a full water bottle, granola or trail mix and rain jacket. Lunch will be provided.

**Destination 1: Tallulah Gorge.** Located 1.5 hrs north of Athens on US 441. One of the most spectacular canyons in the eastern U.S., Tallulah Gorge is two miles long and nearly 1,000 feet deep. Visitors can hike over 2 miles of rim trails to several overlooks. A suspension bridge sways 80 feet above the rocky bottom, providing spectacular views of the river and waterfalls. An easy to moderate hike.



**Destination 2: Panther Creek Falls Trail.** Located 1.5 hrs north of Athens on US 441. This trail follows Panther Creek through stands of white pine and hemlock along the steep, rocky bluffs of the creek. Passing a series of roaring cascades cut through solid rock, the treadway culminates in a waterfall at Panther Creek's junction with Davidson Creek. A more strenuous hike with a round-trip distance of just over 4 miles.

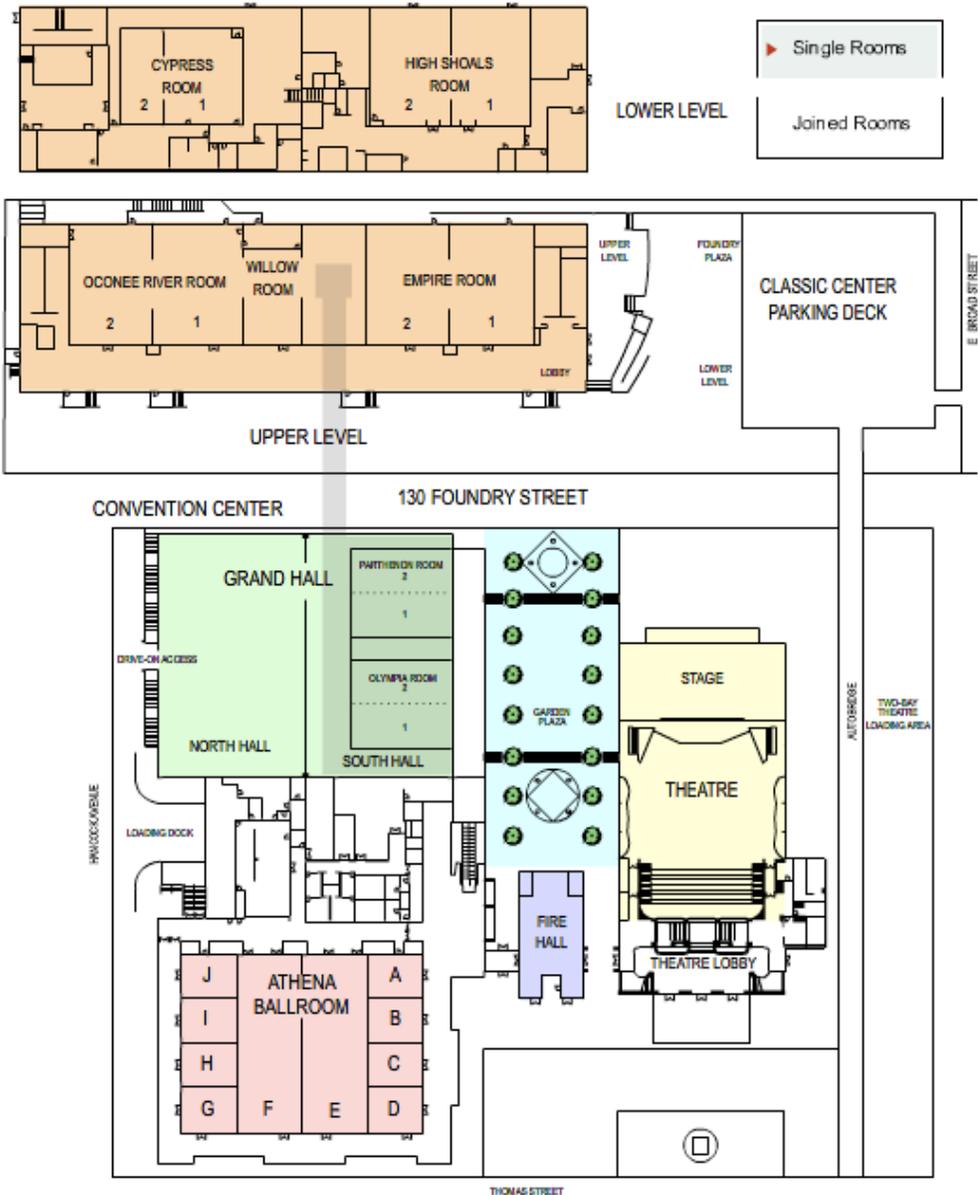


# VENUE

Most conference activities will take place at the **Classic Center** on Thomas St in downtown Athens. Parking at the main Classic Center deck is free for conference participants.

The Firehall at the main entrance on Thomas Street is our venue for the Wednesday evening welcome reception. Talks on Thursday and Friday will take place downstairs in the Grand Hall South, with the Friday Poster session in Grand Hall 1.

The Friday night dinner takes place across Foundry Street on the back side of the Classic Center in the Foundry Park Building's Empire Room. (Note that this is *not* the same location as the Foundry Park Inn and Spa).



## DINING AROUND ATHENS

Athens is known for its rich offering of restaurants and bars, most locally owned and inexpensive. The greatest concentration of restaurants and bars is found north of the University between Broad and Prince Avenues. Southeast of campus, Five Points at the intersection of Lumpkin and Milledge Avenues also offers a few restaurants.

### A few lunch recommendations:

**Taste of India** – Lunch buffet available. Good, inexpensive, gut bust'n potential [may sleep through afternoon talks]. Easy access downtown at the intersection of Lumpkin and Broad. Lunch ~\$7.

**Last Resort Grill** – Simply awesome. Great atmosphere, traditional and vegetarian items [famous for their fried green tomato sandwich] and inexpensive lunch. You can get soup, sandwich and salad for <\$8! Downtown at 174 Clayton Avenue, near the Hull St intersection.

**Athens Sushi Bar Utage** – Excellent sushi and traditional Japanese fare that is really affordable. Downtown at 440 Clayton, near N. Thomas intersection. Lunch ~ \$10.

**The Globe** – A local pub with traditional pub fare. Great fries, appetizing breads and cheeses. Downtown at intersection of Lumpkin and Clayton. Entrees \$7-\$12 [doesn't include the beer].

**Five Star Day** – Affordable and casual 'gourmet' soul food. Great sides include fried green tomatoes, mac-n-cheese, and collard greens. Veggie options are plentiful. Located in downtown at the intersection of Broad and College. Lunch for < \$7.

**Doc Chey's Noodle House** – Delicious noodle and non-noodle Asian fare. Great veggie and meat options (e.g. Mongolian beef, spicy orange chicken, garlic eggplant, an assortment of rice bowls). Downtown at 320 E. Clayton St. ~\$10

**Mellow Mushroom** – Located downtown, this pizzeria offers a wide-range of fun and delicious toppings. Calzones and light snacks (and beer) are also available. Plenty of veggie options! Downtown at 320 E. Clayton St. <\$8

**Which wich** – Delicious sandwiches with pick-your-own ingredients. Casual seating. On Broad near the corner of College.

**Barbaritos** – With four Barbaritos throughout Athens, this local burrito joint is an Athens favorite. The ingredients are fresh and tofu options are available for vegetarians. Downtown at 259 Clayton St. or in Five Points at 1739 S. Lumpkin St. Lunch < \$8.

**Weaver D's** – A local landmark, Weaver D's is famous for its southern soul food and was a popular hangout for the band R.E.M., who included the venue in some of their album art and music videos. "Automatic for the People" is a quote from Weaver himself. Lunch ~\$8. Go east on Broad several blocks from the Classic Center, just before you reach the river.

**Cali N Titos** – Great Cuban and Mexican food. Sandwiches, tacos, empanadas, etc... Seating inside and out. Located east of campus at 1427 Lumpkin Avenue. Lunch <\$8 CASH ONLY!

**Five Points Deli** – Traditional deli, great sandwiches, and convenient to campus at the intersection of Lumpkin and Milledge Avenue.

**Earth Fare** -- A grocery store with fresh and healthy options for the health-minded shoppers. This store is similar to Whole Foods or Trader Joes. There is a buffet for breakfast, lunch, and dinner. In Five Points on Lumpkin St. ~\$10

## **Dinner (many of these suggestions are also great for lunch!)**

### [Grad student budget]

**Clocked** – How good do you have to be to make a great burger or fish sandwich, but be best known for tater tots and macaroni and cheese? Retro atmosphere, limited seating. Downtown at 259 Washinton St. Dinner <\$10.

**The Grill** – An Athens institution voted best burgers in town. Traditional diner fare, but tweaked for the vegetarian as well. Vintage soda fountain and best milk shakes and malts in town. Downtown at 171 College Avenue between Broad and Clayton [across from Starbucks]. Dinner <\$10.

**Transmetropolitan** – Very good pizzas, pastas and sandwiches and very affordable. Great place to go Dutch because you pay first, sit and eat second. Bar upstairs! Downtown at 145 Clayton, Dinner ~\$10.

**Wild Wing Café and Brewery** – Too many kinds of wings to try. Other traditional pub foods and large selection of beer. Downtown at 312 Washington St.

**The Grit** – Athens best known vegetarian café. Great offering for all and affordable. North west end of downtown at 199 Prince Avenue. Entrees <\$10, Dinner ~\$15.

**Casa Mia** – Mexican tapas easily and a large bar selection. Downtown at 269 Hull St. Tapas \$5-15.

**Siri Thai** – This eatery has a large menu with many, many delicious options (including over 20 meat-free meals). Great Thai desserts. Overall, not too pricey. Located at the North west end of downtown at 367 Prince Ave. Dinner ~\$10-15.

### [State and Federal Employees and Non-tenured Faculty Budget]

**Copper Creek** – Local microbrewery and pub. Beers are solid, food is too. Traditional pub fare. Can get a bison burger! Downtown at 140 Washington St. Entrees \$8-\$12 [doesn't include the beer].

**Last Resort Grill** – Great for lunch or dinner. Casual but a tad more upscale at night. Good vegetarian offerings. Dinner entrees \$12-\$20. Downtown at 174 Clayton Avenue.

**Farm 255** – Great food with a conscience. Founded by a group of young entrepreneurs including graduate students from the Odum School of Ecology, the restaurant is connected to a local farm dedicated to sustainably grown foods. Described as Mediterranean food with a southern drawl, it is a great experience, particularly for vegetarians. Downtown at 255 Washington St. Entrees \$15-20.

### [Tenured faculty budget]

**East-West Bistro** – Upscale take on classic American and Asian fare. Nice dining atmosphere. Downtown at 351 Broad Street. Dinner entrees \$9-30.

**Harry Bissetts** – Upscale New Orleans dining. Downtown at 279 Broad Street. Dinner entrees \$15-30.

**Basil Press** – Intimate fine dining. Downtown at 104 Washington St. Dinner entrees \$15-25

**Five & Ten** – Arguably the best restaurant in Athens. It will cost you, but you'll never regret it. Located in Five Points at the corner of Lumpkin and Milledge behind Jittery Joes. Dinner entrees \$16-35. *You ain't gett'n out for less than \$50.*

**The National** – Delicious food with many Mediterranean-themed dishes. Prices are similar to Five and Ten. Located in downtown on W. Hancock Ave near intersection with Hull.

### **Coffee Shops:**

**Walkers Coffee and Pub** – Coffee shop by day, bar by night. Great local venue for coffee, pastries, sandwiches, beer, wine, or liquor. Conveniently located Downtown on 128 College Ave.

**Espresso Royale Coffee** – An Athens favorite when it comes to coffee shops. Great coffee with a good selection of pastries, wraps, and sandwiches. Downtown at 297 E. Broad St.

**Starbucks** – Every town's got one (well, at least one). Ours is located downtown at 100 College Ave.

**Jittery Joes (five points)** – Get your daily dose of locally roasted and brewed Joe. Lots of delicious options to choose from. In Five Points at 1210 S. Milledge Ave.

**Two Story Coffeehouse** – This relatively new coffeehouse has been a huge hit since its doors opened in November 2009. As the name suggests, this is an old two-story house that has been renovated into a coffee shop. It has great outdoor and indoor seating with several cozy rooms to choose from. Two Story offers a great selection of specialty coffees, teas, pastries, and gelato! Located in Five Points at 1680 Lumpkin St.

### **Bars Downtown:**

**The Globe** – This English pub-style bar offers a wide range of drinks (80 beers – including 13 on tap, 66 wines, and 38 single malts). Great indoor seating with an upstairs loft for extra space. The menu has an assortment of pub food including veggie-friendly items. Downtown at 199 N. Lumpkin St.

**Trappeze** – For folks who like beer, Trappeze is the place to go! This bar has 36 taps and 240 types of bottled beer. Even coffee drinks are available. There is a pub-style menu with veggie options and delicious (highly recommended) cookies. Located Downtown at 269 W. Washington St.

**Copper Creek** - Athens's very own microbrewery. Good pub food (with veggie options) and great home-made beer. There are domestic and import beers in addition to the in-house brews. If you are in town for the workshop – be sure to stop by for \$2 Tuesday.

**283 Bar** – A cozy bar on Broad St. with an eclectic crowd. Drink \$1 PBR in a boot-shaped mug or choose from the impressive wine selection. There are six kinds of beer on tap. Downtown at 283 E. Broad St.

**Aromas** - Doubling as an upscale bar/coffee shop, Aromas is great place to meet and chat over wine and cheese or fancy coffee drinks. This is a cozy spot with a fireplace and some plush couches. At Aromas, you will find an extensive wine selection, five beers on tap, light tapas, and locally made deserts. Located in Five Points at 1235 S. Milledge Ave.

### **Other Spots for Athens Night Life:**

**Cine** – This bar/café/cinema shows independent films while providing a wonderfully artsy atmosphere. Cocktails, coffee drinks, and snacks are all allowed in the theatre – making an overall excellent movie-going experience. Look for movie times at <http://www.athenscine.com/intro.php>. Located Downtown on 243 W. Hancock Ave.

**Manhattan** – This dimly-lit townie bar provides a low-key and charming atmosphere. Several beer and wine options, an extensive cocktail menu, and free popcorn. What more could you ask for? Downtown at 337 N. Hull St.

**40 Watt Club** – This small venue is a wonderful spot to check out the local Athens music scene (as well as national bands and singers that commonly pass through). Nice bar inside and plenty of space for dance parties. Located Downtown at 285 W. Washington St.

**Little Kings Shuffle Club** – A great place to go to for after-hours activities. Little Kings offers comfortable seating (both indoors and out), great drinks, and bar snacks. It is common for Little Kings to host bands or DJs – or even screen films. Also – there’s a cornhole setup outside on the patio. Downtown on 223 W. Hancock Ave.

**Go Bar** – A fun and eclectic place to meet up for late-night dancing and/or karaoke. Outdoor seating with an intimate indoor dance floor. The bar offers summery cocktails for beating the Athens heat. Downtown at 195 Prince Ave.

**Flicker Theatre and Bar** –This artsy bar is a townie favorite, and patrons can enjoy free popcorn with their drinks while enjoying a movie or live music performance. Downtown at 263 W. Washington St.

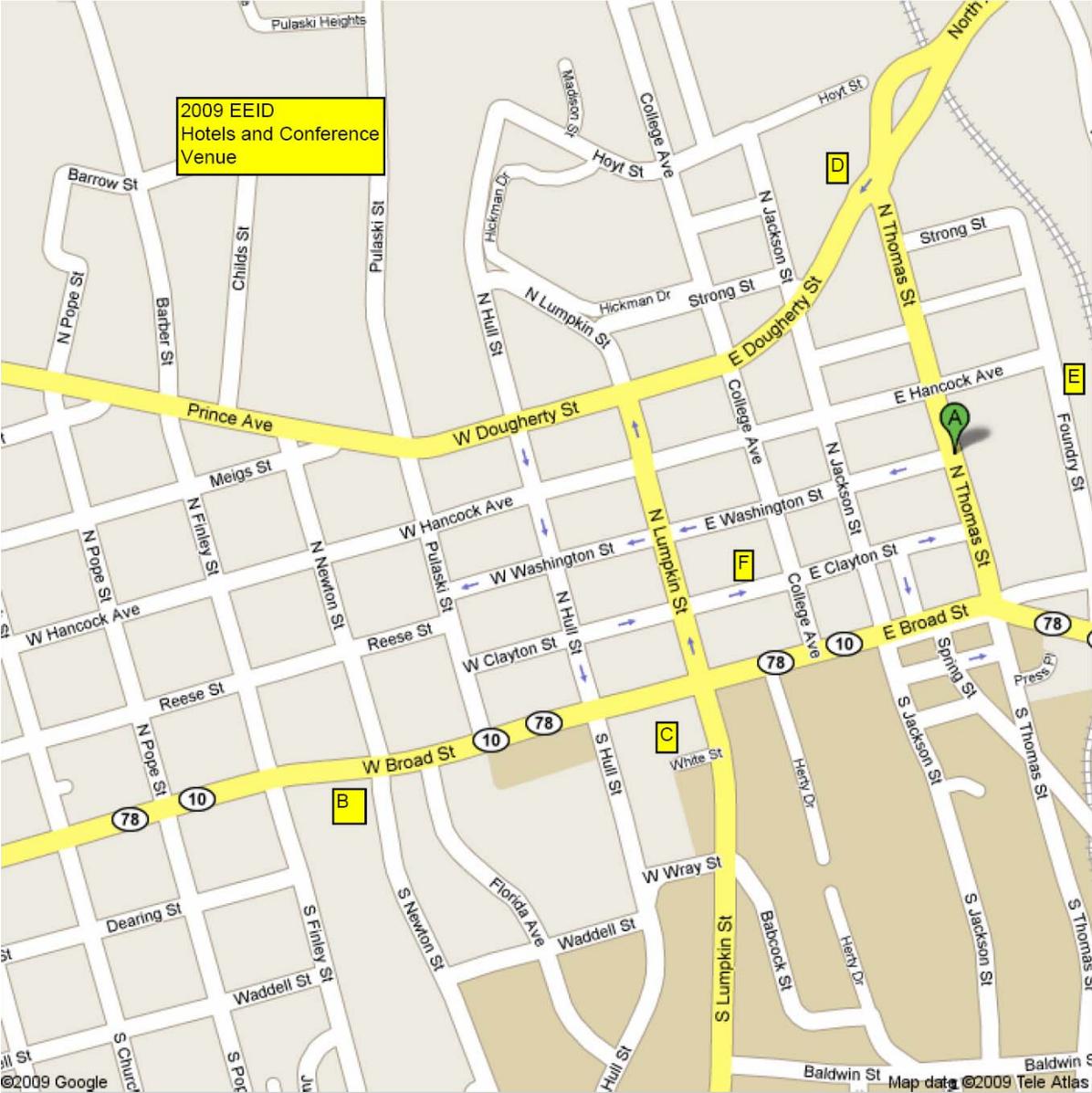
**Public Transportation (Athens Bus System):** <http://athenstransit.com/>

**Taxicab Services:**

A & A Athens Taxi (706) 353-2424  
Top Dawg Taxi (706) 552-0744 (706) 850-6225  
Your Cab Co (706) 546-5844  
United Taxi (706) 549-0808  
The Limousine Company (706) 227-1324

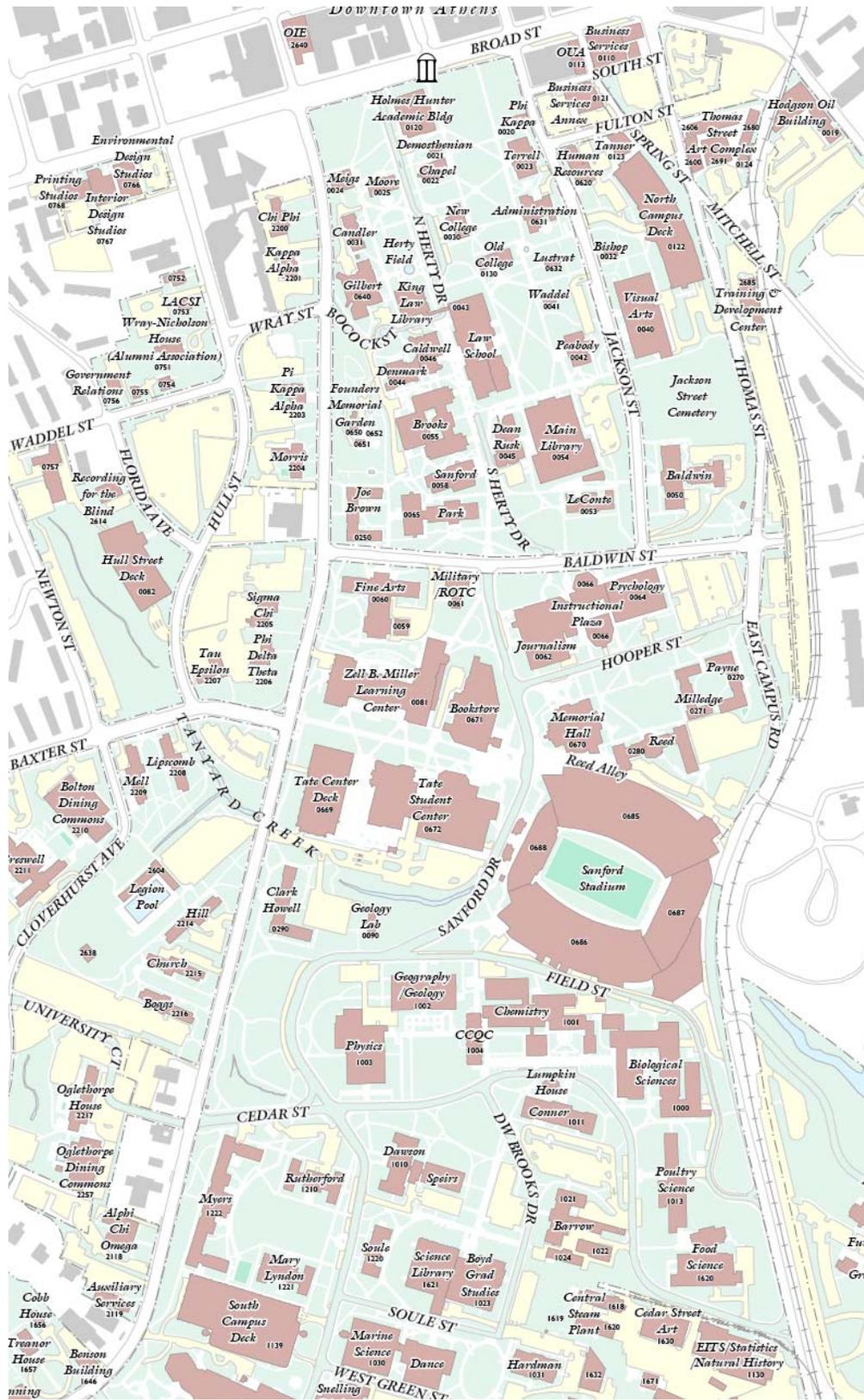
# MAPS

Map of downtown Athens, with Conference Venues and Hotels as indicated:



- A = Classic Center
- B = Holiday Inn Express
- C = Holiday Inn
- D = Foundry Park Inn & Spa
- E = Foundry Park Building
- F = Transmetropolitan Pizza

UGA Campus Map (partial; downtown Athens at north end of map):



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