#beseeid, Tweets by B Koskella, May 2013

- I'll be tweeting all day from the BES Evolutionary ecology of infectious disease symposium.
- And we're off! Starting the day with a talk by Angus Buckling from the university of Exeter, Cornwall campus.
- "The impact of coevolution on evolutionary and ecological processes depends critically on the underlying coevolutionary dynamics."
- Environmental conditions can alter the rate of coevolution... But dynamics in the lab are still mostly "arms race" dynamics"
- Genetic distance of phages from ancestor increases with host range," suggesting an infection genetic structure that predicts ARD
- "Results from soil microcosms are in line with fluctuating selection, not ARD, most likely due to increased costs of resistance."
- However, when soil populations are well-mixed, the dynamics become more like ARD again," so spatial structure matters!
- Rate of amino acid change of phages increases over time, but then plateaus during experimental coevolution," indicative of FSD
- Next up: Andrea Graham from Princeton University, talking about importance of heterogeneity in defence against parasites
- It was!! "@jaytlennon: @bkoskella Sounds like it was a great talk! @meganllarsen"
- How can we explain the vast heterogeneity in immune response among individuals in natural populations? Trade-offs, of course!"
- Soay sheep population on st kildas have been studied since 1985, including plasma and parasite load counts every August."
- Over 50% of individuals die during over-winter 'crashes,' suggesting strong resource and parasite-mediated selection."
- Expect survival benefits of resistance in tough times and fecundity costs of resistance in good times."
- Higher concentration of (both self-reactive and nematode-specific) antibodies correlated with life span and survival over winter"
- "But negative association between annual breeding and antibodies, although lambs of defended mothers who do survive are very fit."
- What about tolerance to nematodes? "Individuals do vary in tolerance to parasite burden, and it's associated with breeding success"
- Ecological feedbacks of tolerance and resistance are very different, and now she and Dylan Childs are teasing apart these dynamics.
- @LouiseJJohnson yeah! There are some really great papers on this, including by Alex Best and Mike Boots
- It's great stuff! "@XavHarrison: @bkoskella Thanks for live-tweeting the soay sheep stuff. Full of interesting science nuggets!"
- Next up, Joanne Webster from Imperial College London will be discussing schistosomes in the lab and field.
- Changing environments for schistosomes, with climate change, treatments, and movement of hosts... Do we still see (co)evolution?"
- "Within China, 30 million at risk for infection, in part due to zoonotic transmission.

Control in human pop h little effect."

- "Snails from hilly regions had higher cercarial shedding than marshes, time of shedding is different for each vertebrate host."
- Modelled roles of host species and found R0 much under 1 for humans, but much higher for cattle in marshes and rodents in hills."
- In Africa, schistosomiasis control initiative underway. No evidence of drug resistance from china, but it does evolve in the lab!"
- Before drug pressure, phylogeny is explained by geography, but after It is not!" Suggesting strong selection and dispersal?
- Clear evidence of a genetic bottleneck in schistosome populations. Could this mean treatment success? Or is it resistance spread?"
- Villages in Niger- not able to decrease prevalence despite treatment success due to reinfection and hybridisation among species!"
- "To control schistosomiasis, we need to understand the ecology and evolution of the many hosts and hybridisation of the parasites!"
- Next up, the amazing Curt Lively (my PhD advisor) from Indiana university, talking about disease and sex!
- "Darwin's conclusion that [the evolution of sex] is hidden in darkness was based on flower morphology that enforces outcrossing."
- "Is asexuality correlated with relaxation of parasite prevalence? Yes! Based on great evidence from the snail-Trematode system in nature .
- Parasite prevalence is negatively correlated with the presence of asexual snails, and parasites track common clones"
- Can parasites prevent the elimination of sexual reproduction? New work by Vergara, Lively and Jokela says yes."
- More sex and more parasites in coevolutionary hotspots - shallow water (work by King, Jokela, Delph and Lively)."
- Are sexual snails, as a group, generally less infected than asexual hosts? Yes! (New work with Daniela Vergara)"
- Are parasites the answer? I don't know, but the data we have consistent with a strong role for parasites in maintenance of sex."
- Bonus story from CL: "outcrossing selected for only by coevolving parasites in C. elegans - great work with Levi Morran."
- Are parasites a general mechanism? Problem is that you often don't see virulent parasites because they are killing their hosts!!
- Best quote of the meeting so far: C Lively in response to a question that may be 'fluffy'- "no, I'm happy with fluff."
- As a side note, this is my first time live tweeting a meeting. I feel a bit like a mole :) but loving it!
- And we're back, with a talk by Heather Ferguson from university of Glagow, who will be discussing mosquito vector behaviour.
- Vector control is the most effective mechanism of control for malaria. But susceptibility to insecticide is fragile."
- "But also, Mosquitos can evolve earlier biting, resting outdoors, or increased zoophily. Some of these would enhance control."
- Mosquitos vectors show large variation across sites in host species choice, but humans

can make up to 100% of the blood contents."

- Can the use of bed netting lead to selection on vectors to feed on non-human hosts?" Great question! I hope there's good news...
- Experiment from Ifakara health institute, where they manipulated host availability and examined mosquito feeding and fitness."
- "Vectors varied in fitness across host species (potential for selection), and did not necessarily do best on their preferred host."
- Putting humans under nets, however, massively increased fitness difference of specialisation on cow versus human hosts."
- Disclaimer: although I am using quotation marks, these are my own words paraphrasing what was said... #shootthemessenger
- Some data suggesting that the use of bed nets has lead to changes in feeding time of vectors." Genetic change? Not obvious....
- "Take home message: changes in vector behaviour are happening, but it is unclear how important these changes might be."
- Next up: Janis Antonovics (who got me interested in science to begin with), talking about disease distribution and abundance.
- "1970s Ecology textbooks had <1% pages devoted to parasites/pathogens."
- Disease dynamics are not two dimensional (number hosts, number parasites). Need an axis for genetics of resistance."
- Disease along environmental gradients - should get a disease free halo at the edge of the species range, as host density drops."
- Evidence for disease free halo from Bombus terrestris and nest parasite.
- However, this may not hold for sterilising parasites, as transmission is not density dependent."
- "Baker and Antonovics show variation in resistance is most common in diseases with longer duration and low efficacy of vaccine"
- Sterilising parasites theoretically lead to extinction oh host at edge of range, rather than halo."
- Prevalence only depends on transmission and death rate, not births nor density."
- Okay, that was in theory, now we're into the real world.... Silene smut!!
- Microbotryum 'smut' is a sexually-transmitted plant disease that sterilises the host and is transmitted by pollinators."
- How to choose a field site: decent food, decent weather, and excellent wine! Decision - Italy."
- Indeed, in this system, disease prevalence does not correlate with density!"
- When you introduce evolution of resistance across the cline (decreasing birth rate), resistance spreads to near edge of range"
- Cool!! “@RobbieAMcDonald: @bkoskella At a local scale same is true of badgers and TB”
- But across increasing death rate, the resistance allele does not spread to edge."
- Take home: need to consider transmission mode, ecological/distributional context, genetic variation to understand disease spread
- Very few studies of disease across a gradient." PhD students and budding researchers, take note!
- Mike Boots summary of meeting: "not enough data on the impact of genetic variation

(and evolution) on the ecology of disease."

- Greg Hurst's summary: "need more data on short term cycling versus long term change and advances."
- Steve Paterson's summary: "need more data on the importance of horizontal gene transfer and hybridisation for disease dynamics."
- Jo Lello's summary: "we still don't really understand the relevant scale of most host-parasite interactions."
- More reactions: "most models are bed on specific systems, and therefore we need more general and generalizable theory -MB"
- And more: "it's not just infection genetics that matter... many plastic responses and immunopathological factors are important."
- And Stuart Auld says: "what about differences in host exposure, can that influence the dynamics? FSD vs ARD.
- Olivier Restif says: "make sure your predictions and observations are based on the same timescales! Equilibrium or dynamics?"
- Janis adds: "gene frequency models are a step forward, but fall very short from capturing complex interactions btwn evol and ecol."
- That's all folks!!! Have a good weekend, and I'll tweet you starting Tuesday from the EEID meeting at penn state!

#EEID2013 Tweets by B Koskella, May 2013

- Arrived at PennState for EEID 2013! Great opening reception, with so many smiling faces. Should be a wonderful few days.
- My live tweets will be posted after the meeting each day, as penn state is charging for wifi and I am a cheap skate.
- Cool format for meeting - moderators are in charge of organising time for discussion after each batch of talks.
- Pete Hudson starts by introducing the meeting, which started 11 years ago as his 50th birthday party.
- First session is on within-host dynamics and is moderated by Charles Godfrey. Starts off with Rustom Antia from Emory.
- Quotes Bruce levin: "an hour's worth of sleep in a lecture is worth two at home."
- We have almost no vaccines against persistent infections, but many against acute infections."
- Rational approach to vaccine design is based on the key immunological factors correlated with the epidemiology of the disease."
- For T-cell vaccines, what causes protection versus pathology? Chronic infection occurs when T cells get 'exhausted'."
- "Narrow vaccination can cause pathology - Oehen et al. Science 1991. Followed by Blattman et al. 2009."
- Having few T cells leads to a chronic infection, intermediate to death, and many leads to clearance or virus escape."
- "Put his model into 'risky contact with data' and found broad vaccines have the same pattern but prevent escape."

- Next up we have Katia Koelle from Duke, discussing within-host models for dengue fever.
- "Malnutrition actually decreases the chance of developing hemorrhagic fever and other acute symptoms."
- Previous infection with a different serotype leads to a much higher chance of developing the DHF."
- Malnourished individuals don't produce cytokines as effectively, leads to a higher viral load but lower interferon response."
- Next up, Katherine Lemon from the Forsyth Institute, discussing bacteria as therapeutic agents.
- "What can we learn about our microbiome, and can we use this knowledge to treat disease?"
- "Commensals and pathobionts (bacteria that usually don't by can cause disease) interact within the host."
- Staphylococcus aureus is a pathobiont in the human nostril, and infects 30% of the us population."
- Three genera make up the majority of bacteria in the human nostril, and these are all interacting within the host."
- "Good bacteria affect bad bacteria by growth inhibition, modifying the host environment, and behaviour."
- Inhibition is dependent on use of tween 80, which releases oleic acid. Thus inhibition occurs in environment-dependent manner."
- Dysbiosis is a shift of microbial community within the host that is associated with disease."
- Will physicians and pediatricians in the future practice ecosystem management with microbiota of patients?"
- First we need to make sure we do no harm, and learn from mistakes made in other ecosystems (invasives!)"
- Next up, Hauke Koch discussing the importance of gut microbiota in driving specificity of host-parasite systems.
- "Bumble bees have an unique microbiota relative to honey bees, and certainly relative to the environment."
- Specific microbiota apparently absent in solitary bees (Martinson 2011), instead they have primarily environmental microbes."
- Crithidia bombi is a trypanosome parasite that infects gut of bumble bees, and infection specificity is affected by microbiome."
- Fecal transplants in sterile bees lead to a decrease in infection by the parasite!"
- Crossed microbiota from multiple colonies with different genotypes of the host and found variation in specificity."
- Next up, me. And since I can't tweet and talk at the same time, I'll start again after lunch.
- No more talks today, just poster teasers and then session. I may bite the bullet and buy Internet tomorrow! See you then.
- New day, new session on human disease starting with Caroline Buckee from Harvard talking about new data and old theory.
- Used cell phone tracking to monitor the movement of individuals around Nairobi. Used these data to parameterize disease model.

- Built a very cool model on movement between high and low malaria risk settlements, put the mobile phone data in...."
- And could show sources and sinks of the parasite (Wesolowski 2012 Science), with clear application to control strategies."
- Take home message- we need to be very cautious about using these models for policy, given we understand so little of the biology"
- Next up is Alison Hill from Harvard, discussing social contagion in networks.
- Human behaviour influences the spread of disease, and behaviours might spread interpersonally just like diseases."
- "Sometimes the two can overlap, e.g. when health behaviours like vaccination spread among individuals and affect disease spread."
- Looked at the dynamics of obesity and could demonstrate interpersonal effects and social contagion (Christakis and Fowler 2007)"
- She then looked into the transmissibility and found it increased over time both spontaneously and through social contagion."
- Contagion is not the main driver of obesity, but accounts for about 7% of prevalence according to their model."
- Just realised that this meeting is also being discussed with #EEID13
- Next up is Petra Klepac from Princeton, talking about the economic incentives for vaccination.
- We can model exactly the proportion of the population that needs to be vaccinated to interrupt transmission/spread based on R0."
- For a vaccination program to succeed, we need cooperation among individuals" [and reporters, I might add]
- An individual's decision to vaccinate should decrease as the level of population level immunity increases (free riders)"
- Government intervention is therefore necessary for complete vaccine coverage and elimination of an infectious disease."
- Modelled cost (of coverage and of infection) to determine optimal equilibrium coverage, independent of R0. (Klepac PNAS 2011)"
- "Local eradication is, of course, hindered by incoming infections. Modelled local and global optima of vaccination coverage..."
- All countries are acting independently to minimise their local costs, and this leads to a lower local optima than global."
- Thus the individual cheating behaviour scales up to the population level, and there is clear benefit for cooperation (sharing)."
- "The more countries you build into the model, the larger the gap between global and local optima becomes. Need treaties!"
- If coalitions are in model, where signatories jointly decide on vaccination strategy, higher coverage occurs at lower costs."
- And this relationship becomes stronger with the more countries in the coalition, and with travel restrictions for non-members."
- Next up, Daniel Streicker from U Georgia talking about rabies in vampire bats: a man made zoonosis?
- "Vampire bat transmission of rabies to humans is increasing with livestock intensification in the amazon."
- "In Peru, rabies is present in all bat colonies in all years surveyed. Exposure only

explained by culling history and bat age."

- But culling had a net positive effect on bat exposure to rabies, perhaps due to changed age structure of colonies."
- "For rabies persistence there needs to be frequent immigration and low probability of lethal infection (Blackwood 2013)"
- "Single bat colony culls are not going to eliminate rabies, we need regional control and more rational culling strategies."
- Male bat dispersal may be key in explaining geographic structure of viral spread observed in phylogeny."
- "Take home: feedbacks between agriculture and rabies, higher connectivity and unexpected effects of culling (control is hard!)"
- Discussion points from morning talks: 1) connectedness is key factor for potential success of disease control. Need more data!
- 2) integrating data from social science, public health, and evol ecol of infectious disease will be an exciting new trajectory.
- 3) we need to understand whether people are acting rationally, changing their behaviour in rational ways, and at what scales.
- 4) no one really understands what makes something contagious - whether it's a pathogen, an idea, or a behaviour.
- 5) big data can be very helpful in elucidating pattern, but doesn't help much with understanding process.
- And we're back, with Derek Cummings from Johns Hopkins, discussing spatial heterogeneity of influenza immunity.
- Fluscape project is a longitudinal cohort study from china to examine population structure and disease dynamics."
- "From urban to rural, censored households at random about social contacts and risk factors for flu. Have two years of data."
- Also examined seriological data from participants and found older individuals tested positive for more strains.
- Peak seriological response correlates with strain cycling when participant was 5-10 years old." Cool!
- Population density also affects titre per strain." Except maybe for most recent flu - increased connectivity?
- Next up, Nim Arinaminpathy from Princeton, discussing cross-protective vaccination.
- Surface antigens of flu have been used as targets for vaccines, but these are also highly variable allowing easy escape."
- "Alternative vaccine targets, including nuclear proteins, MI, and stem HA may provide cross protection and increased stability."
- Cross protective vaccination will lower prevalence and (according to model) slow evolution of the virus (lower antigenic drift)"
- "This is due both to lower viral population size (due to lowered prevalence) and decreased selective advantage of mutant."
- Next up, Michael Worobey from U Arizona talking about avian influenza (which apparently have internal genes w/ mammalian roots)
- He gets bonus points for using Salvador Dali painting to describe the molecular clock!!
- "Perroncito discovered avian influenza in the late 1800s."
- If you apply the same clock to flu strains circulating in different host species, you end up

with a very wrong phylogeny."

- P.s. that's a really really cool and important result! Previous viral phylogenies beware.
- The great epizoonotic of 1872 killed 2-5% of horses in the U.S. and was associated with avian influenza."
- Next up we have Michael Cortz from Georgia Institute if Technology.
- Modelling pathogen evolution. Changes evolutionary speed of pathogen and examines dynamics"
- Slow evolution = stable dynamics, but pathogen is stuck at a fitness minimum. Fast evolution = endemic oscillations."
- "Ecological feedbacks drive oscillations between extreme pathogen types. Evolutionary dynamics also drive these oscillations."
- Next we have Andrew Park from the University of Georgia, talking about transmission and disease hotspots in vector
- Thanks @weitzlab!: @bkoskella Michael Cortez can be found here: http://ecotheory.biology.gatech.edu/mike/ ”
- "Hemorrhagic disease caused by many strains of EHDV and BTV, mainly in white tail deer, vectored by midges.
- No obvious source sink dynamic, no clear metapopulation structure. But morbidity and mortality structure are spatially distinct."
- Enzootic stability (Coleman 2001) is where clinical disease is scarce despite high prevalence in the population."
- **“**Common when older individuals are more infective than young ones, and when there is cross-protective immunity."
- "Modelled enzootic stability and found disease reporting maximised at intermediate seroprevalence." (Park 2013)
- Really nice fit between model and data, where morality increases as a function of gap between outbreak years."
- "Might explain why some geographic regions are mortality hotspots, and other regions are morbidity hotspots...."
- "Assuming that secondary infections have lower mortality than primary infections."
- Hmmmm.... Once again twitter has scrambled the feed from the conference and dropped most of the tweets. Annoying.
- Matt Ferrari recharges the energy in the room with summary of talks, highlighting the progress we are making.
- Nim says: "we are much more intelligent now about knowing what we don't know."
- Andrew adds: "we are acquiring larger and longer term survey data to begin testing the accuracy if the models we are building."
- Mike is a bit more pessimistic, "but is hesitant to suggest more complicated models and more data collection."
- Bryan Grenfell says (in response to: will we ever be disease-free) "we're going to be in business for many many years to come."
- Things were starting to get rowdy, Pete Hudson is calming us all down with his final words.
- Pete says, "the original idea (11 years ago) to have this meeting, was a good one!"