DISSERTATION

INTERACTION AMONG SOCIETAL AND BIOLOGICAL DRIVERS OF POLICY AT THE WILDLIFE-AGRICULTURAL INTERFACE

Submitted by

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In partial fulfillment of the requirements

For the Degree of Doctor of Philosophy

Colorado State University

Fort Collins, Colorado

Summer 2017

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ABSTRACT

INTERACTION AMONG SOCIETAL AND BIOLOGICAL DRIVERS OF POLICY AT THE WILDLIFE-AGRICULTURAL INTERFACE

This dissertation research on wildlife policy and biology focuses on understanding the mechanisms that drive development of wildlife-agricultural policy and also on understanding the underlying ecological drivers of pathogen transmission and population growth for an invasive species. This research uses a combination of meta-analyses, mathematical models, and Bayesian statistics to examine the drivers of emerging wildlife policy, transient population dynamics, and ecological determinants of pathogen prevalence, using wild pigs (*Sus scrofa*) as a study system.

Chapter One investigates cross-species disease transmission between wildlife, domestic animals and humans, which is an increasing threat to public and veterinary health. The risk to agricultural and human health was investigated by evaluating the status of 84 pathogens; the host species most at risk for transmission; and the co-occurrence of wild pigs, agriculture and humans. This was accomplished with a combination of meta-analysis and network analysis approaches. Thirty-four economically important swine pathogens (bacterial, viral, and parasitic) that cause clinical disease in livestock, poultry, wildlife, and humans were identified with the potential for transmission.

Chapter Two investigates the conflicts between wildlife and agriculture and characterizes the processes that drive emergence of policy at the wildlife-agricultural interface. Using data describing congressional policy activity related to wild pigs, generalized linear models were used

to relate the frequency of policy activity to the frequency of negative newspaper articles and amount of the U.S. agricultural industry potentially impacted by wild swine over a 30-year period. A strong linkage between wild pig policy activity and predictors representing news media, specifically negativity of media, geographic distribution of media, and amount of agriculture potentially impacted were identified as important. Results suggest that agriculture and media coverage may act as determinants for wildlife-agricultural policy development.

Chapter Three investigates the ecological drivers of pathogen prevalence, specifically the role of species diversity. To accomplish this, a hierarchical Bayesian model that accounted for imperfect detection probability was used to investigate the influence of species diversity on the infection probability in wild pigs for pathogens with broad and narrow host ranges. Consistent with the species-diversity dilution hypothesis, prevalence of a single-host pathogen, pseudorabies virus, was negatively influenced by increasing richness of non-competent hosts. Contrary to the species-diversity amplification hypothesis, a multi-host pathogen, swine brucellosis, did not increase in prevalence as competent hosts increased in richness. Accounting for imperfect detection was important and indicated that processes other than diagnostic test error alone may be important for determining pathogen prevalence. Environmental gradients associated with changes in pathogen prevalence were linked to host species survival, specifically the severity of temperature and precipitation during the coldest period of the year. This together with species diversity may limit the ability of single-host pathogens to invade populations experiencing stressful conditions.

Chapter Four investigates environmental drivers of short-term population dynamics for invasive and native populations. Short-term transient population dynamics are common in vertebrates, particularly invasive vertebrates, and by their nature are directly influenced by the

interaction of population structure and vital rates. Using a novel methodological framework, we found consistent differences in the way vital rates and age structure in invasive and native wild pig populations contribute to transient dynamics suggesting that invasive and native populations are influenced by differing mechanisms. These dynamics appear to be linked with environmental conditions that regulate demography. Vital rates with the largest influence on population growth had the greatest variability across populations, contrary to the demographic buffering hypothesis. In native populations, vital rates contributed most to population growth. Invasive populations demonstrated a trade-off in the contribution of vital rates and age structure that may have unexpected consequences for invasive species management.

ACKNOWLEDGEMENTS

Many people have shaped my journey though graduate school. In particular, I want to thank my Ph.D. advisor, Colleen Webb, who has been a true mentor. Colleen encouraged me to pursue a Ph.D., believed in me, and showed endless patience. She has influenced me as a scientist in numerous ways. Her door has always been open and I will forever be thankful for her mentorship and friendship.

I also thank my committee members, Tom Hobbs, Susan Opp, and Mike Antolin. They have mentored me in many ways, have help transform my thinking as a scientist, and have provided critical suggestions to improve this dissertation. In particular I thank Tom for his significant influence in my approach ecological problems.

I also thank my wife, Marci Miller, whom without her support and encouragement I would not have been successful. She above all others has sacrificed an immeasurable amount to support me through graduate school. She never let me doubt myself. Thank you Marci. I also want to thank our son, Eli, whom made me smile every day and gave me a reason to finish. I would also like to thank my parents Mary Ann Miller and George Miller, and my sister Tara Miller whom have showed endless support and enthusiasm over the last four years.

Finally, I would like to thank the Webb lab, for encouragement, endless conversations about science, comradery, and laughs. The Webb lab is an impressive and accepting community and I look forward to working with many of you throughout my career. I also thank my coauthors S. Sweeney, C. Slootmaker, D. Grear, P. Salvo, D. Kiser, S. Shwiff, M. Tabak, S. Bevins, K. Pedersen; research librarian M. Foley; and colleagues K. Pepin and A. Davis.

Thanks to all!

DEDICATION

To my son Eli S. Miller, you have reminded me of what is important in life.

To my wife Marci C. Miller, for giving me the courage to seek truth and confidence in myself.

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INTRODUCTION

Policy to address human-wildlife conflict is often controversial. Developing policy to mitigate these conflicts is increasingly important and is often driven by both societal and biological factors. Yet the interaction between societal and biological drivers and the relative contribution of these to environmental policy remains understudied. Understanding these interactions requires both investigation of the latent biological processes that give rise to the conflict and also the societal perceptions of the results of these biological processes.

Conflicts between wildlife and agriculture are increasingly challenging agriculture and wildlife agencies (Krebs et al. 1998, Miller et al. 2013, Miller and Sweeney 2013). The implementation of policy to address human-wildlife conflicts is can be controversial (Messmer 2009). Developing policy to manage interactions between wildlife and agriculture has been identified as critically important (Jones et al. 2013), yet there remains little research on the societal factors that bring these issues to the policy making agenda for wildlife-agricultural conflicts. The policy process literature has identified drivers of policy creation such as problem severity, interest group involvement, media coverage, and public perception as predictive of the passage or modification of policies to address a social problem (Gilliam Jr and Iyengar 2000, Soroka 2003, Walgrave et al. 2008, Baumgartner and Jones 2010). However, these approaches and concepts are often not used by ecologists to understand the relationship between determinants of policy and biological processes.

Policy is often focused on mitigating diseases transmitted between humans, wildlife, and domestic animals that are increasingly challenging public and veterinary health systems (Jones et al. 2013, Miller et al. 2013). In North America, it is estimated that at least 79% of reportable

domestic animal diseases have a wildlife component associated with the transmission or the life cycle of the pathogen (Miller and Sweeney 2013). Similarly three-fourths of all emerging infectious diseases of humans are zoonotic with many having a wildlife reservoir (Taylor et al. 2001, Jones et al. 2008). Therefore, diseases that arise from the livestock–wildlife interface are of importance and must be an area of focus for public and veterinary health systems (Siembieda et al. 2011). Despite this importance, cross-species transmission is one of the least studied aspects of disease ecology (Lloyd-Smith et al. 2009, Luis et al. 2015).

Studies from a diversity of host-pathogen systems indicate that host species' diversity can influence the ability of a pathogen to establish (Johnson et al. (2015). Studies have examined relationships between the spatial distribution of disease and environmental variables for free living populations (Buskirk and Ostfeld 1998, Giraudoux et al. 2003). However there may be tradeoffs between species diversity and environmental conditions (Moore and Borer 2012), and this may be particularly important for invasive species or species pioneering new range. The interaction of environmental conditions influencing the host or pathogen may operate at scales different than those of species diversity generating asymmetric effects on pathogen transmission (Huang et al. 2016). These effects may also be different for pathogens with a narrow versus large host range. Yet studies investigating relationships between pathogen persistence, environmental factors, and species diversity for pathogens with narrow and wide host ranges are still relatively limited, particularly at the macro-scale and for mammal species in North America.

Studies of host–pathogen systems suggest that species diversity of ecological communities can alter the prevalence of pathogens and that this relationship can be influenced by population dynamics (Keesing et al. 2006, Ostfeld and Keesing 2012, Salkeld et al. 2013, Johnson et al. 2015, Huang et al. 2016). Short-term population growth and transient processes

play a key role in our ecological understanding (Hastings 2004) and often explain higher order processes of energy flux and loss that drive ecological systems (Rip and McCann 2011, Gellner et al. 2016). Short-term transient population dynamics are common in vertebrates, particularly invasive vertebrates, and can fundamentally influence the ability of a species to colonize new areas (McMahon and Metcalf 2008, Iles et al. 2016). A central theme in ecology that also bears on transient dynamics is the impact of variability in environmental conditions on population dynamics (Lande et al. 2003). Transient dynamics by their nature are directly influenced by deviations in population structure (e.g. age structure) from equilibrium as mediated by vital rates. These age structure deviations that manifest as transient dynamics are driven by both exogenous and endogenous factors, yet the linkage between transient population dynamics and environmental drivers are rarely studied. These relationships are fundamental to an improved ecological understanding of transient population dynamics, which is particularly relevant to invasive vertebrates and their management.

Here I investigate the interactions among drivers of wildlife-agricultural policy to improve an understanding of both the societal drivers and the biological drivers for a globally important invasive species - *Sus scrofa* the wild pig. I investigated the relative contribution of invasive species population growth and social discourse in generating national invasive species policy. To elucidate these biological processes to better understand policy opportunities and consequences, I investigated hypothesis related to ecological processes important for short-term population dynamics that contribute to invasive species population growth and the ecological drivers of pathogen prevalence for a single host and multi-host pathogen in wild pigs.

REFERENCES

Baumgartner, F. R., and B. D. Jones. 2010. Agendas and instability in American politics. University of Chicago Press.

Buskirk, J. V., and R. S. Ostfeld. 1998. Habitat heterogeneity, dispersal, and local risk of exposure to Lyme disease. Ecological Applications **8**:365-378.

Gellner, G., K. S. McCann, and A. Hastings. 2016. The duality of stability: towards a stochastic theory of species interactions. Theoretical Ecology **9**:477-485.

Gilliam Jr, F. D., and S. Iyengar. 2000. Prime suspects: The influence of local television news on the viewing public. American Journal of Political Science **44**:560-573.

Giraudoux, P., P. Craig, P. Delattre, G. Bao, B. Bartholomot, S. Harraga, J.-P. Quéré, F. Raoul, Y. Wang, and D. Shi. 2003. Interactions between landscape changes and host communities can regulate Echinococcus multilocularis transmission. Parasitology **127**:S121-S131.

Hastings, A. 2004. Transients: the key to long-term ecological understanding? Trends in Ecology & Evolution **19**:39-45.

Huang, Z., F. Van Langevelde, A. Estrada-Peña, G. Suzán, and W. De Boer. 2016. The diversity–disease relationship: evidence for and criticisms of the dilution effect. Parasitology **143**:1075-1086.

Iles, D. T., R. Salguero-Gómez, P. B. Adler, and D. N. Koons. 2016. Linking transient dynamics and life history to biological invasion success. Journal of Ecology **104**:399-408.

Johnson, P. T., R. S. Ostfeld, and F. Keesing. 2015. Frontiers in research on biodiversity and disease. Ecology letters **18**:1119-1133.

Jones, B. A., D. Grace, R. Kock, S. Alonso, J. Rushton, M. Y. Said, D. McKeever, F. Mutua, J. Young, and J. McDermott. 2013. Zoonosis emergence linked to agricultural intensification and environmental change. Proceedings of the National Academy of Sciences **110**:8399-8404.

Jones, K. E., N. G. Patel, M. A. Levy, A. Storeygard, D. Balk, J. L. Gittleman, and P. Daszak. 2008. Global trends in emerging infectious diseases. Nature **451**:990-993.

Keesing, F., R. D. Holt, and R. S. Ostfeld. 2006. Effects of species diversity on disease risk. Ecology letters **9**:485-498.

Krebs, J., R. Anderson, T. Clutton-Brock, C. Donnelly, S. Frost, W. Morrison, R. Woodroffe, and D. Young. 1998. Badgers and bovine TB: conflicts between conservation and health. Science **279**:817-818.

Lande, R., S. Engen, and B.-E. Saether. 2003. Stochastic population dynamics in ecology and conservation. Oxford University Press on Demand.

Lloyd-Smith, J. O., D. George, K. M. Pepin, V. E. Pitzer, J. R. Pulliam, A. P. Dobson, P. J. Hudson, and B. T. Grenfell. 2009. Epidemic dynamics at the human-animal interface. Science **326**:1362-1367.

Luis, A. D., T. J. O'Shea, D. T. Hayman, J. L. Wood, A. A. Cunningham, A. T. Gilbert, J. N. Mills, and C. T. Webb. 2015. Network analysis of host–virus communities in bats and rodents reveals determinants of cross-species transmission. Ecology letters **18**:1153-1162.

McMahon, S. M., and C. Metcalf. 2008. Transient sensitivities of non-indigenous shrub species indicate complicated invasion dynamics. Biological Invasions **10**:833-846.

Messmer, T. A. 2009. Human–wildlife conflicts: emerging challenges and opportunities.

Miller, R. S., M. L. Farnsworth, and J. L. Malmberg. 2013. Diseases at the livestock–wildlife interface: Status, challenges, and opportunities in the United States. Preventive veterinary medicine **110**:119-132.

Miller, R. S., and S. J. Sweeney. 2013. Mycobacterium bovis (bovine tuberculosis) infection in North American wildlife: current status and opportunities for mitigation of risks of further infection in wildlife populations. Epidemiology and infection **141**:1357-1370.

Moore, S. M., and E. T. Borer. 2012. The influence of host diversity and composition on epidemiological patterns at multiple spatial scales. Ecology **93**:1095-1105.

Ostfeld, R. S., and F. Keesing. 2012. Effects of host diversity on infectious disease. Annual Review of Ecology, Evolution, and Systematics **43**:157-182.

Rip, J., and K. McCann. 2011. Cross-ecosystem differences in stability and the principle of energy flux. Ecology letters **14**:733-740.

Salkeld, D. J., K. A. Padgett, and J. H. Jones. 2013. A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. Ecology letters **16**:679-686.

Siembieda, J., R. Kock, T. McCracken, and S. Newman. 2011. The role of wildlife in transboundary animal diseases. Animal Health Research Reviews **12**:95.

Soroka, S. N. 2003. Media, public opinion, and foreign policy. The International Journal of Press/Politics **8**:27-48.

Taylor, L. H., S. M. Latham, and E. Mark. 2001. Risk factors for human disease emergence. Philosophical Transactions of the Royal Society of London B: Biological Sciences **356**:983-989.

Walgrave, S., S. Soroka, and M. Nuytemans. 2008. The Mass Media's Political Agenda-Setting Power A Longitudinal Analysis of Media, Parliament, and Government in Belgium (1993 to 2000). Comparative Political Studies **41**:814-836.

CHAPTER 1

CROSS-SPECIES TRANSMISSION POTENTIAL BETWEEN WILD PIGS, LIVESTOCK, POULTRY, WILDLIFE, AND HUMANS: IMPLICATIONS FOR DISEASE RISK MANAGEMENT OF FREE-RANGING SWINE IN NORTH AMERICA

INTRODUCTION

Diseases transmitted between humans, wildlife, and domestic animals are increasingly challenging public and veterinary health systems (Jones et al. 2013, Miller et al. 2013). In North America, it is estimated that at least 79% of reportable domestic animal diseases have a wildlife component associated with the transmission of the pathogen and at least 40% are zoonotic (Miller et al. 2013). Similarly three-fourths of all emerging infectious diseases of humans are zoonotic and many are associated with wildlife reservoirs (Taylor et al. 2001, Jones et al. 2008). Therefore, diseases occurring at the livestock–wildlife interface are of paramount importance and must be an area of focus for public and veterinary health systems (Siembieda et al. 2011). Despite this importance cross-species transmission is one of the least studied aspects of disease ecology (Lloyd-Smith et al. 2009, Luis et al. 2015).

Wild pigs (*Sus scrofa*), that include feral domestic pigs (*Sus scrofa domestica*), Eurasian wild boar (*Sus scrofa linnaeus*), and hybrids between the two, are the most abundant free-ranging, exotic ungulates in North America (Bevins et al. 2014). Recently, wild pigs in North America have become of increasing concern as a potential veterinary and public health threat for cross-species transmission (Bevins et al. 2014, USDA 2015). Research and policy addressing wild pig disease has received increased attention in recent years (Barrios-Garcia and Ballari 2012, Bevins et al. 2014, USDA 2015). This is driven, in part, by substantial range expansion,

increasing ecological and agricultural damage, and increased involvement of wild pigs in disease transmission (Bevins et al. 2014). In North America, wild pigs have expanded their range to at least 41 states in the United States and three provinces in Canada since the 1960s (Bevins et al. 2014, Brook and van Beest 2014, USDA 2015) and recent modeling indicates that their potential range may be far greater (McClure et al. 2015).

In some parts of the world, wild pigs have been identified as an important reservoir for epidemic diseases, such as classical swine fever virus and African swine fever virus, that have the potential for serious socio-economic consequences (Jori and Bastos 2009, Reiner et al. 2009, Müller et al. 2011). These diseases, often termed transboundary animal diseases, can cause high morbidity and mortality in susceptible animal populations constituting a threat to national economies (Baldock et al. 1999). The cost of an outbreak of foot and mouth disease (FMD) involving wild pigs is estimated to range from USD$11.9 million to USD$5.8 billion (Cozzens et al. 2010). In addition disease risks posed by wild pigs to other domestic animals (i.e. cattle) are increasingly identified (Boadella et al. 2012, Pedersen et al. 2012, Bevins et al. 2014, Torre et al. 2015, USDA 2015). The potential for disease outbreaks to impact international trade may also be important (Coffey et al. 2005).

In addition to agricultural impacts, wild pigs are associated with a diversity of public health issues. Wild pigs have been implicated in the transmission of zoonotic viruses such as hepatitis E virus (HEV) (Li et al. 2005), trichinellosis (Rodríguez et al. 2004, Holzbauer et al. 2014), swine influenza virus (Feng et al. 2014), and Japanese encephalitis virus (Hamano et al. 2007). In addition to direct transmission, wild pigs have been identified as a contributor to O157:H7 *Escherichia coli* contamination in watersheds (Jay et al. 2007). Interest in the role of wild pigs in foodborne diseases has increased after outbreaks of *Salmonella spp*. in leafy green

crops such as spinach were traced back to farms in areas with wild pig populations (Jay et al. 2007, Jay-Russell et al. 2012).

The threats posed by diseases in wild pigs have been recognized in North America as well as globally for some pathogen-host systems (Bevins et al. 2014). A recent evaluation of 80,000 publications addressing wildlife-livestock diseases found that only 18% of the publications addressed the domestic swine interface and that this may be an important knowledge gap given global increases in swine production (Wiethoelter et al. 2015). While there have been numerous system specific studies investigating the role of wild pigs in pathogen transmission these studies are primarily limited to diseases of concern for domestic swine production (Reiner et al. 2009, Müller et al. 2011) or human health (Rodríguez et al. 2004, Holzbauer et al. 2014). As a result there is not currently an assessment across all economically important pathogens known to infect swine (domestic and wild) and the potential transmission of these pathogens between wild pigs, livestock, poultry, wildlife, and humans. Here, our objectives are three fold. First, we identify economically important pathogens (bacterial, viral, and parasitic) that are potentially shared between wild pigs, livestock, poultry, cervids, and humans. Second, we evaluate the reported prevalence of these pathogens in North American wild pig populations to assess any potential gaps in knowledge. Third, to illustrate the importance of disease risk management, we investigate the number of farms potentially at risk in the United States.

To achieve these objectives we used a common risk identification methodology to identify wild pig pathogens that can be shared between livestock, wildlife, and humans by evaluating susceptibility to these pathogens (OIE 1999, Wieland et al. 2011, Miller et al. 2013). We then used these data describing pathogen susceptibility by species to develop transmission

potential networks that describe the potential for pathogen sharing between species (Lloyd-Smith et al. 2009, Pilosof et al. 2015). Network metrics were used to identify species that had the highest potential for sharing of pathogens and identify pathogens that were most common across species. We identify gaps in knowledge required to inform surveillance, risk assessments, scientific studies, and risk mitigations for diseases of wild pigs and provide a discussion of these in the context of wild pig range overlap with agriculture in the United States.

METHODS

Assessment and identification of shared pathogens

First we considered 84 World Organisation for Animal Health (OIE) terrestrial pathogens that were listed beginning in 2013 (bee diseases were excluded) (OIE 2013). Each of these 84 pathogens was evaluated using the published literature to determine its reported ability to infect swine (wild and domestic), cattle, sheep, goats, horses, poultry, cervids (North American deer and elk species only), and humans. A priori we identified and used nine susceptibility categories to characterize the outcome of infection in each of these species (Table 1.1). For each pathogen, the scientific literature was reviewed from 1900 until present, based on this literature each species was assigned to the a-priori categories. The final set of classified pathogens by species was then reviewed independently by five veterinary epidemiologists to achieve consensus based on the supporting evidence for each assigned category. This independent review reduced potential bias that maybe associated with the literature search. Where possible, we used literature to confirm whether wild and domestic swine were equally susceptible to pathogens. When literature was unavailable to discern any differences between wild and domestic swine (the case with most pathogens) we assumed that domestic swine and wild pigs were similarly

affected. We summarized these data to describe the number of pathogens each species was susceptible.

Transmission Potential Networks

To investigate the species (hosts) and pathogens with the greatest potential to be involved in transmission we determined the degree of association among hosts with "transmission-potential networks" (TPN), where hosts were network nodes (swine, sheep, goat, cattle, cervid, poultry, equine, human) that were connected via edges defined by similarity in pathogen susceptibility (Luis et al. 2015, Pilosof et al. 2015). Thus edges are not equivalent to networks based on contact patterns. Edges in our transmission networks depict the potential for transmission between host species based on known etiology and host range for the pathogen rather than pathogen co-occurrence in space and time (VanderWaal et al. 2014, Pilosof et al. 2015). We define transmission potential to mean the likelihood that a given host species group will infect another species group, relative to other species in the network, based on species susceptibility to the pathogen. Thus, connected species form part of the same transmission chain (VanderWaal et al. 2014, Luis et al. 2015, Pilosof et al. 2015). Using methods similar to Pilosof et al. (2015) we generated eight TNPs for pathogens causing clinical or subclinical disease in swine. Four of these TPNs defined edges if two host types were clinically susceptible to the same pathogen and were constructed for all pathogens, bacterial pathogens, viral pathogens, and parasitic pathogens. An additional four TPNs defined edges if two host types were clinically, subclinically, or affected by the same pathogen.

The structural characteristics of these networks were evaluated using both edge and node level statistics. Edge weights in the TPNs where calculated for bacterial, viral and parasitic pathogens using the Jaccard index (Koleff et al. 2003), assuming a positive relationship of

pathogen infections shared by species and the likelihood that a pathogen would infect them both. An edge received a value of 0 when the species did not share any pathogens and a value of 1 when the pair of species was susceptible to the exact same pathogens. Index values closer to 1 indicate greater potential for transmission of pathogen types while values close to zero indicate no or limited potential transmission.

Eigenvalue centrality (EC) was used to quantify the importance of a species (node) in terms of promoting pathogen transmission potential among all species. With EC, a species group's importance increases when connections to other species that are themselves important increases (Newman 2010). EC thus enables quantification of the transmission potential of a species group among all species in the network (Canright and Engø-Monsen 2006, Griffin and Nunn 2012). To evaluate the relative importance of individual pathogens in the networks we used normalized degree centrality (DC) and EC metrics among the TPNs defined by each group of pathogens (Bacterial, viral, parasitic and all pathogens considered together). DC increases as more species are susceptible and received its maximum value of 1 when all species were susceptible to the pathogen (Everett and Borgatti 2005). EC for pathogens can be interpreted in the same way as species.

Assessment of current status of pathogens in North America

To generate data describing the current status of OIE listed pathogens in wild pigs in North America, we developed a method to sample from the scientific literature. First we used keywords to search three databases (PubMed, Scopus, and Web of Science) for papers reporting surveillance results, pathology, and case reports for wild pigs for any pathogen (Khan et al. 2003, Moher et al. 2009, Okoli 2012). We confined our search to the literature published in English since 1900. All scientific peer reviewed literature describing any wild pig pathogens in

North America was considered eligible. We assumed that these papers represented the known status of pathogens in swine in North America. Once all relevant sources were identified we reviewed each paper to ensure relevance. The numbers of papers reporting pathogen findings in wild pigs were tallied by pathogen to determine variability in known pathogen occurrence in wild pigs. We report the number of studies and the range of reported prevalence for pathogens in the North America.

Assessment of farms potentially at risk

To illustrate the potential risk to agriculture and humans resulting from exposure to wild pigs, we examined the co-occurrence of wild pigs, farms, and rural human population in the United States. A measure of the annual co-occurrence was developed using three data sources. Data reporting the number of farms by agricultural commodity and county was compiled from the National Agricultural Statistics Service (NASS) Quick Stats database (USDA 2014). We restricted our investigation to the commodities associated with the species investigated in Table 2. The county-level number of farms is available at a national scale for 2002, 2007, and 2012. For completeness, we included rural human population as a proxy for potential human-wild pig interaction. County-level estimates of rural human population are available from the 2010 census (Bureau 2010). The county level distribution (presence/absence) of wild pigs, were compiled from the Southeast Cooperative Wildlife Disease Study (SCWDS) (SCWDS 2013). The SCWDS data represent the known distribution of wild pig populations from 1982 until present. These data were merged to generate a database describing at the county-level the number of farms, rural human population size, and the presence or absence of wild pigs. We used only the 2010 census because it was closets to the mid-point of the wild pig data and changes in rural populations were small (mean = 0.29%) and bounded zero (range -0.07 to

0.74%) from 2012 to 2013 providing a good approximation of rural populations (Cromartie 2015). The national proportion of farms and rural populations co-occurring with wild pigs was then calculated for the years 2002, 2007, and 2012. We estimated the increase in the number of farms and human populations co-occurring with wild pigs using linear regression.

RESULTS

Identification of shared pathogens

Our assessment using a structured literature review and expert panel identified 39 (46%) of the 84 OIE terrestrial pathogens as those that can affect swine, with 22 (56%) viral, 9 (23%) bacterial, and 8 (21%) parasitic pathogens (Table 2). Of these 39 pathogens affecting swine, 33 (85%) caused clinical disease while only a few (4; 10%) were categorized as causing asymptomatic (or subclinical) infection or had documented natural infections in swine with unknown consequences in (2; 5%). Our assessment of all species' susceptibility to pathogens of swine found that of these 39 pathogens, 34 (87.2%) caused clinical or sub-clinical disease in at least one other species. On average 70% (±25%; ±StDev) of swine pathogens could infect other species (Table 3). Specifically, non-swine hosts were susceptible (clinical, subclinical, affected, and occasionally affected) to 80% (±32%) of bacterial, 56% (±13%) of viral, and 73% (±24%) of parasitic swine pathogens. All species except for poultry were susceptible to greater than 75% of bacterial pathogens. All species except poultry and cervids were susceptible to more than 75% of parasites; humans had the greatest number, being susceptible to 100% of parasites evaluated. Susceptibility to viral pathogens was the lowest among species. *Bovidea* (cattle, sheep, goat) were susceptible to greater than 60% of viral pathogens (see Table 2). Pathogens of swine causing clinical disease in other species were fewer on average with 58% (±25%) known to cause clinical disease in the species evaluated (Table 4). On average 73% (±29%) of bacterial,

39% (±13%) of viral, and 63% (±20%) of swine parasitic pathogens caused clinical disease in other species. All species except poultry and cervids had greater than 75% of swine bacterial pathogens causing clinical disease. Humans accounted for the greatest proportion of swine viral pathogens causing clinical disease (88%) while cattle, humans, and horse accounted for the greatest number of parasitic pathogens causing clinical disease. We also documented studies that specifically investigated wild pigs for susceptibility to domestic swine diseases. Nearly all 8 (80%) of the bacterial diseases had been investigated using wild pigs. Only 10 (37%) of the viral pathogens and 3 (37%) of the parasitic pathogens had been investigated in wild pigs.

Transmission Potential

Transmission potential, measured using the Jaccard index, between swine and other species demonstrated heterogeneity. Figure 1.1 illustrates the transmission potential between swine and other species. Members of the family *Bovidae* (cattle, sheep, goat) were important (upper 75th quartile Jaccard index) for all but parasitic pathogens causing clinical disease. When all pathogens were considered together cattle was the only species group in the upper 75th quartile. Transmission potential between swine and multiple species was greatest for bacterial pathogens with cattle, sheep, goat, and horse all having Jaccard index values in the upper 75th quartile. Viral pathogen transmission with swine was greatest for cattle and goats. In our study parasitic pathogen transmission potential with swine was highest for humans. In networks considering all types of susceptibility cattle, sheep, and goat had the greatest relative transmission potential with swine. There was little difference between bacterial pathogen networks for clinical susceptibility and all susceptibilities. Parasitic transmission potential with swine increased with sheep, horse and humans all in the upper 75th quartile.

Centrality for species demonstrated less heterogeneity (Table 3, Table 4). Cattle, sheep and goat consistently had the greatest centralities (EV=0.99±0.02) while poultry had the lowest network centrality (EV=0.40±0.16) across all networks and had the lowest centrality (EV=0.17) for bacterial pathogens. Human centrality (EV=0.85±0.06) was also low for all but the network considering all potential species susceptibilities to parasitic pathogens, in which it had the largest centrality (EV=0.99).

Pathogen centrality had greater heterogeneity when compared to species centrality. Twenty four (70.6%) pathogens had eigenvector centralities greater than 0.5 and normalized degree centralities greater than 0.5, indicating they could be transmitted to at least half of the species considered. Only nine (26.5%) of the pathogens had centrality values below 0.5. Bacteria on average had greater centrality (EV=0.86±0.13; DG=0.78±0.15) than viruses (EV=0.58±0.30; DG=0.52±0.30) and parasites (EV=0.73±0.29; DG=0.68±0.30). The upper 75th quartile of centralities were composed of three bacterial pathogens (*Bacillus anthracis, M. tuberculosis, B. abortus*), three parasitic pathogens (*Chrysomya putoria, Cochliomyia hominivorax, Echinococcosis sp.*), and one virus (*lyssaviruses sp.*) (Table S2). Pathogens with the smallest centralities were largely viral, with the lower 25th quartile of centralities composed of six viruses (*Equine influenza, Asfivirus sp., Pestivirus sp., Arterivirus sp., Enterovirus B, Alphacoronavirus 1*), and two parasites (*Trichinella spp, Taenia solium*).

Evaluation of Surveillance

Sampling of the literature for surveillance studies in North American wild pigs identified 72 publications reporting studies for 48 pathogens. The majority of studies 70 (97%) described surveillance findings from wild pig populations in the United States. The first publication we identified was from 1962 describing epidemiological findings for leptospirosis in Georgia while

the majority (61%) of publications were from the last 20 years. Ten pathogens accounted for 64% of the scientific studies with two, *Brucella suis* and *Suid herpesvirus* (Aujeszky's disease virus), accounting for 30% of studies (Figure 2; Table S3). Viral pathogens accounted for the largest number (49%) of surveillance studies while bacterial pathogens accounted for 35%. Thirteen parasites had surveillance studies and *Toxoplasma gondii* accounted for 33% of these studies. Only 49% of OIE listed swine diseases had published surveillance studies reporting findings (positive or negative) in wild pigs and 41% of studies described surveillance results for non-OIE listed pathogens. For pathogens of swine that cause clinical disease in other species 15 (45%) had surveillance studies published. Reported prevalence for these 18 pathogens ranged from 0% to 100%, with vesicular stomatitis virus having the highest reported prevalence (100%) for a single population on Ossobaw island, Georgia (Stallknecht et al. 1986).

Co-occurrence of Farms, Rural Populations and Wild pigs

The co-occurrence of wild pigs and farms for all commodities increased across the ten years investigated (Figure 1.3). For the year 2012 on average 47.7% (range 56.5-36.5%) of all farms were in counties with wild pigs representing 46.6% (range 77.3-11.3%) of all domestic animals. The geographic co-occurrence for 2012 is illustrated in Figure 4 and shows high densities of concordance in the Midwestern states of Texas, Oklahoma, Arkansas, western states of California and Oregon, and eastern states of South Carolina, North Carolina and Florida. Farmed cervids had the largest increase resulting in a 66.6% percentage increase in co-occurrence across the ten years. In 2012, 56.5% of all cervid farms representing 77.3% of all animals were in counties were wild pigs were present. Four of the seven agricultural commodities investigated had over 40% of farms in counties with wild pigs. Domestic swine, an agricultural commodity of concern for disease transmission from wild pigs, had a 58% increase

in co-occurrence and an annual rate of increase of 1.3% (95% CI = 1.0–1.7%), with 36.5% farms and 11.3% of animals in counties with wild pigs. Rural human populations had a 29.9% increase in co-occurrence with wild pigs and an annual rate of increase of 1.07% (95% CI = 0.5–1.7%). In 2012 an estimated 46.5% of all rural Americans lived in counties with wild pigs.

DISCUSSION

Properties of the transmission potential networks provide an increased understanding of the potential risks of pathogen sharing among species. The majority (87%) of swine pathogens can be transmitted to other species; however this transmission potential was not evenly distributed across species. Both the co-occurrence of wild pigs with family *Bovidae* (cattle, sheep, goat) and the importance of these species in the transmission networks indicate a risk for transmission between *Bovidae* species and wild pigs. *Bovidae* had the highest network metrics indicating greater relative importance among the species and across all swine pathogens. Central nodes are often interpreted in epidemiological networks as being important for network wide transmission (Craft and Caillaud 2011, Paull et al. 2012, Luis et al. 2015), and this interpretation has also been used for transmission networks based on pathogen susceptibility (Pilosof et al. 2015). This suggests that the family *Bovidae,* particularly cattle, may be important for transmitting pathogens between swine and other species. Commingling of cattle, sheep and goat with wild pig is common where domestic and wild ruminants share pasture resources throughout North America (Cooper et al. 2010, Miller et al. 2013). Based on our analysis of wild pig occurrence data, greater than 50% of all U.S. cattle, sheep and goat co-occur in a county with wild pigs. The introduction of pathogens into wildlife populations has been associated with commingling of livestock with wildlife, particularly cattle, in North America (Cross et al. 2007, Maichak et al. 2009).

In addition to species heterogeneity, pathogens demonstrated heterogeneity that maybe important for transmission. Vector borne pathogens made up less than 23% of pathogens indicating that those pathogens with direct transmission or transmission via fomites maybe relatively more important for cross species transmission of swine pathogens. Despite their low frequency vector borne pathogens were among the highest centralities (see Tables 3 and 4) for viral pathogens. The high potential for cross species transmission and the potential for expanding vector populations due to climate change (Rochlin et al. 2013) highlights the potential risk posed by these pathogens. Vector borne pathogens can be among the most difficult to control once established (Gubler 1998) and often cause long term challenges for disease risk mitigation.

Excluding vector borne pathogens, fourteen pathogens accounted for 77.4% of the pathogen network centrality, with greater than two thirds of these being bacterial and parasitic. In the case of bacterial pathogens, *B. abortus* and *M. bovis* had the highest centrality, when *B. anthracis* a pathogen commonly transmitted in the environment, was excluded. These two pathogens have challenged disease control programs in North America for over a century. More recently wild pigs have been established as a maintenance host for *M. bovis* in several populations globally (Aranaz et al. 2004, Santos et al. 2009) and may pose a risk for transmission in North America (Pedersen et al. 2016). Cross species transmission may be of particular concern in regions with increased commingling of at-risk cattle with wild pigs (Cooper et al. 2010) and in regions such as Michigan where *M. bovis* is endemic in wildlife (Ramsey et al. 2014). Broadly our network centrality findings were similar to an inventory of known livestock pathogens that found 77% infect multiple hosts (Cleaveland et al. 2001), a study of human pathogens that found 73% are zoonotic (Woolhouse and Gowtage-Sequeria 2006), and a

study of OIE domestic animal pathogens that found 79% can be transmitted between wildlife and domestic animals (Miller et al. 2013).

Non-vector borne viral pathogens with the largest connectance between wild pigs and other species included avian influenza, the causative agents for foot and mouth disease and Aujeszky's disease. Given the recent emergence of highly pathogenic avian influenza in North America (Bevins et al. 2016), the potential for swine (domestic or wild) to influence antigenic changes in the virus (Kuntz-Simon and Madec 2009), and serologic evidence of wild pigs being exposed to influenza (Hall et al. 2008, Feng et al. 2014), highlights the potential importance of surveillance in domestic and wild pigs for pathogens such as influenza. In North America, wild pigs have been documented to be actively infected with and having contributed to the transmission of only a fraction of the pathogens we investigated and their contribution to the persistence of these pathogens is still largely uncharacterized (Bevins et al. 2014) . Given the large number of swine pathogens we found shared among species, the potential for wild pigs to become an unmonitored reservoir for pathogens may be a concern requiring further inquiry.

Despite effort to establish prevalence estimates for wild pigs (see supplemental Table 3), there are gaps for pathogens of interest for human, wildlife, and livestock health. We found discordance between the available surveillance studies and the pathogens that can be shared across species. More than 50% of pathogens that cause clinical disease in other species did not have any North America studies of prevalence in wild pigs. This contrasts with the potential exposure of livestock to wild pigs; domestic animals such as cattle and sheep, that are largely pasture raised in North America, have a potential for coming into contact (directly or indirectly) with wild pigs (Barasona et al. 2014, Cowie et al. 2016) and share nearly 90% (see Table 4) of swine pathogens causing clinical infection. Those studies that do report prevalence are generally

limited to local or regional studies (Van der Leek et al. 1993b, Corn et al. 2009). While providing important data, local studies may not represent regional or national prevalence. We found only a few studies (Pedersen et al. 2012, Pedersen et al. 2013) that report prevalence and epidemiological patterns of infection at national or near national scales. Pathogens that did have multiple studies in different regions (e.g. leptospirosis, pseudorabies virus, swine brucellosis, and bovine tuberculosis) had prevalence estimates that ranged from 0-87% indicating spatial heterogeneity in prevalence and in turn transmission risk likely occur. This result may be complicated by true and false detection errors that few studies addressed when reporting findings (McClintock et al. 2010) and can have large effects on estimated disease prevalence in wildlife (Jennelle et al. 2007). Comprehensive surveillance systems integrating livestock, wildlife, and human components have been identified as a need (Stallknecht 2007). Explicitly accounting for the transmission potential and historic geospatial distribution of pathogens to prioritize surveillance (both livestock and wildlife) may offer benefits and reduce knowledge gaps for pathogens of concern for human, wildlife, and livestock health (McKenzie et al. 2007). Developing a comprehensive national monitoring system that integrates domestic and wild animal surveillance, prioritizes pathogens based on transmission risk, potential consequences, and knowledge of occurrence could yield economic benefits for livestock health by reducing spillover events through early detection and risk mitigation (Jebara 2004, Wendt et al. 2015).

Incomplete knowledge of the presence of pathogens in wild pig populations and the transmission potential we found may pose risks for foreign animal diseases in North America where wild pigs are potential hosts. The potential economic impacts resulting from disease outbreaks that include wildlife can be large (Epstein et al. 2006, Anderson et al. 2010, Cozzens et al. 2010, O'Brien et al. 2011) and have long lasting effects on economies and production

systems (Epstein et al. 2006, Knight-Jones and Rushton 2013). Livestock production in the United States that is increasingly interconnected and concentrated (Reimer 2006, Martinez 2012), is also becoming more globally reliant (Bonanno 1994, McCullough et al. 2008). The importance of exports in sustaining market opportunities for U.S. agriculture has increased, with over 20% of production value exported in 2012 (Jerardo 2012). As a result disease threats to food safety or livestock health that may originate in wildlife have the potential to impact economies (Fidler 1996, Daszak et al. 2000, Jones et al. 2008). Despite potential economic impacts, assessments that explicitly link disease outbreaks involving wildlife and livestock with changes in export value are currently unavailable. Methods that link disease risk at the wildlife-livestock interface and compare the benefits and costs of risk management (e.g. surveillance, bio-security, etc) in both livestock, wildlife have been proposed (Horan and Fenichel 2007, Miller et al. 2013, Shwiff et al. 2016), however they have not been extended to risk management at a macro-economic scale.

Further, the expansion of wild pigs has resulted in a large portion of agriculture production and human populations occurring in regions where wild pigs are present (Figure 4). For the livestock commodities we investigated all had large proportions of farms in regions with wild pigs and none had declines in co-occurrence with wild pig populations. This large proportion of overlap of agricultural and rural populations is increasing as wild pig populations expand in North America (Snow et al. 2017). Pathogen exposure risk to both agriculture and humans, along with the potential economic impacts (Anderson et al. 2016), highlights the need for quantitative analysis and consequence assessments of the risks wild pigs pose to agriculture and human health (Miller et al. 2013). Recent analysis by Tompkins et al. (2015) found that disease emergence at the wildlife-livestock interface is often driven by human-induced activities

and exposure to domestic animals. Further, Jones et al. (2013) estimated that the rate of future zoonotic disease emergence/reemergence will be closely linked to changes in the agricultural-wildlife nexus. Several studies (Jones et al. 2013, Miller et al. 2013, Tompkins et al. 2015) have also found that available research and tools inadequately addresses these complex problems limiting prediction, prevention, and mitigation. Given the findings of these studies and ours, developing approaches for the wildlife-livestock interface that link risk assessments and economic consequence assessments allowing evaluation of the relative benefits and costs of surveillance and risk mitigation would be broadly useful, not only for invasive wild pigs, but for a diversity of wildlife-agricultural disease conflicts.

Our transmission networks highlight the potential for cross species transmission between wild pigs, livestock, cervids, and humans. They also highlight heterogeneity in both species and pathogens indicating some species are more important and that some pathogens maybe more frequently transmitted. Additional work is needed to establish the risk of exposure and transmission for pathogens of concern to humans and livestock and may necessitate surveillance studies elucidating potential risks for pathogens of greatest transmission potential. While a complete picture of the risks of wild pig associated diseases is not currently possible, the risk assessment process is valuable for prioritizing knowledge gaps. Evaluation of potential, but unstudied, impact of wild pigs on the consequences of reportable diseases (e.g. outbreak duration, extent, effectiveness of disease management) maybe warranted. As the first assessment of cross-species diseases associated with wild pigs, these results are a first step to characterizing and prioritizing the disease risks as wild pig populations expand.

Table 1.1. Susceptibility categories used to describe infection in the species group. Categories were established a-priori and used to denote the potential impact in each of these species based on available scientific literature.

Category	Code	Description
Clinical	C	Capable of developing clinical disease but can also be subclinical in some circumstances.
Subclinical	SC	Can be infected but does not develop clinical disease.
Affected	A	Species group is known to be susceptible (including seropositive) however it is unclear if they become clinical or subclinical hosts.
Occasional	O	Occasionally reported, but is rare or atypical in species group.
Uncertain	U	Some evidence suggests the species may be affected; however scientific evidence is currently unclear, lacking, or anecdotal.
Experimental	EX	Species group can become experimentally infected however natural infection is unknown or not reported.
Definitive Host	DH	Species group is considered the definitive host for the parasite.
Intermediate Host	IH	Species group is considered the intermediate host for the parasite.
Dead-end Host	*	Species group is considered a dead-end host for the parasite.

Table 1.3. All swine pathogens causing clinical, sub-clinical disease in livestock, poultry, cervids and humans.

	Cattle	Sheep	Goats	Horse	Cervids	Poultry	Humans	Mean	StDev
% Shared									
Bacterial	100	100	100	100	75	12.5	75	80.4	32.2
Parasitic	75	87.5	75	87.5	62.5	25	100	73.2	24.4
Viral	66.7	61.9	71.4	52.4	47.6	33.3	57.1	55.8	12.8
All	75.7	75.7	78.4	70.3	56.8	27	70.3	64.9	18.1
Eigenvalue Centrality									
Bacterial	1	1	1	1	0.8	0.171	0.8	0.824	0.303
Parasitic	0.993	0.979	1	0.876	0.784	0.65	0.923	0.886	0.130
Viral	0.983	1	0.983	0.97	0.774	0.42	0.991	0.874	0.216
All	0.995	0.992	1	0.924	0.79	0.456	0.904	0.866	0.195

Table 1.4. Swine pathogens that cause clinical disease in livestock, poultry, cervids and humans.

	Cattle	Sheep	Goats	Horse	Cervids	Poultry	Humans	Mean	StdDev
% Shared									
Bacterial	87.5	87.5	87.5	100	62.5	12.5	75	73.2	29.3
Viral	75	62.5	62.5	75	50	25	87.5	62.5	20.4
Parasitic	42.9	42.9	47.6	42.9	28.6	14.3	52.4	38.8	13
All	59.5	56.8	59.5	62.2	40.5	16.2	64.9	51.4	17.4
Eigenvalue Centrality									
Bacterial	1	1	1	0.982	0.774	0.2	0.768	0.818	0.292
Viral	0.906	1	0.977	0.831	0.769	0.415	0.877	0.825	0.198
Parasitic	1	0.977	0.977	1	0.77	0.53	0.847	0.872	0.175
All	0.975	1	0.993	0.925	0.774	0.373	0.839	0.840	0.222

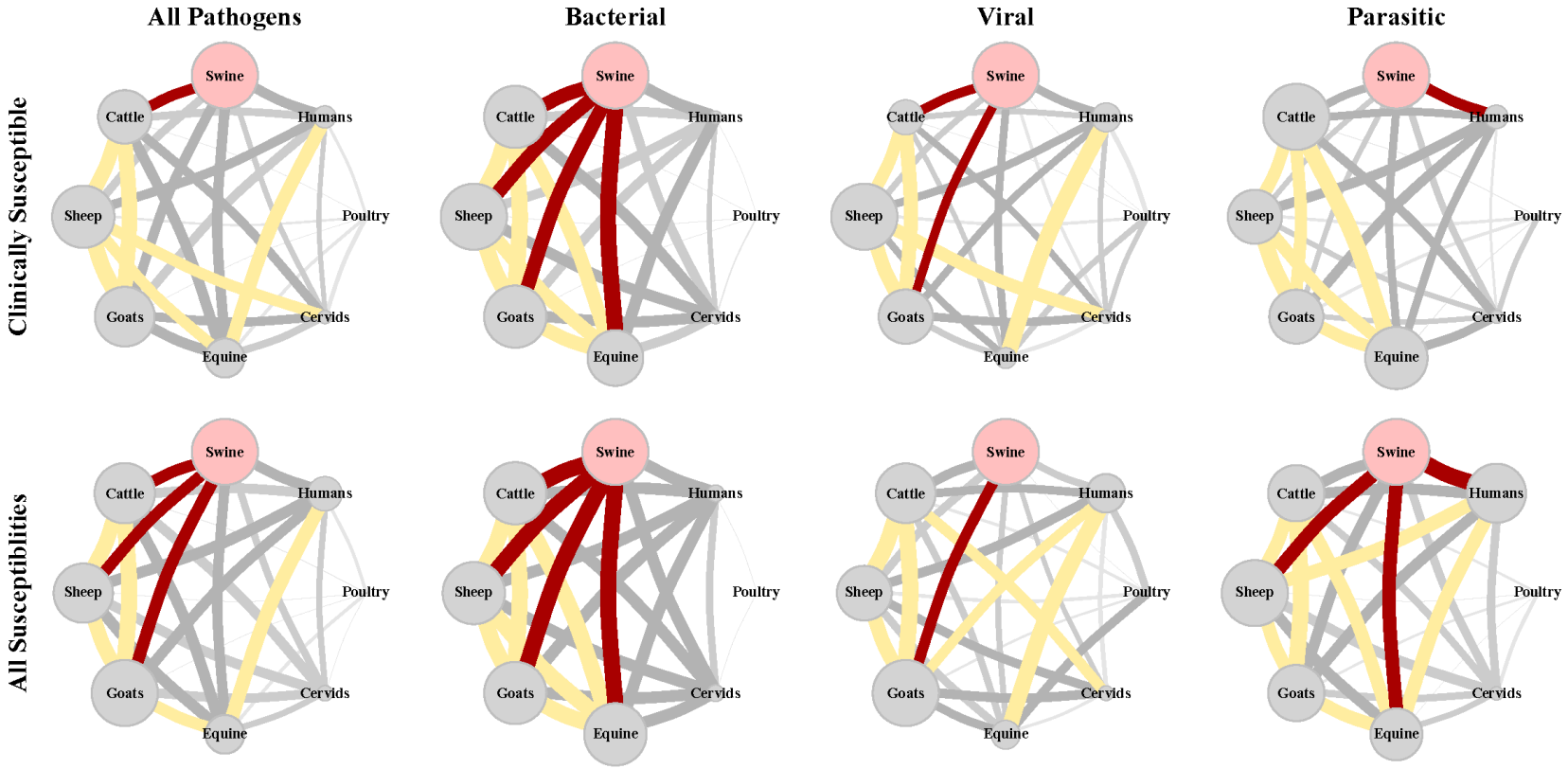


Figure 1.1. Transmission potential networks used in this study created by connecting two host species if they were susceptible to the same pathogen causing clinical or subclinical disease in swine. Top row are pathogens causing clinical disease in non-swine hosts and the bottom row are all pathogens affecting non-swine hosts. Edge weight between two species is the similarity in the pathogens infecting a pair of host species calculated with the Jaccard index. Red edges denote Jaccard index in the upper 75th quartile, while light gray are edges in the lower 25th quartile. Node size indicates the relative centrality of the species group in the transmission network, calculated using the eigenvalue centrality – more central nodes are larger.

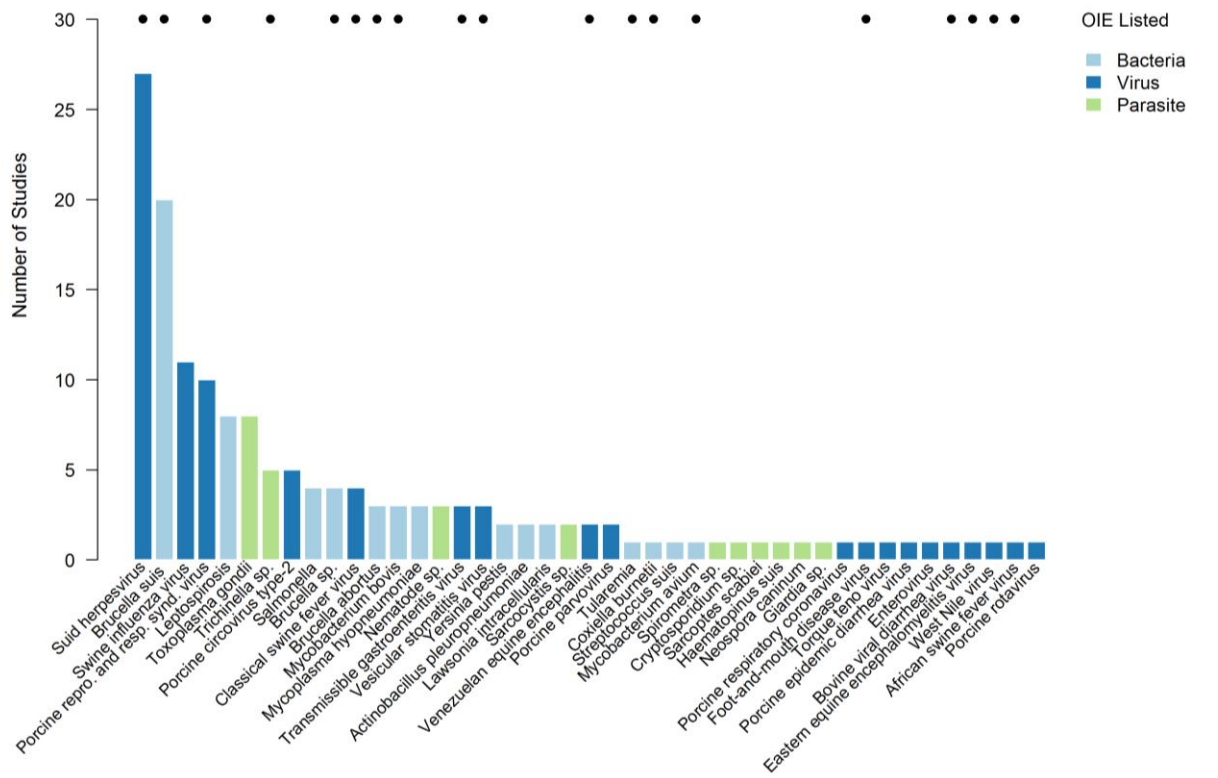


Figure 1.2. Total number of scientific peer reviewed publications reporting results of prevalence studies for wild pigs in North America. Dots along top margin indicate those that are OIE listed pathogens. Ten pathogens accounted for 69% of the scientific studies with two, *Brucella suis* and *Suid herpesvirus*, accounting for 30% of studies and viral pathogens accounted for the largest number (49%) of surveillance studies. Only 49% of OIE listed swine diseases had published surveillance studies reporting findings (positive or negative) in wild pigs and many prevalence studies reported findings for non-OIE listed pathogens.

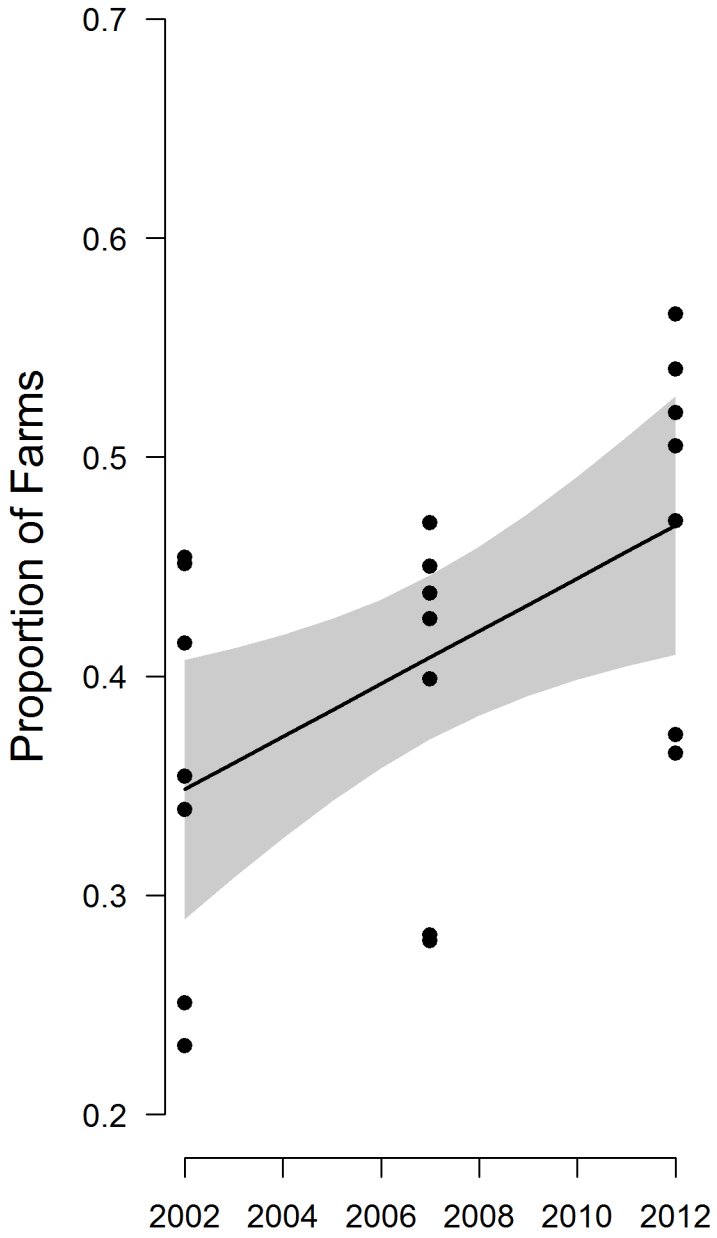


Figure 1.3. Increase in the proportion of United States farms co-occurring with wild pigs over the ten years we investigated. Solid black line is a linear regression line fit to the data and gray bands indicate 95% confidence intervals.

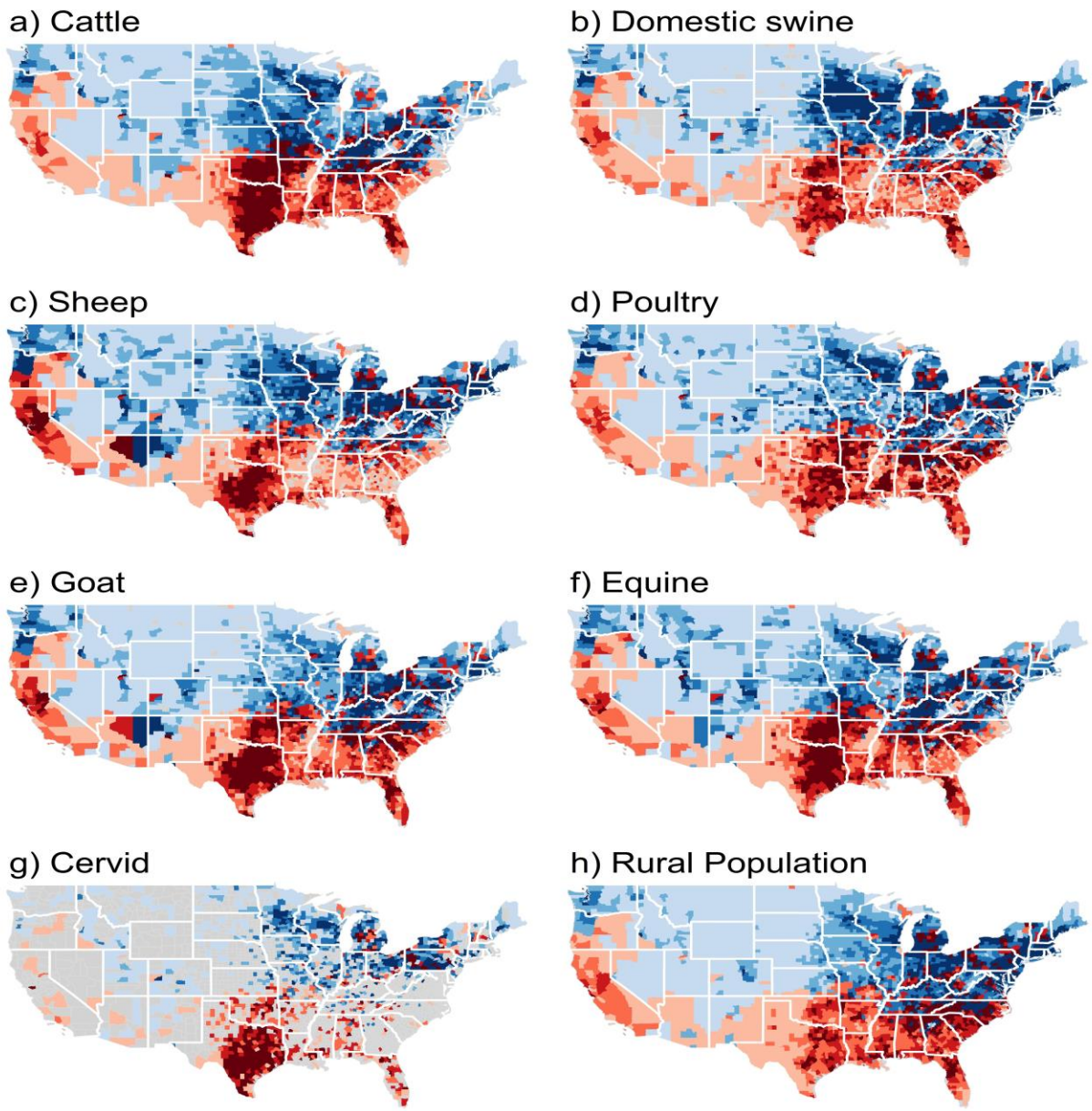


Figure 1.4. County level co-occurrence of wild pigs, agricultural commodities, and rural human populations in the contiguous United States for 2012. Red shading denotes by quartile the absolute farms density (farms per km2) or rural human population density (people per km2) within counties co-occurring with wild pigs while blue shading indicates counties without wild pigs. Maps were generated by combining publically available data (see methods) describing wild pig distribution from Southeast Cooperative Wildlife Disease Study (SCWDS), agriculture data from National Agricultural Statistics Service (NASS) Quick Stats database, and rural human population data available from the United States Census Bureau. Maps were created using the maptools package version 0.9.2 in R version 3.3.0.

REFERENCES

Aitken, I. 2008. Diseases of sheep. 4 edition. Blackwell Publishing, Victoria, Australia.

Anderson, A., C. Slootmaker, E. Harper, J. Holderieath, and S. A. Shwiff. 2016. Economic estimates of feral swine damage and control in 11 US states. Crop Protection **89**:89-94.

Anderson, D., A. Hagerman, P. Teel, G. Wagner, J. Outlaw, and B. Herbst. 2010. Economic impact of expanded fever tick range. Agricultural & Food Policy Center, Texas A&M University, College Station, TX.

Aranaz, A., L. de Juan, N. Montero, C. Sánchez, M. Galka, C. Delso, J. Alvarez, B. Romero, J. Bezos, and A. I. Vela. 2004. Bovine tuberculosis (Mycobacterium bovis) in wildlife in Spain. Journal of Clinical Microbiology **42**:2602-2608.

Baker, S. R., K. M. O'Neil, S. A. Dee, and M. R. Gramer. 2011. Estimates of the seroprevalence of production-limiting diseases in wild pigs. Veterinary record **168**:564.

Baldock, C., T. Forman, B. Geering, B. Taylor, G. Myrdal, R. Mayorga, E. Beaumont Roveda, C. A. Quesada Mateo, M. Carley, R. Pampillón Olmedo, and S. Fankhauser. 1999. New Technologies in the fight against transboundary animal diseases, Food and Agriculture Organizations of the United Nations, Rome, Italy.

Barasona, J. A., M. C. Latham, P. Acevedo, J. A. Armenteros, A. D. M. Latham, C. Gortazar, F. Carro, R. C. Soriguer, and J. Vicente. 2014. Spatiotemporal interactions between wild boar and cattle: implications for cross-species disease transmission. Veterinary research **45**:122.

Barrett, T., and P. Rossiter. 1999. Rinderpest: the disease and its impact on humans and animals. Advances in Virus Research **53**:89-110.

Barrios-Garcia, M. N., and S. A. Ballari. 2012. Impact of wild boar (Sus scrofa) in its introduced and native range: a review. Biological Invasions **14**:2283-2300.

Bevins, S. N., R. J. Dusek, C. L. White, T. Gidlewski, B. Bodenstein, K. G. Mansfield, P. DeBruyn, D. Kraege, E. Rowan, C. Gillin, B. Thomas, S. Chandler, J. Baroch, B. Schmit, M. J. Grady, R. S. Miller, M. L. Drew, S. Stopak, B. Zscheile, J. Bennett, J. Sengl, C. Brady, H. S. Ip, E. Spackman, M. L. Killian, M. K. Torchetti, J. M. Sleeman, and T. J. Deliberto. 2016. Widespread detection of highly pathogenic H5 influenza viruses in wild birds from the Pacific Flyway of the United States. Scientific Reports **6**:28980.

Bevins, S. N., K. Pedersen, M. W. Lutman, T. Gidlewski, and T. J. Deliberto. 2014. Consequences Associated with the Recent Range Expansion of Nonnative Feral Swine. BioScience **64**:291-299.

Boadella, M., C. Gortázar, J. Vicente, and F. Ruiz-Fons. 2012. Wild boar: an increasing concern for Aujeszky's disease control in pigs? BMC veterinary research **8**:7.

Bonanno, A. 1994. From Columbus to ConAgra: The globalization of agriculture and food. University Press of Kansas.

Breard, E., G. Belbis, C. Viarouge, M. Riou, A. Desprat, J. Moreau, E. Laloy, G. Martin, P. Sarradin, and D. Vitour. 2013. Epizootic hemorrhagic disease virus serotype 6 experimentation on adult cattle. Research in veterinary science **95**:794-798.

Brody, J. A., and W. A. Murray Jr. 1959. Arthropod-borne encephalitis in the United States, 1957. Public health reports **74**:461.

Brook, R. K., and F. M. van Beest. 2014. Feral wild boar distribution and perceptions of risk on the central Canadian prairies. Wildlife Society Bulletin **38**:486-494.

Bureau, U. S. C. 2010. Census 2010; Summary File 1; Table P2. American FactFinder

Canright, G. S., and K. Engø-Monsen. 2006. Spreading on networks: a topographic view. Complexus **3**:131-146.

Chatfield, J., M. Milleson, R. Stoddard, D. M. Bui, and R. Galloway. 2013. Serosurvey of leptospirosis in feral hogs (Sus scrofa) in Florida. Journal of Zoo and Wildlife Medicine **44**:404-407.

Cleaveland, S., M. Laurenson, and L. Taylor. 2001. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. Philosophical Transactions of the Royal Society of London B: Biological Sciences **356**:991-999.

Coetzer, J., G. Thomson, and R. Tustin. 2004. Infectious diseases of livestock. Oxford University Press Southern Africa.

Coffey, B., J. Mintert, S. Fox, T. C. Schroeder, and L. Valentin. 2005. The economic impact of BSE on the US beef industry: Product value losses, regulatory costs, and consumer reactions. Kansas State University. Manhattan, Kansas.

Cook, R. A. 2005. Emerging diseases at the interface of people, domestic animals and wildlife. The role of wildlife in our understanding of highly pathogenic avian influenza. The Yale journal of biology and medicine **78**:343.

Cooper, S. M., H. M. Scott, G. R. De la Garza, A. L. Deck, and J. C. Cathey. 2010. Distribution and interspecies contact of feral swine and cattle on rangeland in south Texas: implications for disease transmission. Journal of wildlife diseases **46**:152-164.

Corn, J. L., J. C. Cumbee, R. Barfoot, and G. A. Erickson. 2009. Pathogen exposure in feral swine populations geographically associated with high densities of transitional swine premises and commercial swine production. Journal of wildlife diseases **45**:713-721.

Corn, J. L., D. E. Stallknecht, N. M. Mechlin, M. P. Luttrell, and J. R. Fischer. 2004. Persistence of pseudorabies virus in feral swine populations. Journal of wildlife diseases **40**:307-310.

Cowie, C. E., M. R. Hutchings, J. A. Barasona, C. Gortázar, J. Vicente, and P. C. White. 2016. Interactions between four species in a complex wildlife: livestock disease community: implications for Mycobacterium bovis maintenance and transmission. European journal of wildlife research **62**:51-64.

Cozzens, T., K. Gebhardt, S. Shwiff, M. Lutman, K. Pedersen, and S. Swafford. 2010. Modeling the economic impact of feral swine-transmitted foot-and-mouth disease: a case study from Missouri. Pages 308-311 *in* R. M. Timm and K. A. Fagerstone, editors. Proceedings 24th Vertebrate Pest Confrence. University of California Davis.

Craft, M. E., and D. Caillaud. 2011. Network models: an underutilized tool in wildlife epidemiology? Interdisciplinary perspectives on infectious diseases **2011**:12.

Cromartie, J. 2015. Population and Migration. USDA Economic Research Service.

Cross, P. C., W. H. Edwards, B. M. Scurlock, E. J. Maichak, and J. D. Rogerson. 2007. Effects of management and climate on elk brucellosis in the Greater Yellowstone Ecosystem. Ecological Applications **17**:957-964.

Dardiri, A., L. Logan, and C. Mebus. 1987. Susceptibility of white-tailed deer to experimental heartwater infections. Journal of wildlife diseases **23**:215-219.

Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife--threats to biodiversity and human health. Science **287**:443.

Drew, M. L., D. A. Jessup, A. A. Burr, and C. Franti. 1992. Serologic survey for brucellosis in feral swine, wild ruminants, and black bear of California, 1977 to 1989. Journal of wildlife diseases **28**:355-363.

Duncan, C., H. Van Campen, S. Soto, I. K. LeVan, L. A. Baeten, and M. W. Miller. 2008. Persistent bovine viral diarrhea virus infection in wild cervids of Colorado. Journal of veterinary diagnostic investigation **20**:650-653.

Elvinger, F., C. A. Baldwin, A. D. Liggett, K. N. Tang, and D. E. Stallknecht. 1996. Prevalence of Exposure to Eastern Equine Encephalomyelitis Virus in Domestic and Feral Swine in Georgia. J Vet Diagn Invest **8**:481-484.

Emord, D., and C. Morris. 1984. Epizootiology of eastern equine encephalomyelitis virus in upstate new york, usa vi. Antibody prevalence in wild birds during an interepizootic period. Journal of medical entomology **21**:395-404.

Epstein, J. H., H. E. Field, S. Luby, J. R. Pulliam, and P. Daszak. 2006. Nipah virus: impact, origins, and causes of emergence. Current Infectious Disease Reports **8**:59-65.

Essey, M., R. Payne, E. Himes, and D. Luchsinger. 1981. Bovine tuberculosis surveys of axis deer and feral swine on the Hawaiian island of Molokai. Pages 538-549 *in* Proceedings of the United States Animal Health Association.

Everett, M. G., and S. P. Borgatti. 2005. Extending centrality. Models and methods in social network analysis **35**:57-76.

Feng, Z., J. A. Baroch, L.-P. Long, Y. Xu, F. L. Cunningham, K. Pedersen, M. W. Lutman, B. S. Schmit, A. S. Bowman, and T. J. DeLiberto. 2014. Influenza A Subtype H3 Viruses in Feral Swine, United States, 2011–2012. Emerging Infectious Diseases **20**:843.

Fidler, D. P. 1996. Globalization, international law, and emerging infectious diseases. Emerging Infectious Diseases **2**:77.

Gajadhar, A. A., J.-R. Bisaillon, and G. D. Appleyard. 1997. Status of Trichinella spiralis in domestic swine and wild boar in Canada. Canadian Journal of Veterinary Research **61**:256.

Gibbs, S. E., N. L. Marlenee, J. Romines, D. Kavanaugh, J. L. Corn, and D. E. Stallknecht. 2006. Antibodies to West Nile virus in feral swine from Florida, Georgia, and Texas, USA. Vector-Borne & Zoonotic Diseases **6**:261-265.

Gresham, C. S., C. A. Gresham, M. J. Duffy, C. T. Faulkner, and S. Patton. 2002. Increased prevalence of Brucella suis and pseudorabies virus antibodies in adults of an isolated feral swine population in coastal South Carolina. Journal of wildlife diseases **38**:653-656.

Griffin, R. H., and C. L. Nunn. 2012. Community structure and the spread of infectious disease in primate social networks. Evolutionary Ecology **26**:779-800.

Gubler, D. J. 1998. Resurgent vector-borne diseases as a global health problem. Emerging Infectious Diseases **4**:442.

Guo, Y., M. Wang, Y. Kawaoka, O. Gorman, T. Ito, T. Saito, and R. G. Webster. 1992. Characterization of a new avian-like influenza A virus from horses in China. Virology **188**:245-255.

Guo, Y., M. Wang, G.-S. Zheng, W.-k. Li, Y. Kawaoka, and R. G. Webster. 1995. Seroepidemiological and molecular evidence for the presence of two H3N8 equine influenza viruses in China in 1993–94. Journal of general virology **76**:2009-2014.

Hall, J. S., R. B. Minnis, T. A. Campbell, S. Barras, R. W. DeYoung, K. Pabilonia, M. L. Avery, H. Sullivan, L. Clark, and R. G. McLean. 2008. Influenza exposure in United States feral swine populations. Journal of wildlife diseases **44**:362-368.

Hamano, M., C. Lim, H. Takagi, K. Sawabe, M. Kuwayama, N. Kishi, I. Kurane, and T. Takasaki. 2007. Detection of antibodies to Japanese encephalitis virus in the wild boars in Hiroshima prefecture, Japan. Epidemiology and infection **135**:974-977.

Hartin, R. E., M. R. Ryan, and T. A. Campbell. 2007. Distribution and disease prevalence of feral hogs in Missouri. **1**:186-191.

Holzbauer, S. M., W. A. Agger, R. L. Hall, G. M. Johnson, D. Schmitt, A. Garvey, H. S. Bishop, H. Rivera, M. E. de Almeida, and D. Hill. 2014. Outbreak of Trichinella spiralis infections associated with a wild boar hunted at a game farm in Iowa. Clinical Infectious Diseases **59**:1750-1756.

Horan, R. D., and E. P. Fenichel. 2007. Economics and ecology of managing emerging infectious animal diseases. American journal of agricultural economics **89**:1232-1238.

Jay-Russell, M., A. Bates, L. Harden, W. Miller, and R. Mandrell. 2012. Isolation of Campylobacter from feral swine (Sus scrofa) on the ranch associated with the 2006 Escherichia coli O157: H7 spinach outbreak investigation in California. Zoonoses and public health **59**:314-319.

Jay, M. T., M. Cooley, D. Carychao, G. W. Wiscomb, R. A. Sweitzer, L. Crawford-Miksza, J. A. Farrar, D. K. Lau, J. O'Connell, and A. Millington. 2007. Escherichia coli O157: H7 in feral swine near spinach fields and cattle, central California coast. Emerging Infectious Diseases **13**:1908.

Jebara, K. B. 2004. Surveillance, detection and response: managing emerging diseases at national and international levels. Rev Sci Tech **23**:709-715.

Jennelle, C. S., E. G. Cooch, M. J. Conroy, and J. C. Senar. 2007. State-specific detection probabilities and disease prevalence. Ecological Applications **17**:154-167.

Jerardo, A. 2012. Export Share of Production. USDA Economic Research Service.

Jones, B. A., D. Grace, R. Kock, S. Alonso, J. Rushton, M. Y. Said, D. McKeever, F. Mutua, J. Young, and J. McDermott. 2013. Zoonosis emergence linked to agricultural intensification and environmental change. Proceedings of the National Academy of Sciences of the United States of America **110**:8399.

Jones, K. E., N. G. Patel, M. A. Levy, A. Storeygard, D. Balk, J. L. Gittleman, and P. Daszak. 2008. Global trends in emerging infectious diseases. Nature **451**:990-993.

Jori, F., and A. D. Bastos. 2009. Role of wild suids in the epidemiology of African swine fever. EcoHealth **6**:296-310.

Kalthoff, D., B. Hoffmann, T. Harder, M. Durban, and M. Beer. 2008. Experimental infection of cattle with highly pathogenic avian influenza virus (H5N1). Emerging Infectious Diseases **14**:1132.

Khan, K. S., R. Kunz, J. Kleijnen, and G. Antes. 2003. Five steps to conducting a systematic review. Journal of the Royal Society of Medicine **96**:118-121.

Knight-Jones, T., and J. Rushton. 2013. The economic impacts of foot and mouth disease–What are they, how big are they and where do they occur? Preventive veterinary medicine **112**:161-173.

Koleff, P., K. J. Gaston, and J. J. Lennon. 2003. Measuring beta diversity for presence–absence data. Journal of animal ecology **72**:367-382.

Kramer, L. D., J. Li, and P.-Y. Shi. 2007. West Nile virus. The Lancet Neurology **6**:171-181.

Kumar, R. 1999. Viral encephalitis of public health significance in India: Current status. The Indian Journal of Pediatrics **66**:73-83.

Kuntz-Simon, G., and F. Madec. 2009. Genetic and antigenic evolution of swine influenza viruses in Europe and evaluation of their zoonotic potential. Zoonoses and public health **56**:310-325.

Leiby, P. D., W. P. Carney, and C. E. Woods. 1970. Studies on sylvatic echinococcosis. III. Host occurrence and geographic distribution of Echinococcus multilocularis in the north central United States. The Journal of parasitology:1141-1150.

Li, T.-C., K. Chijiwa, N. Sera, T. Ishibashi, Y. Etoh, Y. Shinohara, Y. Kurata, M. Ishida, S. Sakamoto, and N. Takeda. 2005. Hepatitis E Virus Transmission from Wild Boar Meat. Emerging Infectious Diseases **11**:1958.

Lipatov, A. S., Y. K. Kwon, L. V. Sarmento, K. M. Lager, E. Spackman, D. L. Suarez, and D. E. Swayne. 2008. Domestic pigs have low susceptibility to H5N1 highly pathogenic avian influenza viruses. PLoS pathogens **4**:e1000102.

Lloyd-Smith, J. O., D. George, K. M. Pepin, V. E. Pitzer, J. R. Pulliam, A. P. Dobson, P. J. Hudson, and B. T. Grenfell. 2009. Epidemic dynamics at the human-animal interface. science **326**:1362-1367.

Luis, A. D., T. J. O'Shea, D. T. Hayman, J. L. Wood, A. A. Cunningham, A. T. Gilbert, J. N. Mills, and C. T. Webb. 2015. Network analysis of host–virus communities in bats and rodents reveals determinants of cross-species transmission. Ecology letters **18**:1153-1162.

Maichak, E. J., B. M. Scurlock, J. D. Rogerson, L. L. Meadows, A. E. Barbknecht, W. H. Edwards, and P. C. Cross. 2009. Effects of management, behavior, and scavenging on risk of brucellosis transmission in elk of western Wyoming. Journal of wildlife diseases **45**:398-410.

Martinez, S. 2012. Vertical coordination of marketing systems: lessons from the poultry, egg and pork industries. Page 45 *in* Department of Agriculture Economic Research Service, editor. United States of America.

McClintock, B. T., J. D. Nichols, L. L. Bailey, D. I. MacKenzie, W. Kendall, and A. B. Franklin. 2010. Seeking a second opinion: uncertainty in disease ecology. Ecology letters **13**:659-674.

McClure, M. L., C. L. Burdett, M. L. Farnsworth, M. W. Lutman, D. M. Theobald, P. D. Riggs, D. A. Grear, and R. S. Miller. 2015. Modeling and Mapping the Probability of Occurrence of Invasive Wild Pigs across the Contiguous United States. PloS one **10**:e0133771.

McCullough, E. B., P. L. Pingali, and K. G. Stamoulis. 2008. The transformation of agri-food systems: globalization, supply chains and smallholder farmers. Food and Agriculture Organization of the United Nations and Earthscan, London, United Kingdom.

McKenzie, J., H. Simpson, and I. Langstaff. 2007. Development of methodology to prioritise wildlife pathogens for surveillance. Preventive veterinary medicine **81**:194-210.

Miller, D. L., Z. A. Radi, C. Baldwin, and D. Ingram. 2005. Fatal West Nile virus infection in a white-tailed deer (Odocoileus virginianus). Journal of wildlife diseases **41**:246-249.

Miller, R. S., M. L. Farnsworth, and J. L. Malmberg. 2013. Diseases at the livestock–wildlife interface: Status, challenges, and opportunities in the United States. Preventive veterinary medicine **110**:119-132.

Moher, D., A. Liberati, J. Tetzlaff, D. G. Altman, and P. G. The. 2009. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med **6**:e1000097.

Morens, D. M., and J. K. Taubenberger. 2010. An avian outbreak associated with panzootic equine influenza in 1872: an early example of highly pathogenic avian influenza? Influenza and other respiratory viruses **4**:373-377.

Morner, T. 1992. The ecology of tularaemia. Rev Sci Tech **11**:1123-1130.

Müller, T., E. Hahn, F. Tottewitz, M. Kramer, B. Klupp, T. Mettenleiter, and C. Freuling. 2011. Pseudorabies virus in wild swine: a global perspective. Archives of Virology **156**:1691-1705.

Murrell, K., F. Stringfellow, J. Dame, D. Leiby, C. Duffy, and G. Schad. 1987. Trichinella spiralis in an agricultural ecosystem. II. Evidence for natural transmission of Trichinella spiralis spiralis from domestic swine to wildlife. The Journal of parasitology **73**:103-109.

Nemeth, N. M., and R. A. Bowen. 2007. Dynamics of passive immunity to West Nile virus in domestic chickens (Gallus gallus domesticus). The American journal of tropical medicine and hygiene **76**:310-317.

Nettles, V. F., J. L. Corn, G. A. Erickson, and D. A. Jessup. 1989. A survey of wild swine in the United States for evidence of hog cholera. Journal of wildlife diseases **25**:61-65.

New Jr, J. C., K. Delozier, C. E. Barton, P. J. Morris, and L. N. Potgieter. 1994. A serologic survey of selected viral and bacterial diseases of European wild hogs, Great Smoky Mountains National Park, USA. Journal of wildlife diseases **30**:103-106.

Newman, M. 2010. Networks: an introduction. Oxford University Press, Oxford, United Kingdom.

O'Brien, D. J., S. M. Schmitt, S. D. Fitzgerald, and D. E. Berry. 2011. Management of bovine tuberculosis in Michigan wildlife: current status and near term prospects. Veterinary microbiology **151**:179-187.

OIE. 1999. Working group on risk analysis., World Organisation for Animal Health Regional Commission for the Americas.

OIE. 2013. Terrestrial Animal Health Code. World Organisation for Animal Health, Paris, France.

Okoli, C. 2012. A Critical Realist Guide to Developing Theory with Systematic Literature Reviews. Social Science Research Network.

Olsen, B., V. J. Munster, A. Wallensten, J. Waldenström, A. D. Osterhaus, and R. A. Fouchier. 2006. Global patterns of influenza A virus in wild birds. science **312**:384-388.

Passler, T., P. H. Walz, S. S. Ditchkoff, M. D. Givens, H. S. Maxwell, and K. V. Brock. 2007. Experimental persistent infection with bovine viral diarrhea virus in white-tailed deer. Veterinary microbiology **122**:350-356.

Paull, S. H., S. Song, K. M. McClure, L. C. Sackett, A. M. Kilpatrick, and P. T. Johnson. 2012. From superspreaders to disease hotspots: linking transmission across hosts and space. Frontiers in Ecology and the Environment **10**:75-82.

Pedersen, K., S. N. Bevins, J. A. Baroch, J. C. Cumbee Jr, S. C. Chandler, B. S. Woodruff, T. T. Bigelow, and T. J. DeLiberto. 2013. Pseudorabies in feral swine in the United States, 2009-2012. Journal of wildlife diseases **49**:709-713.

Pedersen, K., S. N. Bevins, B. S. Schmit, M. W. Lutman, M. P. Milleson, C. T. Turnage, T. T. Bigelow, and T. J. Deliberto. 2012. Apparent prevalence of swine brucellosis in feral swine in the United States. Human-Wildlife Interactions **6**:38-47.

Pedersen, K., R. S. Miller, T. D. Anderson, K. L. Pabilonia, J. R. Lewis, R. L. Mihalco, C. Gortazar, and T. Gidlewski. 2016. Limited Antibody Evidence of Exposure to

Mycobacterium bovis in Feral Swine (Sus scrofa) in the USA. Journal of wildlife diseases **53**:30-36.

Pilosof, S., S. Morand, B. R. Krasnov, and C. L. Nunn. 2015. Potential parasite transmission in multi-host networks based on parasite sharing. PloS one **10**:e0117909.

Ramsey, D. S., D. J. O'brien, M. K. Cosgrove, B. A. Rudolph, A. B. Locher, and S. M. Schmitt. 2014. Forecasting eradication of bovine tuberculosis in Michigan white-tailed deer. The Journal of Wildlife Management **78**:240-254.

Randhawa, A., V. Kelly, and E. Baker Jr. 1977. Agglutinins to Coxiella burnetii and Brucella spp, with particular reference to Brucella canis, in wild animals of southern Texas. Journal of the American Veterinary Medical Association **171**:939-942.

Reimer, J. J. 2006. Vertical integration in the pork industry. American journal of agricultural economics **88**:234-248.

Reiner, G., C. Fresen, S. Bronnert, and H. Willems. 2009. Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) infection in wild boars. Veterinary microbiology **136**:250-258.

Rochlin, I., D. V. Ninivaggi, M. L. Hutchinson, and A. Farajollahi. 2013. Climate change and range expansion of the Asian tiger mosquito (Aedes albopictus) in Northeastern USA: implications for public health practitioners. PloS one **8**:e60874.

Rodríguez, d. l. P. E., M. Rodriguez-Ferrer, J. Nieto-Martinez, F. Ubeira, and T. Garate-Ormaechea. 2004. Trichinellosis outbreaks in Spain (1990-2001). Enfermedades infecciosas y microbiología clínica **22**:70-76.

Rossiter, P., E. S. Williams, L. Munson, and S. Kennedy. 2001. Morbilliviral diseases. Infectious Diseases of Wild Mammals, Third Edition:37-76.

Ruder, M. G., A. B. Allison, D. E. Stallknecht, D. G. Mead, S. M. McGraw, D. L. Carter, S. V. Kubiski, C. A. Batten, E. Klement, and E. W. Howerth. 2012. Susceptibility of white-tailed deer (Odocoileus virginianus) to experimental infection with epizootic hemorrhagic disease virus serotype 7. Journal of wildlife diseases **48**:676-685.

Saliki, J. T., S. J. Rodgers, and G. Eskew. 1998. Serosurvey of selected viral and bacterial diseases in wild swine from Oklahoma. Journal of wildlife diseases **34**:834-838.

Sandfoss, M., C. DePerno, S. Patton, J. Flowers, and S. Kennedy-Stoskopf. 2011. Prevalence of antibody to Toxoplasma gondii and Trichinella spp. in feral pigs (Sus scrofa) of eastern North Carolina. Journal of wildlife diseases **47**:338-343.

Sandfoss, M. R., C. S. de Perno, M. B. Palamar, C. W. Betsill, G. Erickson, and S. Kennedy-Stoskopf. 2012. A serosurvey for Brucella suis, classical swine fever virus, porcine circovirus type 2, and pseudorabies virus in feral swine (Sus scrofa) of eastern North Carolina. Journal of wildlife diseases **48**:462-466.

Santos, N., M. Correia-Neves, S. Ghebremichael, G. Källenius, S. B. Svenson, and V. Almeida. 2009. Epidemiology of Mycobacterium bovis infection in wild boar (Sus scrofa) from Portugal. Journal of wildlife diseases **45**:1048-1061.

Schmitt, S. M., T. M. Cooley, S. D. Fitzgerald, S. R. Bolin, A. Lim, S. M. Schaefer, M. Kiupel, R. K. Maes, S. A. Hogle, and D. J. O'brien. 2007. An outbreak of eastern equine encephalitis virus in free-ranging white-tailed deer in Michigan. Journal of wildlife diseases **43**:635-644.

Scott, G. R. 1963. Pigs and Rift Valley Fever. Nature **200**:919-920.

SCWDS. 2013. National Feral Swine Distribution Maps. National Feral Swine Mapping System. Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA.

Shwiff, S. A., S. J. Sweeney, J. L. Elser, R. S. Miller, M. Farnsworth, P. Nol, S. S. Shwiff, and A. M. Anderson. 2016. A benefit-cost analysis decision framework for mitigation of disease transmission at the wildlife–livestock interface. Human-Wildlife Interactions **10**:91.

Siembieda, J., R. Kock, T. McCracken, and S. Newman. 2011. The role of wildlife in transboundary animal diseases. Animal Health Research Reviews **12**:95.

Smith, P. 1968. Bovine-Type Tuberculosis Infection in Feral Swine. Master Thesis. University of California, Davis, California.

Snow, N. P., M. A. Jarzyna, and K. C. VerCauteren. 2017. Interpreting and predicting the spread of invasive wild pigs. Journal of Applied Ecology. DOI: 10.1111/1365-2664.12866.

Spickler, A. R. 2007. Anthrax, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2008. Eastern Equine Encephalomyelitis, Western Equine Encephalomyelitis and Venezuelan Equine Encephalomyelitis, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2009a. Bovine Brucellosis: Brucella abortus, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2009b. Bovine Tuberculosis, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2009c. Hemorrhagic Septicemia, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2010. Emerging and exotic diseases of animals. CFSPH Iowa State University.

Spickler, A. R. 2011. Echinococcosis, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2013. Leptospirosis, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Spickler, A. R. 2014. Influenza, <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php> (Accessed April 2, 2016).

Stallknecht, D. 2007. Impediments to wildlife disease surveillance, research, and diagnostics. Pages 445-461 *in* J. E. Childs, J. S. Mackenzie, and J. A. Richt, editors. Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances and Consequences of Cross-Species Transmission. Springer Berlin Heidelberg.

Stallknecht, D., V. Nettles, G. Erickson, and D. Jessup. 1986. Antibodies to vesicular stomatitis virus in populations of feral swine in the United States. Journal of wildlife diseases **22**:320-325.

Stallknecht, D., V. Nettles, W. Fletcher, and G. Erickson. 1985. Enzootic vesicular stomatitis New Jersey type in an insular feral swine population. American journal of epidemiology **122**:876-883.

Stallknecht, D. E., D. M. Kavanaugh, J. L. Corn, K. Eernisse, J. A. Comer, and V. F. Nettles. 1993. Feral swine as a potential amplifying host for vesicular stomatitis virus New Jersey serotype on Ossabaw Island, Georgia. Journal of wildlife diseases **29**:377-383.

Stevenson, G. W., H. Hoang, K. J. Schwartz, E. B. Burrough, D. Sun, D. Madson, V. L. Cooper, A. Pillatzki, P. Gauger, and B. J. Schmitt. 2013. Emergence of porcine epidemic diarrhea virus in the United States: clinical signs, lesions, and viral genomic sequences. Journal of veterinary diagnostic investigation. ***25***:649-654.

Stoffregen, W. C., S. C. Olsen, C. J. Wheeler, B. J. Bricker, M. V. Palmer, A. E. Jensen, S. M. Halling, and D. P. Alt. 2007. Diagnostic characterization of a feral swine herd enzootically infected with Brucella. Journal of veterinary diagnostic investigation **19**:227-237.

Storandt, S., D. Virchow, M. Dryden, S. E. Hygnstrom, and K. Kazacos. 2002. Distribution and prevalence of Echinococcus multilocularis in wild predators in Nebraska, Kansas, and Wyoming. Journal of Parasitology **88**:420-422.

Storandt, S. T., and K. R. Kazacos. 1993. Echinococcus multilocularis identified in Indiana, Ohio, and east-central Illinois. The Journal of parasitology. **79**:301-305.

Tate, C. M., E. W. Howerth, D. E. Stallknecht, A. B. Allison, J. R. Fischer, and D. G. Mead. 2005. Eastern equine encephalitis in a free-ranging white-tailed deer (Odocoileus virginianus). Journal of wildlife diseases **41**:241-245.

Taylor, L. H., S. M. Latham, and E. Mark. 2001. Risk factors for human disease emergence. Philosophical Transactions of the Royal Society of London B: Biological Sciences **356**:983-989.

Thompson, R., A. Boxell, B. Ralston, C. Constantine, R. Hobbs, T. Shury, and M. Olson. 2006. Molecular and morphological characterization of Echinococcus in cervids from North America. Parasitology **132**:439-447.

Tompkins, D. M., S. Carver, M. E. Jones, M. Krkošek, and L. F. Skerratt. 2015. Emerging infectious diseases of wildlife: a critical perspective. Trends in parasitology **31**:149-159.

Torre, A., J. Bosch, I. Iglesias, M. Muñoz, L. Mur, B. Martínez-López, M. Martínez, and J. Sánchez-Vizcaíno. 2015. Assessing the risk of African swine fever introduction into the European Union by wild boar. Transboundary and emerging diseases **62**:272-279.

Turnbull, P., M. Doganay, P. Lindeque, B. Aygen, and J. McLaughlin. 1992. Serology and anthrax in humans, livestock and Etosha National Park wildlife. Epidemiology and infection **108**:299-313.

USDA. 2014. Quick stats. National Agricultural Statistics Service, US Department of Agriculture, Washington, DC.

USDA. 2015. Final Environmental Impact Statement, Feral Swine Damage Management: A National Approach. Page 550 *in* Department of Agriculture, editor. United States of America, Washington D.C.

Van Der Leek, M., H. Becker, P. Humphrey, C. Adams, R. Belden, W. Frankenberger, and P. Nicoletti. 1993a. Prevalence of Brucella sp. antibodies in feral swine in Florida. Journal of wildlife diseases **29**:410-415.

Van der Leek, M., H. Becker, E. Pirtle, P. Humphrey, C. Adams, B. All, G. Erickson, R. Belden, W. Frankenberger, and E. Gibbs. 1993b. Prevalence of pseudorabies (Aujeszky's disease) virus antibodies in feral swine in Florida. Journal of wildlife diseases **29**:403-409.

Van der Meulen, K., M. Pensaert, and H. Nauwynck. 2005. West Nile virus in the vertebrate world. Archives of Virology **150**:637-657.

VanderWaal, K. L., E. R. Atwill, L. A. Isbell, and B. McCowan. 2014. Quantifying microbe transmission networks for wild and domestic ungulates in Kenya. Biological Conservation **169**:136-146.

Webb, P. A., R. G. McLean, G. C. Smith, J. H. Ellenberger, D. B. Francy, T. E. Walton, and T. P. Monath. 1987. Epizootic vesicular stomatitis in Colorado, 1982: some observations on the possible role of wildlife populations in an enzootic maintenance cycle. Journal of wildlife diseases **23**:192-198.

Wendt, A., L. Kreienbrock, and A. Campe. 2015. Zoonotic disease surveillance–inventory of systems integrating human and animal disease information. Zoonoses and public health **62**:61-74.

Wieland, B., S. Dhollander, M. Salman, and F. Koenen. 2011. Qualitative risk assessment in a data-scarce environment: A model to assess the impact of control measures on spread of African Swine Fever. Preventive veterinary medicine **99**:4-14.

Wiethoelter, A. K., D. Beltrán-Alcrudo, R. Kock, and S. M. Mor. 2015. Global trends in infectious diseases at the wildlife–livestock interface. Proceedings of the National Academy of Sciences **112**:9662-9667.

Williams, E. S., and I. K. Barker. 2008. Infectious diseases of wild mammals. 3 edition. John Wiley & Sons, Ames, Iowa, USA.

Woods, R., E. Pirtle, J. Sacks, and E. Gibbs. 1990. Serologic survey for transmissible gastroenteritis virus neutralizing antibodies in selected feral and domestic swine sera in the southern United States. Journal of wildlife diseases **26**:420-422.

Woolhouse, M. E., and S. Gowtage-Sequeria. 2006. Host range and emerging and reemerging pathogens. The National Academies Press, Washington, D.C.

Wyckoff, A. C., S. E. Henke, T. A. Campbell, D. G. Hewitt, and K. C. VerCauteren. 2009. Feral swine contact with domestic swine: a serologic survey and assessment of potential for disease transmission. Journal of wildlife diseases **45**:422-429.

Zimmerman, J. J., L. A. Karriker, A. Ramirez, K. J. Schwartz, and G. W. Stevenson. 2012. Diseases of Swine. 10 edition. Wiley-Blackwell, West Sussex, UK.

Zygmont, S., V. Nettles, E. Shotts Jr, W. Carmen, and B. Blackburn. 1982. Brucellosis in wild swine: a serologic and bacteriologic survey in the southeastern United States and Hawaii. Journal of the American Veterinary Medical Association **181**:1285-1287.

CHAPTER 2

DETERMINANTS OF EMERGING POLICY ASSOCIATED WITH WILDLIFE AGRICULTURAL CONFLICTS

INTRODUCTION

Conflicts between wildlife and agriculture are increasingly challenging agriculture and wildlife agencies (Krebs et al. 1998, Miller et al. 2013, Miller and Sweeney 2013). Policy to address human-wildlife conflicts is often controversial (Messmer 2009). Developing policy to manage interactions between wildlife and agriculture has been identified as critically important (Jones et al. 2013), yet there remains little research on the societal factors that bring these issues to the policy making agenda for wildlife-agricultural conflicts. The policy process literature has identified drivers of policy creation such as problem severity, interest group involvement, media coverage, and public perception as predictive of the passage or modification of policies to address a social problem (Gilliam Jr and Iyengar 2000, Soroka 2003, Walgrave et al. 2008, Baumgartner and Jones 2010).

Wildlife-agriculture conflict and policy development is often exacerbated by invasive or exotic animals (Pimental 2007). In the United States (U.S.) there are at least 30 species of exotic free-ranging mammals which have become established since European colonization, causing an estimated $46 billion in damage annually (McKnight 1964, Mayer and Brisbin 1991, Pimental 2007). Wild swine (*Sus scrofa*), which include wild swine (*Sus scrofa domestica*), Eurasian Russian boar (*Sus scrofa linnaeus*), and hybrids between the two, are the most abundant free-ranging, invasive ungulate in the U.S. and annually cause an estimated USD$1.5 billion in damage (Pimental 2007, Bevins et al. 2014). Since the 1960s wild swine have expanded their

range to at least 38 States and 3 provinces in Canada and continue to increase (Bevins et al. 2014, Brook and Beest 2014). This range expansion has contributed to the impact of wild swine on ecosystems and both livestock and crop agricultural systems in North America (Bevins et al. 2014). However economic impacts and perceived problem severity often differ regionally and by agricultural commodity. For example annual crop damage in California is estimated to be USD$1.7 million while in Georgia it is estimated to be at least USD$57 million annually (Frederick 1998, Mengak 2012). In contrast the economic impacts associated with a cattle-wild swine outbreak of foot and mouth disease (FMD) in the United States have been estimated to be has high as USD$14 billion (Paarlberg et al. 2002). Despite the economic costs wild swine is also valued as a hunting resource and in many states is managed as a wildlife resource.

Wild swine impacts are often recognized at a local or regional scale, with national scale policy development only recently evident. In addition, there is a diversity of public attitudes toward wild swine encompassing, agricultural pest, disease hazard, commodity, source of income, and recreational resource (Tisdell 1982, Izac and O'Brien 1991). Izac and O'Brien (1991) found that these perceptions changed with location, time and individual. The combination of public attitude heterogeneity in the regions with wild swine and a neutral or undefined policy image - how a problem and its solution is defined, understood, and discussed - may contribute to the diversity of management positions on wild swine damage control and mitigation. Schattschneider (1960) noted that the essence of policy conflict over a public issue is the scope of participation and the importance of policy image in defining the problem and the solutions. Kingdon and Thurber (1984) found that identifying the visible, and presumably most affected, participants in a policy issue is central to understanding the dynamics of agenda setting and the resulting policy development. Furthermore it is traditionally accepted that problem

severity is a significant stimulus for the adoption of policy innovations (Sapat 2004). In the case of wild swine, the primary interest group impacted by increasing problem severity is agriculture (Bevins et al. 2014). However, public perception concerning an issue is also important in prioritizing the policy agenda (Baumgartner and Jones 2010).

Media coverage of public issues – both quantity and tone - has been widely recognized as an important driver in shaping national public perception and policy agendas (Gilliam Jr and Iyengar 2000, Walgrave et al. 2008, Baumgartner and Jones 2010). Media coverage is generally thought to influence policy agendas in two primary ways. First, media coverage can influence the relative salience (importance or prominence) of a particular pubic issue through repeated coverage over time (Soroka 2003, Baumgartner and Jones 2010). Second, media coverage can influence public and policy conceptualization about an issue and coalescence – how an issue is understood, defined, and framed (Elder and Cobb 1983). This conceptualization of an issue can influence the perception of the possible solutions and the importance of addressing the problem with governmental policy (Weart 1988, Baumgartner and Jones 2010). However, there is mixed evidence for how these factors - problem severity, interest groups, media coverage, and public perception - may act together to influence policy generation (McCombs and Shaw 1972, Funkhouser and Shaw 1990, Entman 1993, Koch-Baumgarten and Voltmer 2010).

Our objectives were to characterize the relative influence of the factors that led to the establishment of the APHIS National Feral Swine Damage Management Program in 2013 (federal government fiscal year 2014). Specifically we wanted to understand 1) the significance of public policy image on congressional policy activity; 2) to assess the influence of problem severity and broad governmental institutional pressures associated with expansion of wild swine at a national level on policy activity; and 3) to identify predictors of policy activity for informing

wildlife and agricultural interface management; specifically program assessments and new program development. Here we use the term 'policy' in its broadest definition referring not only to operational policies of government but also including all dialogue related to the development of policy. To investigate the relationship between policies, wild swine, and agriculture we use 29 years of data from three primary datasets – number of wild swine related policy actions (response variable), newspaper headline data, and the amount of agriculture in wild swine regions. Based on studies suggesting a strong dependence of policy change on changes in public policy image (Jones and Baumgartner 2004, Baumgartner and Jones 2010), specifically increased policy activity when public policy images become negative, we hypothesized that significant increase in the number of negative newspaper articles would act as a mechanism for influencing policy activity and provide a link between changes in policy and expanding wild swine populations. Because governmental institutions tend to increase stability in policy areas (Jones et al. 2003, Baumgartner and Jones 2010), we hypothesized that increasing the amount of agriculture in wild swine regions might be related to increasing problem severity and result in increased pressures on federal governmental institutions. Thus increasing policy activity as agricultural related interests increased demands for policy solutions to wild swine related issues – agricultural damage and economic losses. In our statistical models, we wanted to estimate these effects and determine if these patterns are consistent with increased policy activity. The broader goal of this analysis is to provide a mechanistic understanding of the policy image and institutional conditions that give rise to variations in the policy process, which enables improved response to changes in conditions that impact both wildlife and agricultural policy.

METHODS

Congressional Policy Action Data

A systematic search of the United States Government Printing Office Federal Digital System (FDsys) (GPO 2014) was used to generate data describing congressional activity related to wild swine. FDsys is an official repository of all official publications from all three branches of the United States Federal Government and currently contains over 7.4 million electronic documents from 1969 to present. Our search included congressional hearings, congressional record, congressional reports, bills, and changes to the code of federal regulations from 1985 until 2013 when the APHIS National Feral Swine Damage Management Program was established. Documents included in our study contained any of the following terms: 'feral swine', 'feral hog', or 'feral pig', 'wild swine', 'wild hog', or 'wild pig'. Each document was considered an independent policy action, and the number of documents by year was tallied to generate count data by document type, primary agricultural commodity (livestock or crop) the document addressed, and year. Our method may have included documents which were not specifically addressing wild swine related policy; to evaluate this assumption a 5% random sample was taken and the documents were classified as addressing wild swine related policy or not. Based on the results of this assessment we assumed that if the document contained reference to wild swine the issue of wild swine was either on the policy agenda or influencing the agenda in some way.

Media Data

To generate data on media reporting of wild swine related topics a systematic search of four major news consolidators was performed – Newsbank, LexisNexis, EBSCO, and ProQuest (EBSCO 2016, LexisNexis 2016, NewsBank 2016, ProQuest 2016). Our review was restricted to newspaper articles published from 1985 to 2013 in the United States. In order for an article to

be included it must have contained the terms ‘feral swine’, ‘feral hog’, or ‘feral pig’, ‘wild swine’, ‘wild hog’, or ‘wild pig’ in the title or lead in to the article. Articles published by the same media source and author on the same date were considered duplicates and removed. The data were summarized generating three annual predictors, the number of articles, the number of different media sources, and the number of states with at least one article.

Each article headline was classified as positive or negative. Our assumption here was that the article headline summarized the overall content, or conclusion of the article. In order to classify articles as having positive or negative tone we used a polarity index described by Rinker (2013) and Breen (2012). In general this polarity algorithm uses a word sentiment (positive or negative) dictionary (Hu and Liu 2004) to tag polarized words in the article headline. A context cluster of six words is extracted from around each polarized word (positive / negative) in the article. The words in this cluster are identified as neutral, negator, amplifier, or de-amplifier. Neutral words hold no value but do affect word count, while each polarized word is counted and weighted in the context cluster. The context clusters for the article headline are summed and divided by the square root of the word count yielding an unbounded score for article describing the negative or positive tone of the headline.

For our purposes we are interested in the cumulative influence of article tone and media sources. In order to produce a measure of this annual cumulative article tone we generated the annual mean tone. This was then multiplied by the number of articles published in the year and by the number of sources creating two predictor variables describing the annual tone for media sources (source tone) and the annual tone for articles (article tone). Classification of newspaper headlines and generation of the media tone indices were done using the qdap qualitative data and

quantitative analysis package (Rinker 2013) within the R computing environment (RCoreTeam 2016).

Agriculture in Wild Swine Affected Regions

To generate a measure of the amount of agriculture present in regions (defined as counties) with wild swine we compiled two data sources. National Agricultural Statistics Service (NASS) data reporting the number of farm operations present in each U.S. County were used as a measure of all farms (USDA 2014). An aggregate of data describing the distribution (presence/absence) of wild swine at the county level was compiled from the Southeast Wildlife Disease Cooperative Study (SCWDS) (SCWDS 2013) and two publications Waithman et al. (1999) and Hanson and Karstad (1959). These data represent the known county level distribution of wild swine over the past 50 years (Figure 2.5). These data were merged with NASS data describing the number of farm operations to generate a national level measure of the proportion of farm operations in counties where wild swine were known to occur.

Formulation of competing models

Co-occurrence of Wild swine and Agriculture Models

Because data describing the distribution of wild swine are not available for all years and represent samples of the known distribution of wild swine over time, models were fit to these data to estimate the national proportional change in the number of farms in counties where wild swine occur for each year from 1959 to 2013. This allowed estimation of the expected proportion of farms co-occurring with wild swine in years without data. We determined relative support in the data for four candidate models - linear, exponential, power, and logistic - to describe the phenomenological change in national wild swine-agriculture co-occurrence. The best approximating model was used to represent the proportion of agriculture in regions with wild swine for each year and was used as a predictor in the policy models.

Policy Models

We evaluated competing models for the relationship between the annual count of policy actions (response) and six variables of interest measuring annually the 1) number of newspaper articles, 2) number of news sources, 3) number of states with newspaper articles, 4) negative tone for news sources 5) negative tone for newspaper articles, 6) and the proportion agriculture in regions with wild swine, here on referred to as agriculture. Specifically, these independent variables represent hypotheses about specific mechanisms that resulted in congressional policy activity that eventually resulted in the establishment of a National program to address the problem.

1. Relative Salience: An increase in the number of newspaper articles, media sources, and the number of states with media reporting would increase the salience of the policy image increasing congressional policy actions.
2. Problem Coalescence: An increase in negative media tone for wild swine represents coalescence of the policy image increasing congressional policy actions.
3. Institutional Pressures: Increasing the amount of agriculture in wild swine regions is related to increasing problem severity and results in increased pressures on Federal government institutions to find a policy solution thus increasing Federal congressional policy activity.

These processes act as surrogates to capture important policy related mechanisms. The increase in the number of negative newspaper articles indicates a change in the policy image (salience and coalescence) and the increase in the amount of agriculture potentially impacted, that is associated with the geographic expansion of wild swine, indicates a change in problem severity

and federal government institutional policy pressure (Elder and Cobb 1983, Jones et al. 2003, Soroka 2003, Sapat 2004, Baumgartner and Jones 2010).

Model selection

We used multi-model inference within an information-theoretic framework, (Burnham and Anderson 2002, Burnham et al. 2011) to estimate model parameters describing the probability of congressional policy actions related to wild swine. All models were fit using a generalized linear model and assumed a Poisson error structure with a log link function. Akaike information criterion with a correction for small sample size (AICc) was used to assess the relative support of the models given the data. We fit all potential subsets of the global model calculating the cumulative AICc weights of evidence as a measure of variable importance, model-averaged regression coefficients and unconditional standard errors (SE) for coefficients (Burnham and Anderson 2002, 2004, Burnham et al. 2011), and 95% confidence intervals (Burnham and Anderson 2002, 2004). To produce unconditional model averaged parameter estimates we used a shrinkage estimation approach (Burnham and Anderson 2002). The explanatory power of the regression coefficients was evaluated using three measures: 1) the weights of evidence (i.e. higher weights indicated greater relative importance); 2) the 95% confidence interval for regression coefficient did not overlap zero; and 3) effect sizes for each regression coefficient. The final inferential model was used to estimate the relative annual contribution of each predictor to policy activity across the 29 years investigated and to estimate the relative contribution of livestock and crop agriculture to annual policy activity for wild swine. Maximum likelihood estimates, confidence intervals on model parameters, and AICc values were obtained using MuMIn Multi-Model Inference package (Barton and Barton 2015) available in R (RCoreTeam 2016).

Model validation

AICc does not represent a goodness-of-fit metric hence we assessed model fit using k-fold cross-validation which contrasts the number of policy actions predicted by the model and the observed frequency of policy actions (Kohavi 1995). Using Huberty's rule (Huberty 1994), we randomly divided the wild swine policy action data among four cross-validation bins. Each possible set of three bins was used to fit a predictive model using multi-model averaging that was then used to predict the fourth withheld bin. This process was repeated for 100 iterations using a new random distribution of data across four cross-validation bins for each iteration. This process avoids the dependence of validation results on a single random allocation of data. To assess the models predictive capacity we calculated the Pearson correlation between predicted number of policy actions and the observed number of policy actions within each bin. The Pearson correlation provides a measure of the linear agreement between predicted and observed policy activity thus providing a measure of model performance. Because validation results can be sensitive to binning method (Boyce et al. 2002), we applied and compared the results using a quantile binning method for 4, 10 and 20 bins. Cross-validation was implemented in R statistical software (RCoreTeam 2016).

RESULTS

Congressional Policy Actions

Our search of policy documents identified 421 documents related to wild swine. Figure 2.6 presents the distribution of these documents by type along with key milestones in the emergence of the wild swine policy area. Evaluation of a random sample of 22 (5%) of these documents to determine if the assumption that documents containing a reference to wild swine were an indicator of wild swine policy activity found that all (100%) were related to wild swine policy. This indicated that our assumption was valid and the policy document frequency data

represented policy activity related to wild swine. Assessment of these documents identified roughly four policy periods of increasing policy activity – no activity, regulatory, hearings, and implementation. The period from 1985 to 1993 showed no observable policy activity. This was followed by a brief period from 1994 to 1998 of changes to the federal register and the code of federal regulations (i.e. regulatory activity). From 1999 to 2007 in addition to regulatory activity, discourse on feral swine began in the form of congressional hearings. The last stage was dominated by policy implementation from 2008 to 2013 which accounted for 63.9% of the total activity and comprised both regulatory and distributive policies. Across all years the policy actions largely represented agricultural related issues (68.8%) and were dominated by concerns associated with livestock agriculture (46.1%). In general there is a rapid increase in activity related to wild swine, starting with the minor regulatory changes, followed by congressional hearings, then policy implementation, and in 2013 the establishment of a new national program to address wild swine damage.

Media Data

We identified 1,016 unique newspaper articles related to wild swine between 1985 and 2013. Figure 2.7 illustrates the media data from 1985 through 2013. As illustrated, the number of newspaper articles, number of media sources and number of states with newspaper articles were relatively constant prior to 1998 with a rapid increase in articles, sources, and states after 1999. This period from 1999 to 2013 accounted for 95.7% of articles and 84.8% of news sources. The number of states with wild swine related media reports continued to increase throughout the study period with 45 states having at least one article.

Analysis of newspaper article tone found that mean tone was close to neutral for both number of media sources (-0.40±1.39) and number of articles (-0.25±1.55) from 1985 to 1998.

Figure 2.7 also presents the change in media tone over time. From 1999 to 2006 polarity became increasingly negative for both the media sources (-3.77±2.43) and number of newspaper articles (-3.77±2.38). During the implementation period from 2007 to 2013 polarity continued to become negative for media sources (-13.14±5.82) and number of newspaper articles (-17.78±4.58).

Co-occurrence of Wild Swine and Agriculture Models

The co-occurrence of wild swine and agricultural operations expanded at an increasing rate from 1959 until 2013. Based on the AICc weights the best approximating model was a logistic model (Table 2.1). For our study period the proportion of farms in wild swine regions increased from 0.17 in 1985 to 0.41 in 2013 (Figure 2.8). This represented an annual rate of increase of 1.01 (stdev <0.01) during this period. The estimated inflection year was 2034 with 69.9% of farms in regions with wild swine. Based on the strong predictive capacity of this distribution (Adjusted $R^2 = 0.99$) it was used as a predictor in the policy models to represent the number of farm operations potentially impacted by wild swine annually which we use as a surrogate for changes in institutional policy pressure.

Policy Models

Based on the final inferential model policy activity was most strongly associated with the number of states with newspaper articles, polarity of media sources, polarity of newspaper articles, and the proportion of agriculture in wild swine regions (Table 3). Covariates representing each of these factors had high AICc weights of evidence and 95% confidence intervals that did not include zero indicating high predictive importance. Cross-validation indicated that our final model had strong predictive capacity. The quantile binning method produced similar Pearson correlations of 0.969 (4 bins), 0.915 (10 bins) and 0.957 (20 bins)

between median predicted policy actions and the observed policy actions in each bin, indicating low sensitivity

Parameter estimates and odds ratios for the parameters considered are shown of the cross-validation results to binning method in Table 2.3. The number of states with wild swine related newspaper articles was a positive predictor of wild swine policy activity (odds ratio = 2.08). For every additional 5 states with newspaper headlines related to wild swine there was a 3.65% increase in the number of policy actions. Increasing negative tone of both number of newspaper articles (odds ratio = 1.95) and number of media sources (odds ratio = 1.14), increased in the number of policy actions. That is for every 10 negative newspaper articles and 10 additional negative media sources wild swine policy activity increased by 6.7% and 1.3%. The proportion of agriculture in regions with wild swine was the most significant predictor of policy actions (odds ratio = 4.09); that is for every 1% increase in the proportion of agriculture in regions with wild swine policy activity increases by 41%. This increase appeared most sensitive to livestock related policy activity. Livestock policy activity (41%) increased at nearly twice the rate of crop policy activity (23%) for every 1% increase in the proportion of agriculture in wild swine regions and was also a significant predictor of livestock (odds ratio = 4.08) and crop (odds ratio = 3.43) specific policy activity for wild swine. Figure 2.9. illustrates the functional relationship between increasing agriculture in wild swine regions and the resulting change in all wild swine policy activity and policy activity specific to livestock and crop agriculture.

The contribution of the four most important predictors, number of states with newspaper articles, polarity of media sources, polarity of newspaper articles, and national proportion of agriculture in wild swine regions, to policy activity changed across the 29 years evaluated (Figure 2.10). The annual contribution of the proportion of agriculture in wild swine regions to

policy activity varied the most, with a 54.9% change from 5.5% of policy activity in 1985 to 60.7% of policy activity in 2012. Both media source and newspaper article tone had declining annual contribution to policy activity, declining 37.5% and 17.2%. Combined media source and newspaper article polarity contributed to 30.5% of policy activity in 2013 compared to a combined 71.7% in 1985. The number of states with newspaper articles contributed a consistent amount annually (mean=22.8%; 95% CI = 21.1%-124.5%) to policy activity across all years.

DISCUSSION

Our models found a linkage between policy activity and four predictors representing number of states with media, media tone and agriculture. These predictors have been described in previous studies as representing specific policy processes associated with policy image salience (number of states with news articles) (Gilliam Jr and Iyengar 2000, Schnell 2001, Soroka 2003, Walgrave et al. 2008, Baumgartner and Jones 2010), policy image coalescence (newspaper article and source tone) (Elder and Cobb 1983, Weart 1988, Baumgartner and Jones 2010) and institutional pressures (feral swine-agriculture co-occurrence) (Kingdon and Thurber 1984, Sapat 2004). Further we found the contribution of these predictors to policy activity changed across the 29 years analyzed indicating the development of federal feral swine policy went through a continuum of policy development. Understanding how these predictors that serve as proxy measures of policy stages contribute to policy development can provide a better understanding of important latent processes that give rise to national policies to address wildlife problems. This in turn can support the development of programs and policies that best address the social issues. Here we provide a discussion of these predictors and their potential contribution to the latent policy process that may link the observed policy activity with social and institutional processes.

Our model suggests that for wild swine policy, increasing negative newspaper articles may have acted as a mechanism for influencing initial policy activity. This may have been particularly important for issue emergence and salience as media related predictors contributed most during early phases of policy development. In addition our results suggest that increasing the amount of agriculture in wild swine regions influenced policy activity the most, particularly during later policy phases of issue coalescence and policy implementation. Media predictors for newspaper article and media source tone became less important once policy implementation began, indicating that the introduction of potential policy solutions by governmental institutions may have increased stability in the policy area (Jones et al. 2003, Baumgartner and Jones 2010). The emergence of wild swine as a policy issue was characterized by decades of general inattention and no observed policy development (see Figure 2.6). The lack of policy attention and media coverage that was neutral indicates that wild swine was not a broad issue prioritized by society nor did the issue have a distinct policy image prior to 1994. This lack of policy image may have contributed to policy inattention despite recognition in the scientific literature that wild swine were damaging to both the environment and agriculture (Hanson and Karstad 1959, Wood and Barrett 1979). Policy images have implication for which interest groups and governmental institutions become involved, how an issue is understood, and which institutional venue an issue will be addressed by government (Baumgartner and Jones 1991). The scope of participation by stakeholders and the clarity of a policy image is central to defining the policy agenda and potential policy solutions (Schattschneider 1960). However, public perception concerning an issue is also important in prioritizing the policy agenda (Baumgartner and Jones 2010). In the case of wild swine without a distinct policy image prior to 1999 the primary agricultural interests – livestock and crop agriculture – may not have been able to pursue a policy

solution at the national level and as a result did not contribute significantly to policy activity prior to 2005. Furthermore, the lack of public attention and limited access by the primary interests may have limited the issue from being considered on the policy agenda.

National policy issues of wild swine appear to have emerged sometime after 1994 when the first policy activity for wild swine occurred. The five years from 1994 to 1998 had a nearly six fold increase in news coverage. However the news coverage tone during this period was not significantly different than neutral, indicating that the general public was aware of the emerging issue but there was no consensus, and the issue had not yet become salient. The lack of salience during this time may also be evidenced by the continued importance of news predictors relative to proportion agriculture in regions with swine (Figure 2.10). Previous studies (Jones and Baumgartner 2004, Baumgartner and Jones 2010) have proposed that increasing news media, specifically negative news media, indicates public policy image coalescence and policy issue salience. Prior to 2005 the relative contribution of agriculture to policy activity was less than media predictors indicating a lack of issue salience.

As news media became increasingly negative there was a rapid increase in total policy activity and the relative contribution of agricultural interests to policy activity increased. This rapid change may indicate that the issue became broadly salient and the policy image coalesced around this period. Salience of social issues in public discourse may determine whether or not issues expand on the government agenda (Koch-Baumgarten and Voltmer 2010). For example issue salience can determine voter turnout and choice preferences (Becker 1977). In our analysis news media may have provided a method for establishing issue salience and coalescence, serving to bring the issue to the governmental agenda. In addition, the number of states with negative media was important in all models. However, the relative contribution to policy activity was

nearly the same across all years. This may indicate that the geographic distribution of media was important across the entire continuum of policy development but was not related to any one phase of the policy process. Previous authors have proposed that news media can serve as an agenda-setting mechanism (Scheufele and Tewksbury 2007).

Social problems that receive media attention do not automatically receive attention on the policy agenda or generate policy actions. The policy image - how a problem and its solution is defined, understood, and discussed – must be established (Baumgartner and Jones 2010). In the case of wild swine, the policy image does not appear to have begun to become broadly salient and coalesce until the 2000s when media tone became significantly negative. The first congressional hearing addressing wild swine was conducted in 1999 and addressed issues related to U.S. Department of Agriculture's (USDA) policy for addressing wildlife transmission of diseases to domestic livestock and specifically brucellosis in wild swine (Senate 1999). This has been identified as a potentially significant issue facing agriculture and wildlife management (Miller et al. 2013). However congressional hearings did not begin in earnest until 2005 and 2006 when ten hearings were held – over double from the previous five years. Hearings in these two years were largely related to potential animal agricultural impacts associated with classical swine fever, a swine disease with international trade implications for the U.S. swine industry (Paarlberg et al. 2009).

Once the issue of feral swine was on the policy agenda, we found that the presence of agriculture in wild swine regions, a proxy for institutional pressures on policy makers, had a greater influence on the overall frequency of policy activity (Figure 2.10). The relative contribution to policy activity shifted from primarily media related to primarily agricultural interests related sometime after 2005. This indicates that agricultural interests in wild swine

regions may have been the primary driver in bringing wild swine to the institutional agenda. In addition the focus of wild swine related policy activity on agricultural damage related issues indicates that agricultural interests had influence in setting the agenda. Further, livestock agricultural interests appear to have dominated the problem definition period, contributing to as much as 80% of all policy activity prior to 2005 (see top panel in Figure 2.7). Previous studies have proposed that actors which are able to define the problem early in the issue emergence stage tend to control future policy development even if new actors inter the policy arena (Schattschneider 1960).

A policy image serves to link the problem with the governmental solution (Baumgartner and Jones 2010). The coalescence of a policy image of wild swine negatively impacting agriculture was succeeded by a flurry of policy activity. Between 2007 and 2013 there were an average of 38 wild swine related policy actions a year with 67% of these directly related to implementing policy, rulemaking, or appropriations. This included the Feral Swine Eradication and Control Pilot Program Acts of 2009 and 2011 that authorized the secretary of the Department of Interior (DOI) to provide financial assistance to specific states for eradication efforts (Landrieu and Vitter 2009, Landrieu 2011). This was followed by the establishment of the USDA Animal Plant Health Inspection Service (APHIS) National Feral Swine Damage Management Program in 2013 (USDA 2013). The focus of policy activity during this policy implementation stage remained dominated by agriculture (69.1%), specifically livestock disease related concerns (46.1%). Crop agricultural concerns remained much lower with only 23% of activity related to crop damage. This period also represented the greatest number of negative newspaper articles, which occurred at a rate of 107 per year in 27 states. The proportion of agriculture in regions with wild swine continued to increase with an estimated 38% of farms in

the United States potentially impacted. Our model indicates that both media and livestock agriculture were predictive of increased policy activity.

Increasing, problem severity and the resulting institutional pressures by agricultural interests to address agricultural damage likely contributed most to the development of policy. This is evidenced by the proportion of agriculture in regions with wild swine being the most significant predictor of wild swine policy actions in our model (odds ratio = 4.09). Furthermore, agricultural interests accounted for over two-thirds of all wild swine related policy activity and nearly dominated policy activity (~95%) during the issue emergence stage. Our analysis also suggested the emergence of a policy subsystem with relatively few actors dominated by agricultural interests and particularly livestock agriculture (Heichel 1990). Livestock agriculture had nearly twice the influence on wild swine policy activity and likely contributed most to forming the policy image supporting the notion of a policy subsystem. The interaction of beliefs and values concerning a particular policy with the existing set of institutions, in this case USDA and DOI, acted as the venues of policy action (Baumgartner and Jones 1991). This often results in policy subsystems that are oriented towards a given industry (Baumgartner and Jones 2002a). Furthermore interactions of venue and image can produce self-reinforcing system characterized by a positive feedback that tends to create stability over time (Baumgartner and Jones 2002b). In the case of wild swine this may represent a relatively stable policy subsystem devoted to wild swine control and agricultural damage mitigation.

This study is based on a large search of government documents and media data; therefore there are inherent constraints on inference. While our objective was to investigate the relative contribution of media and institutional pressures on national wildlife-agricultural policy development, there are other potential drivers of policy activity. Previous studies have found

that interest group access to congressional committees and advisory committees are influential in the development of policy (Balla and Wright 2001), although this is also influenced by the number of stakeholders in a policy area (Baumgartner et al. 2009). In our study we only considered three actors – livestock agriculture, crop agriculture, and the public – although there may have been additional actors that contributed to the generation of national policy. We also did not consider other potential processes that may have influenced national policy activity such as policy diffusion (Berry and Berry 1999) or policy entrepreneurs (Mintrom and Norman 2009). These policy processes may also have contributed to the observed policy activity. While our study provides insights into drivers of policy activity addressing the wildlife-livestock interface, it could be enhanced by investigating these other mechanisms that may also be important in creation of policy.

News media coverage has been identified in previous studies to influence policy development (Gilliam Jr and Iyengar 2000, Schnell 2001, Soroka 2003, Walgrave et al. 2008, Baumgartner and Jones 2010). However, we found that institutional pressures applied by actors may have a far greater contribution to the development of policy. This may be particularly important when economic impacts are a result of the issue. This effect maybe even greater early in the emergence of an issue when fewer actors are involved and the ability for actors to define the problem and the eventual policy image is greater (Schattschneider 1960). Furthermore, among potential interests involved during this stage, those with the greatest potential risk for damage may have the greatest impact on the formation of policy and in the case of wild swine this appears to be livestock agriculture (Baumgartner et al. 2009).

This is the first analysis we are aware of that examines the role of public sentiment and institutional pressures on the development of policies that address the wildlife-livestock

interface. In the case of wild swine, our model suggests that changes in co-occurrence of wild swine and agriculture over the last 29 years, resulting in increased problem severity (increased agricultural damage), likely contributed most to the eventual development of policy to mitigate the issue. This likely resulted from increasing industry pressure on agricultural agencies to protect or mitigate damage associated with wild swine. This was evidenced by our model results and also the significant consideration given to agricultural damage caused by wild swine during congressional hearings and in congressional reports (GPO 2001, 2013). Congressional hearings and reports often focused on specific mechanisms, rules, or procedures the USDA had in place to mitigate livestock and crop damage caused by wild swine (GPO 2013). In addition the livestock agricultural sector likely contributed more to the development of policy with nearly half of policy addressing livestock related issues, particularly the potential for disease outbreaks. This may be driven by the potential for large economic losses – USD$5.8 billion - associated with a single livestock-wild swine disease outbreak (foot and mouth disease) compared with the currently estimated USD$800 million in damage caused by wild swine to crop agriculture (Paarlberg et al. 2002, Pimentel et al. 2002).

Given the scarcity of rigorous quantitative policy work in this system – wildlife-agriculture interface, specifically wild swine – greater attention is needed to disentangle the mechanisms driving policy development. Although work has been conducted examining the influence of public sentiment and news tone influence on policy development (Baumgartner and Jones 2010), there remains a lack of information linking measures of public perception and institutional pressures specifically for the wildlife-agricultural interface. Such information could provide valuable insight into the observed variability in policy approaches addressing wildlife-agriculture interactions. Unravelling the role of news media coverage, public knowledge, public

sentiment, agricultural impacts, and environmental damage—as it relates to changes and differences in policy approaches at local and national levels—will require large-scale studies on local and national policy drivers which have not been attempted to date for the wildlife-livestock interface; but could provide valuable information for improving policy systems for control and mitigation of damage associated with wild swine and wildlife in the United States. Policy makers can in turn use analyses such as this to better design policies that align with public interests and benefactors ensuring long term success of policies by incorporating all interests (Loomis and Helfand 2001).

Table 2.1. Candidate models use to estimate the county scale co-occurrence of wild swine and agricultural that was used as a predictor in the policy models. The best approximating model in the candidate set of models given the available data was a logistic model and had good predictive capacity with an adjusted $R^2 = 0.99$.

Candidate Models	df	AICc	Δ AICc	Aikaike Weight	Adjusted R^2
Logistic	7	-58.04	0.00	$7.63x10^{1}$	0.99
Linear	7	-36.31	21.73	$1.46x10^{5}$	0.92
Exponential	7	-27.74	30.30	$2.01x10^{7}$	0.99
Power	7	-27.16	30.88	$1.51x10^{7}$	0.99

Table 2.2. Candidate set of models used in the model averaging procedure to generate the final inferential model. These eight models account for 99.9% of the AICc weight given the candidate set of 64 models. The null model (intercept only) was ranked as the least informative model and the top model was 485 AICc units better (i.e. lower) than the null model suggesting the selected covariates approximated the data well.

Model						K	Log Likelihood	AICc	Δ AICc	AICc Weight
Agriculture			# States	Article Tone	Source Tone	4	-70.30	153.21	0.00	0.313
Agriculture	# Articles	# Sources	# States	Article Tone		5	-69.04	153.91	0.70	0.221
Agriculture	# Articles		# States	Article Tone	Source Tone	5	-69.14	154.10	0.89	0.201
Agriculture		# Sources	# States	Article Tone	Source Tone	5	-69.57	154.95	1.74	0.131
Agriculture	# Articles	# Sources	# States	Article Tone	Source Tone	6	-68.28	155.89	2.68	0.082
Agriculture			# States	Article Tone		3	-74.47	158.61	5.40	0.021
Agriculture	# Articles	# Sources	# States			4	-73.11	158.82	5.61	0.019
Agriculture	# Articles		# States	Article Tone		4	-73.58	159.76	6.55	0.012

Table 2.3. Parameter estimates for the final inferential model describing the relationship between the number of policy actions, newspaper headlines, and proportion of agricultural operations in wild swine regions. This table includes the model-averaged parameter estimates, odds ratio, unconditional standard errors, unconditional 95% confidence intervals (CIs), and cumulative Akaike's Information Criterion weights for all covariates used to model the number of policy actions for wild swine.

Parameter	Odds Ratio	Estimate	Unconditional Standard Error	2.5%	97.5%	AICc Weight
All Agriculture	4.09	1.41	0.25	1.32	1.50	1
Livestock	4.08	1.41	0.34	1.32	1.49	1
Crop	3.43	1.23	0.53	0.96	1.51	0.92
# States with Newspaper Articles	2.08	0.73	0.17	0.69	0.78	1
Newspaper Article Polarity	1.95	0.67	0.23	0.59	0.75	0.98
Media Source Polarity	1.14	0.13	0.08	0.11	0.15	0.73
# Newspaper Articles	1.98	0.68	1.04	-0.88	2.25	0.53
# Media Sources	0.53	-0.64	1.36	-2.72	1.44	0.46

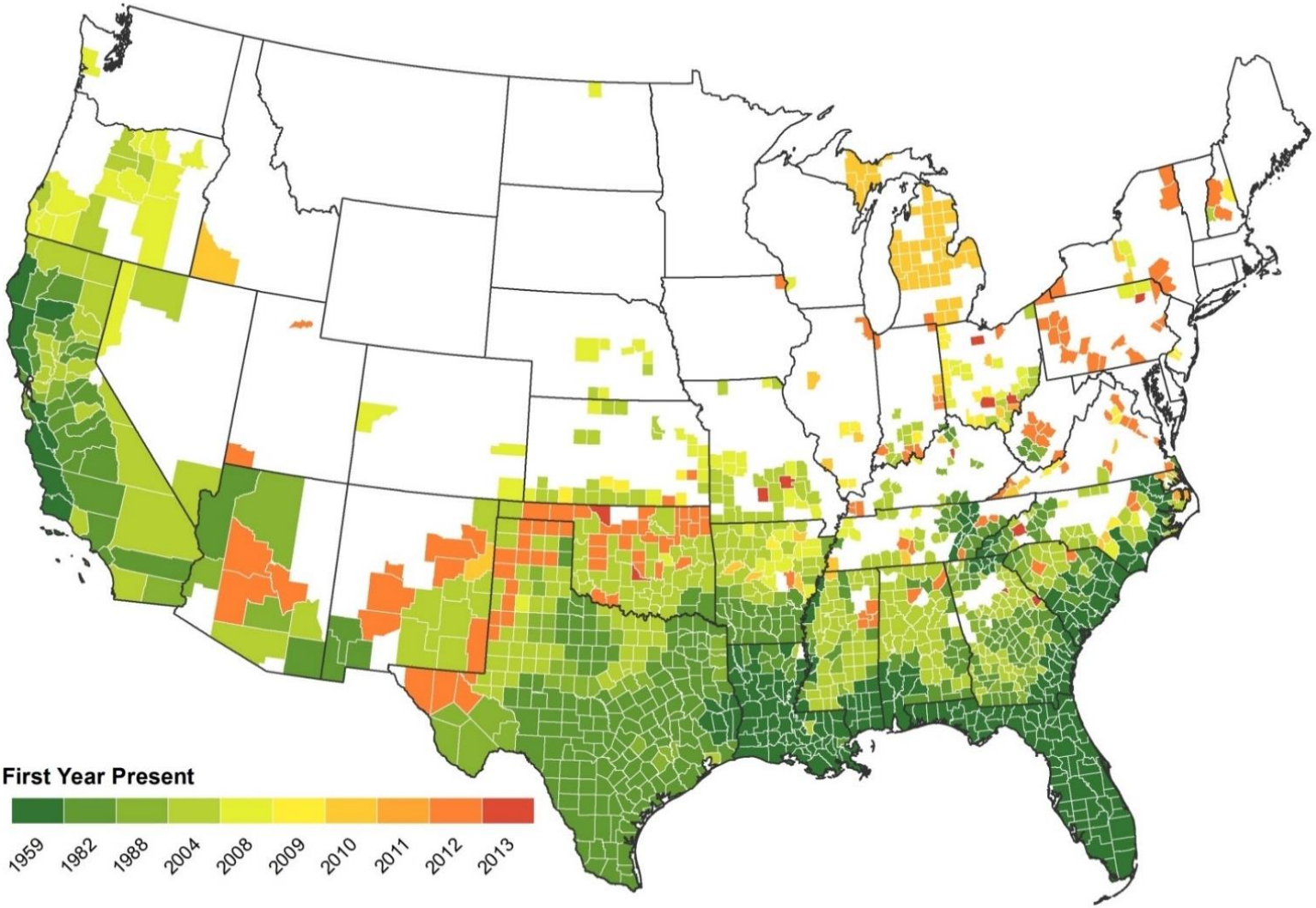


Figure 2.5 Current known county level distribution of wild swine in the United States. Years indicate the first year wild swine were identified within the county.

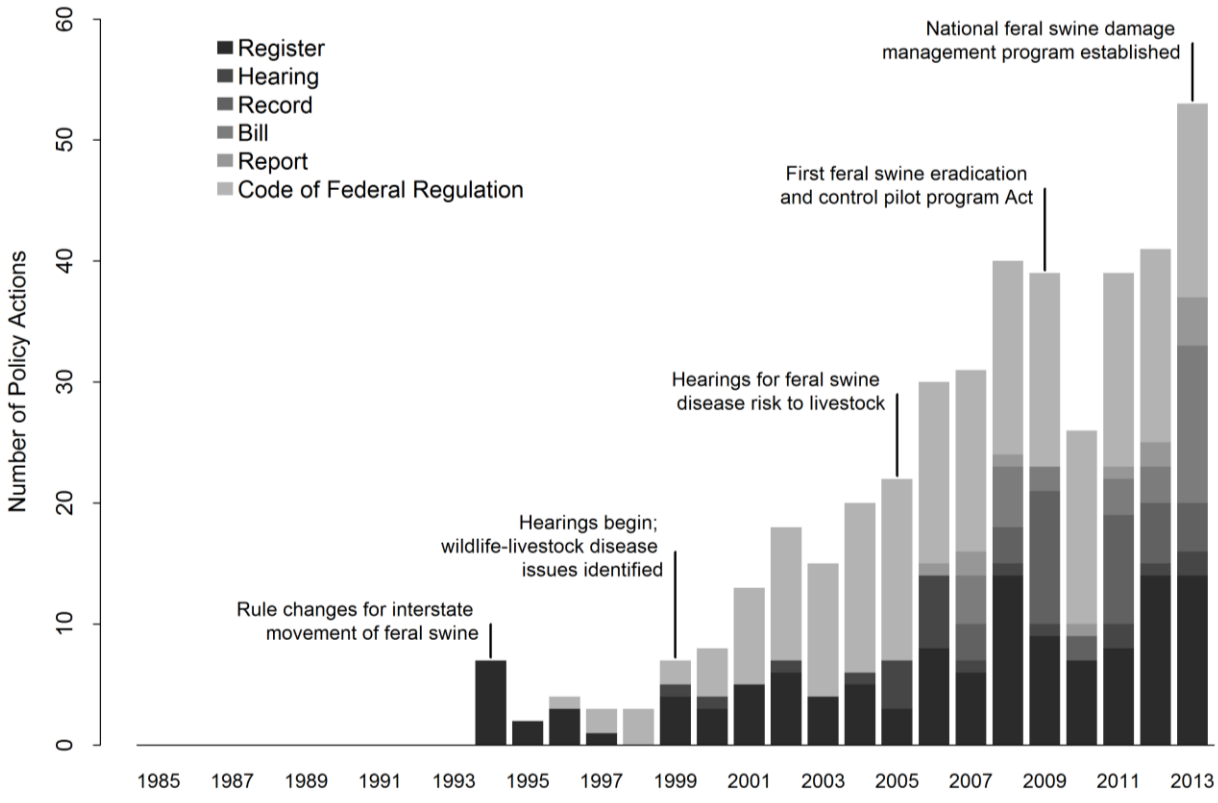


Figure 2.6. Policy activity and major policy milestones for wild swine from 1985 through 2013.

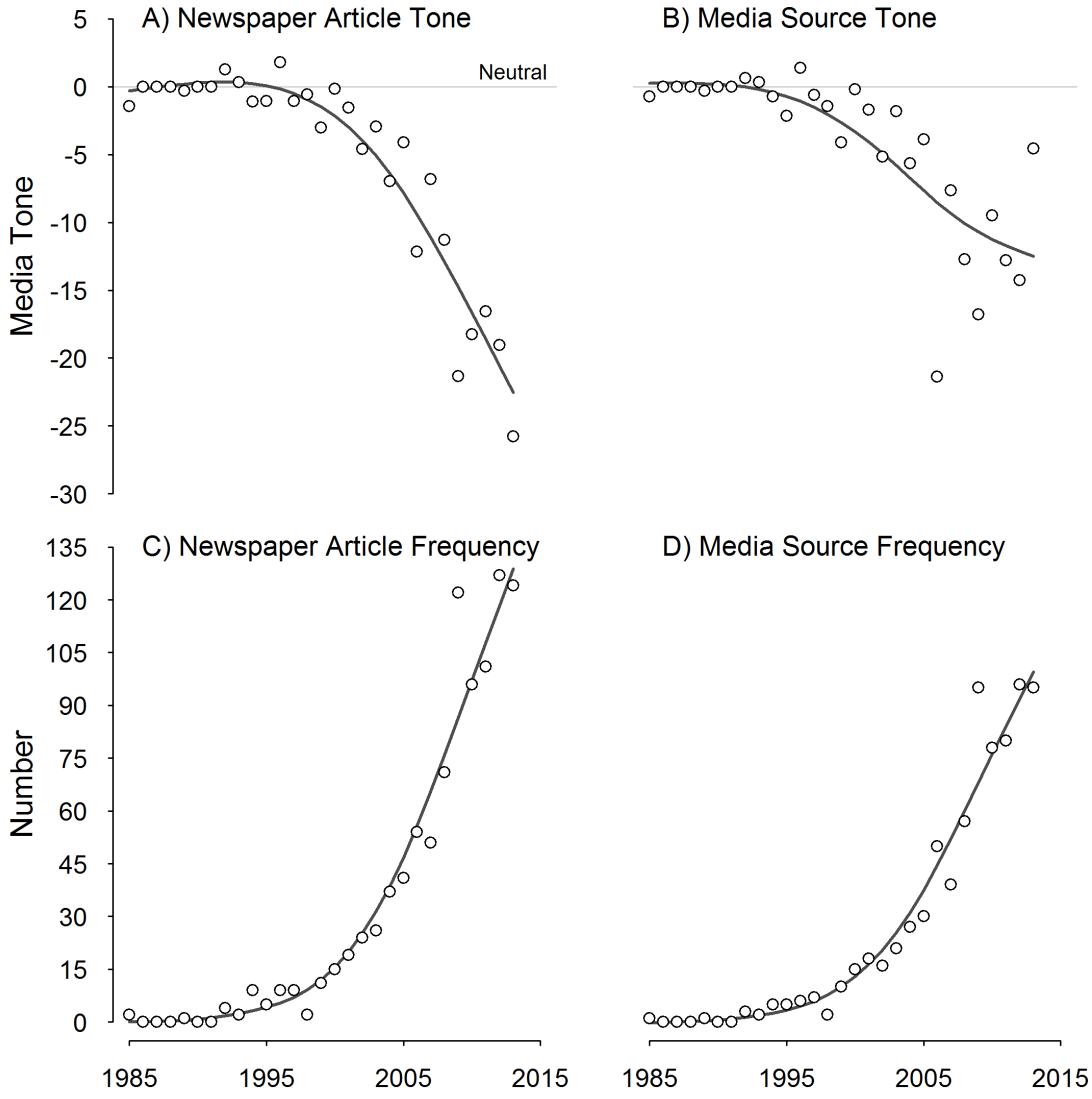


Figure 2.7. Media data used as predictors in the policy models. Panels represent annual mean media tone (A and B) and annual media frequency (C and D). Vertical axis on upper panels indicates media tone. Gray horizontal line represents neutral media tone while positive values indicate positive tone and negative values indicate negative tone. Trend line is a spline fit to the observed data.

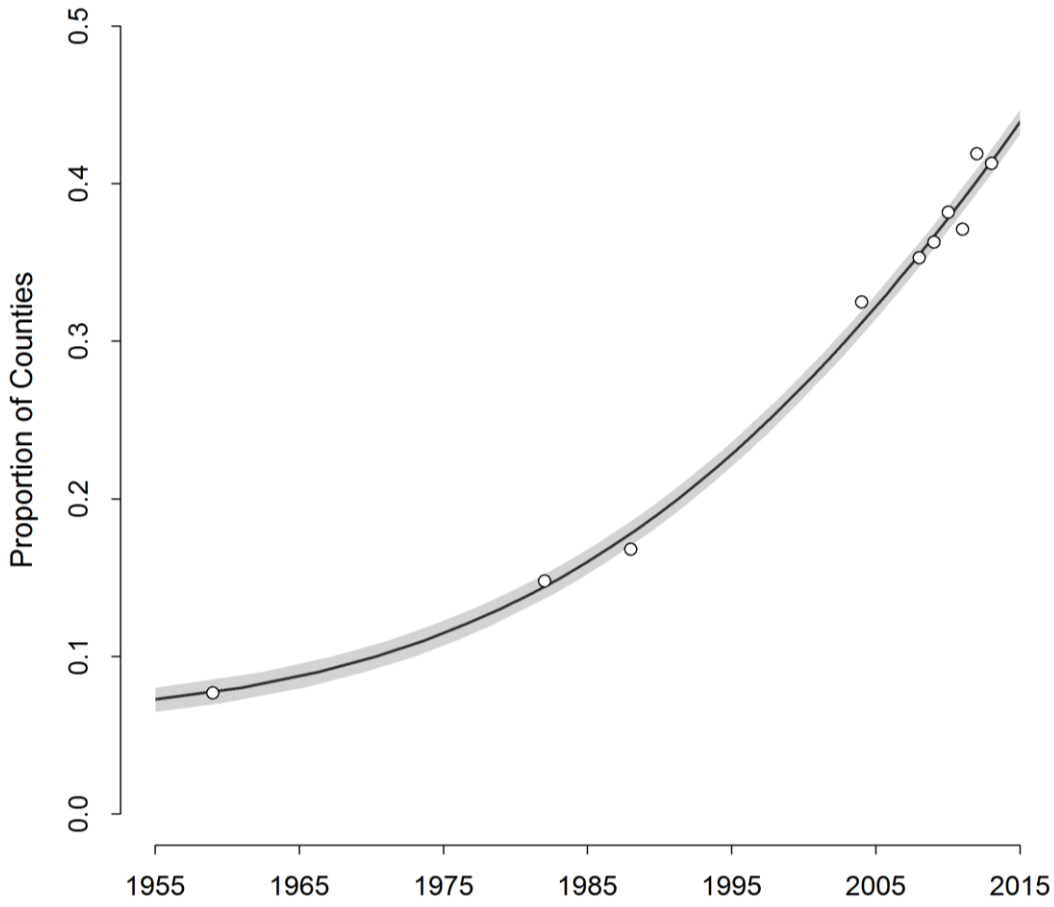


Figure 2.8. Logistic model used to estimate the county level co-occurrence of wild swine and agricultural operations in the United States. Solid line indicates model estimated mean and gray band is the 95% confidence interval. The annual rate of increase was estimated as 1.01 (stdev <0.01) from 1959 to 2013 with the estimated inflection year being 2034 with 69.9% of agricultural operations located in regions with wild swine. The model had good predictive capacity having an adjusted $R^2 = 0.99$.

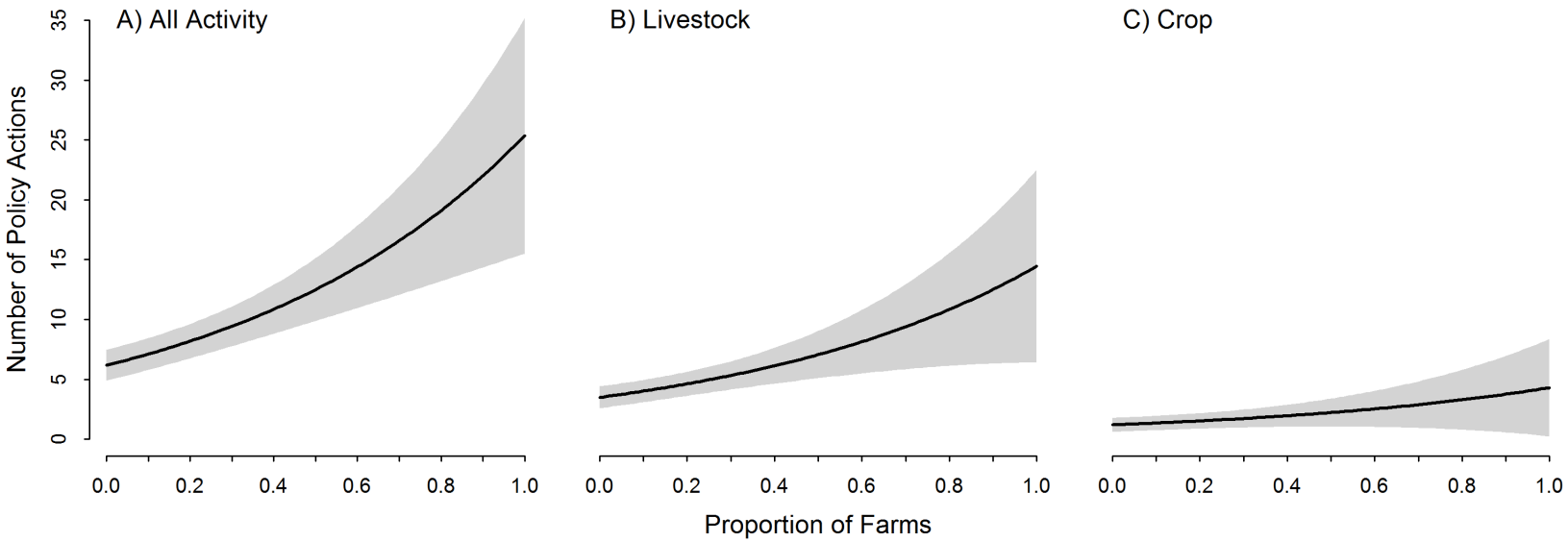


Figure 2.9. Mean functional relationship between wild swine congressional policy activity and the national proportion of agriculture co-occurring wild swine in the United States. Solid black line indicates predicted mean relationship and gray band indicates unconditional 95% confidence interval. Panels represent the functional relationship for (A) all wild swine policy activity, (B) wild swine policy activity specific to livestock agriculture, and (C) wild swine policy activity specific to crop agriculture (C).

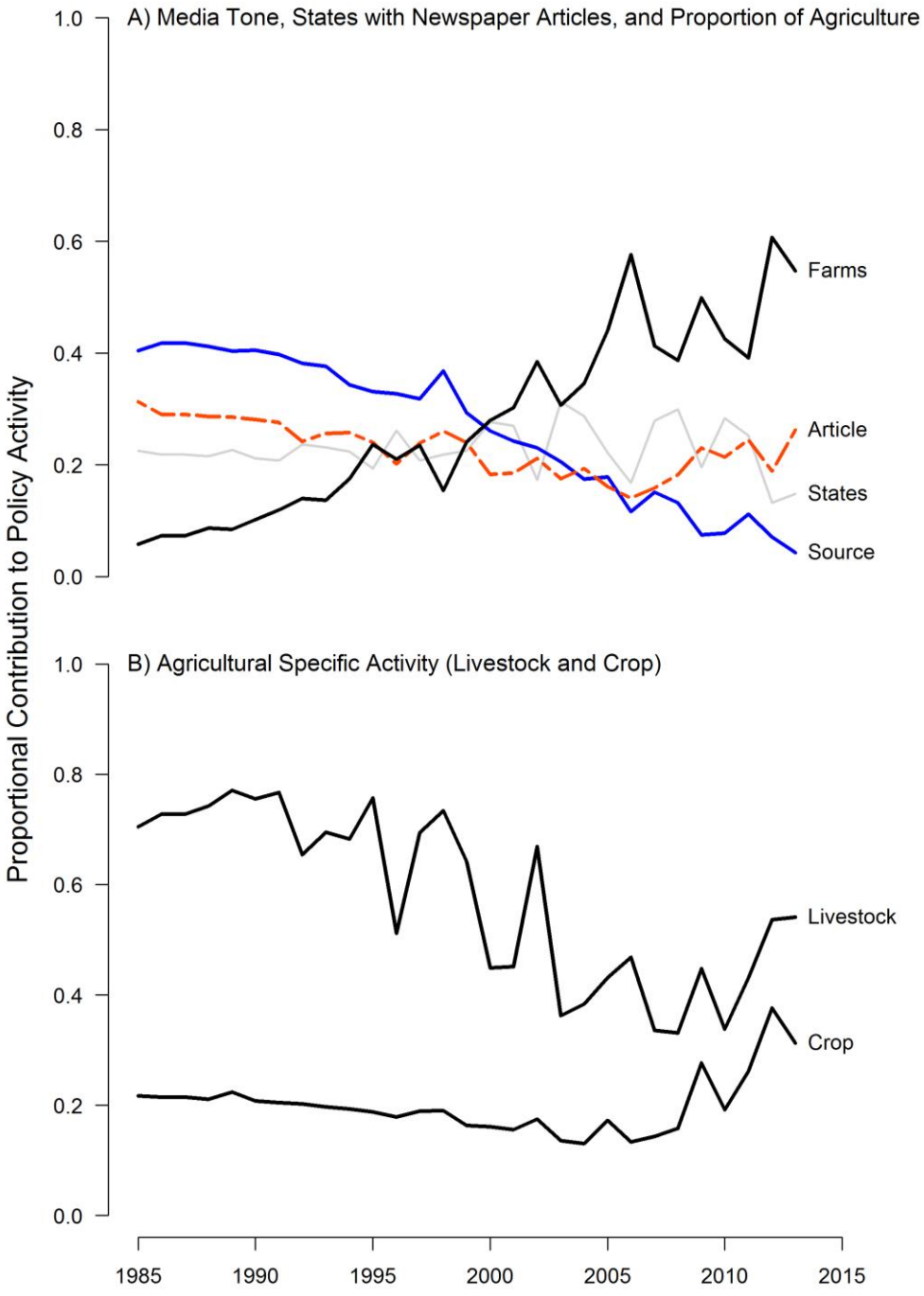


Figure 2.10. The model predicted change in relative contribution of predictors to annual policy activity for wild swine. Panel A contrasts the changes in the annual contribution of media tone, number of states with newspaper articles, and the proportion of agriculture in wild swine regions. Panel shows shift in contribution sources to total policy activity between 2000 and 2005. Panel B describes the relative contribution of livestock and crop specific activity to overall wild swine policy excluding media tone and number of states with newspaper articles.

REFERENCES

Balla, S. J., and J. R. Wright. 2001. Interest groups, advisory committees, and congressional control of the bureaucracy. American Journal of Political Science:799-812.

Barton, K., and M. K. Barton. 2015. Package 'MuMIn'. Version **1**:18.

Baumgartner, F. R., J. M. Berry, M. Hojnacki, B. L. Leech, and D. C. Kimball. 2009. Lobbying and policy change: Who wins, who loses, and why. University of Chicago Press.

Baumgartner, F. R., and B. D. Jones. 1991. Agenda dynamics and policy subsystems. The journal of Politics **53**:1044-1074.

Baumgartner, F. R., and B. D. Jones. 2002a. Policy dynamics. University of Chicago Press.

Baumgartner, F. R., and B. D. Jones. 2002b. Positive and negative feedback in politics. Policy dynamics:3-28.

Baumgartner, F. R., and B. D. Jones. 2010. Agendas and instability in American politics. University of Chicago Press.

Becker, L. B. 1977. The Impact of Issue Salience.*in* D. L. Shaw and M. E. McCombs, editors. The Emergence of American Political Issues: The Agenda Setting Function of the Press. West Publishing, Saint Paul, MN.

Berry, F. S., and W. D. Berry. 1999. Innovation and diffusion models in policy research. Theories of the policy process **169**.

Bevins, S. N., K. Pedersen, M. W. Lutman, T. Gidlewski, and T. J. Deliberto. 2014. Consequences Associated with the Recent Range Expansion of Nonnative Feral Swine. BioScience **64**:291-299.

Boyce, M. S., P. R. Vernier, S. E. Nielsen, and F. K. Schmiegelow. 2002. Evaluating resource selection functions. Ecological modelling **157**:281-300.

Breen, J. O. 2012. Mining twitter for airline consumer sentiment. Practical Text Mining and Statistical Analysis for Non-structured Text Data Applications:133.

Brook, R. K., and F. M. v. Beest. 2014. Feral wild boar distribution and perceptions of risk on the central Canadian prairies. Wildlife Society Bulletin.

Burnham, K. P., and D. R. Anderson. 2002. Model selection and multimodel inference: a practical information-theoretic approach. Springer.

Burnham, K. P., and D. R. Anderson. 2004. Multimodel inference understanding AIC and BIC in model selection. Sociological methods & research **33**:261-304.

Burnham, K. P., D. R. Anderson, and K. P. Huyvaert. 2011. AIC model selection and multimodel inference in behavioral ecology: some background, observations, and comparisons. Behavioral Ecology and Sociobiology **65**:23-35.

EBSCO. 2016. EBSCO Newspaper Source Database.

Elder, R. W., and C. D. Cobb. 1983. Participation in American politics: The dynamics of agenda-building. Baltimore: John Hopkins University Press.

Entman, R. M. 1993. Framing: Towards clarification of a fractured paradigm. McQuail's reader in mass communication theory:390-397.

Frederick, J. 1998. Overview of wild pig damage in California.*in* Proceedings-Vertebrate Pest Conference.

Funkhouser, G., and E. F. Shaw. 1990. How synthetic experience shapes social reality. Journal of communication **40**:75-87.

Gilliam Jr, F. D., and S. Iyengar. 2000. Prime suspects: The influence of local television news on the viewing public. American Journal of Political Science:560-573.

GPO. 2001. Agriculture, Rural Development, Food And Drug Administration, And Related Agencies Appropriations for 2001. Page 461 Subcommittee on Agriculture, Rural Development, Food and Drug Administration, and Related Agencies. U.S. Government Printing Office.

GPO. 2013. Agriculture, Rural Development, Food And Drug Administration, And Related Agencies Appropriations for 2014. Page 487 Subcommittee on Agriculture, Rural Development, Food and Drug Administration, and Related Agencies. U.S. Government Printing Office.

GPO. 2014. Federal Digital System (FDsys). United States Government Printing Office.

Hanson, R., and L. Karstad. 1959. Feral swine in the southeastern United States. The Journal of wildlife management:64-74.

Heichel, G. 1990. Communicating the agricultural research agenda: Implications for policy. Journal of Production Agriculture **3**:20-24.

Hu, M., and B. Liu. 2004. Mining and summarizing customer reviews. Pages 168-177 *in* Proceedings of the tenth ACM SIGKDD international conference on Knowledge discovery and data mining. ACM.

Huberty, C. J. 1994. Applied discriminant analysis. Wiley New York.

Izac, A., and P. O'Brien. 1991. Conflict, uncertainty and risk in feral pig management Australian approach. Journal of Environmental Management **32**:1-18.

Jones, B. A., D. Grace, R. Kock, S. Alonso, J. Rushton, M. Y. Said, D. McKeever, F. Mutua, J. Young, and J. McDermott. 2013. Zoonosis emergence linked to agricultural

intensification and environmental change. Proceedings of the National Academy of Sciences **110**:8399-8404.

Jones, B. D., and F. R. Baumgartner. 2004. Representation and agenda setting. Policy Studies Journal **32**:1-24.

Jones, B. D., T. Sulkin, and H. A. Larsen. 2003. Policy punctuations in American political institutions. American Political Science Review **97**:151-169.

Kingdon, J. W., and J. A. Thurber. 1984. Agendas, alternatives, and public policies. Little, Brown Boston.

Koch-Baumgarten, S., and K. Voltmer. 2010. Public Policy and the Mass Media: The Interplay of Mass Communication and Political Decision Making. Routledge.

Kohavi, R. 1995. A study of cross-validation and bootstrap for accuracy estimation and model selection. Pages 1137-1145 *in* Ijcai.

Krebs, J., R. Anderson, T. Clutton-Brock, C. Donnelly, S. Frost, W. Morrison, R. Woodroffe, and D. Young. 1998. Badgers and bovine TB: conflicts between conservation and health. Science **279**:817-818.

Landrieu, M. L. 2011. Feral swine eradication and control pilot program act of 2011. 16 U.S.C. 3951.

Landrieu, M. L., and D. Vitter. 2009. Feral swine eradication and control pilot program act of 2009. 16 U.S.C. 3951.

LexisNexis. 2016. LexisNexis Academic Database.

Loomis, J., and G. E. Helfand. 2001. Environmental policy analysis for decision making. Kluwer Academic Amsterdam,, Netherlands.

Mayer, J., and I. Brisbin. 1991. Wild pigs in the United States: their life history, morphology and current status. University of Georgia Press. Athens, Georgia, USA.

McCombs, M. E., and D. L. Shaw. 1972. The agenda-setting function of mass media. Public opinion quarterly:176-187.

McKnight, T. L. 1964. Feral livestock in Anglo-America. University of California Press.

Mengak, M. T. 2012. Georgia Wild Pig Survey: final report. Warnell School of Forestry and Natural Resources, University of Georgia.

Messmer, T. A. 2009. Human–wildlife conflicts: emerging challenges and opportunities.

Miller, R. S., M. L. Farnsworth, and J. L. Malmberg. 2013. Diseases at the livestock–wildlife interface: Status, challenges, and opportunities in the United States. Preventive veterinary medicine **110**:119-132.

Miller, R. S., and S. J. Sweeney. 2013. Mycobacterium bovis (bovine tuberculosis) infection in North American wildlife: current status and opportunities for mitigation of risks of further infection in wildlife populations. Epidemiology and infection **141**:1357-1370.

Mintrom, M., and P. Norman. 2009. Policy entrepreneurship and policy change. Policy Studies Journal **37**:649-667.

NewsBank. 2016. NewsBank NewsFile Collection. NewsBank.

Paarlberg, P. L., J. G. Lee, and A. H. Seitzinger. 2002. Potential revenue impact of an outbreak of foot-and-mouth disease in the United States. Journal of the American Veterinary Medical Association **220**:988-992.

Paarlberg, P. L., A. H. Seitzinger, J. G. Lee, and K. Mathews. 2009. Supply reductions, export restrictions, and expectations for hog returns in a potential classical swine fever outbreak in the United States. Journal of swine health and production **17**:155-162.

Pimental, D. 2007. Environmental and economic costs of vertebrate species invasions into the United States. Managing Vertebrate Invasive Species.

Pimentel, D., L. Lach, R. Zuniga, and D. Morrison. 2002. Environmental and economic costs associated with non-indigenous species in the United States. Biological Invasions. CRC, Boca Raton:285-303.

ProQuest. 2016. ProQuest Newspaper Database.

RCoreTeam. 2016. R: A language and environment for statistical computing. R foundation for Statistical Computing, Vienna, Austria.

Rinker, T. W. 2013. qdap: Quantitative Discourse Analysis Package. version 1.3.6., University at Buffalo. Buffalo, New York.

Sapat, A. 2004. Devolution and innovation: The adoption of state environmental policy innovations by administrative agencies. Public Administration Review **64**:141-151.

Schattschneider, E. E. 1960. The Semisovereign People: A Realist's View of Democracy in America. Holt, Rinehart, and Winston, New York.

Scheufele, D. A., and D. Tewksbury. 2007. Framing, agenda setting, and priming: The evolution of three media effects models. Journal of communication **57**:9-20.

Schnell, K. C., Frauke. 2001. Assessing the democratic debate: How the news media frame elite policy discourse. Political communication **18**:183-213.

SCWDS. 2013. National Feral Swine Distribution Maps.*in* N. F. S. M. System, editor. Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA.

Senate. 1999. Agriculture, rural development, and related agencies appropriations for fiscal year 2000. Page 1433 *in* U. S. Senate, editor. Subcommittee of the Committee on Appropriations United States Senate. U.S. Government Printing Office, Washington D.C.

Soroka, S. N. 2003. Media, public opinion, and foreign policy. The International Journal of Press/Politics **8**:27-48.

Tisdell, C. A. 1982. Wild pigs: environmental pest or economic resource. Pergamon Press Ltd.

USDA. 2013. Environmental Impact Statement; Feral Swine Damage Management.*in* USDA, editor. 78 FR 27937. Office of the Federal Register, National Archives and Records Administration, Federal Register Volume 78, Issue 92.

USDA. 2014. Quick stats. National Agricultural Statistics Service, US Department of Agriculture, Washington, DC.

Waithman, J. D., R. A. Sweitzer, D. Van Vuren, J. D. Drew, A. J. Brinkhaus, I. A. Gardner, and W. M. Boyce. 1999. Range expansion, population sizes, and management of wild pigs in California. The Journal of wildlife management:298-308.

Walgrave, S., S. Soroka, and M. Nuytemans. 2008. The Mass Media's Political Agenda-Setting Power A Longitudinal Analysis of Media, Parliament, and Government in Belgium (1993 to 2000). Comparative Political Studies **41**:814-836.

Weart, S. 1988. Nuclear Fear: A history of images Harvard university Press, Cambridge, MA.

Wood, G. W., and R. H. Barrett. 1979. Status of wild pigs in the United States. Wildlife Society Bulletin:237-246.

CHAPTER 3

DISEASE PREVALENCE IN AN INVASIVE SPECIES DEMONSTRATES TRADEOFFS BETWEEN SPECIES DIVERSITY AND ENVIRONMENTAL CONDITIONS

INTRODUCTION

Emerging and re-emerging diseases of wildlife increasingly pose threats to animal and human health. Studies across a range of host-pathogen systems indicate that host species' diversity can regulate the ability for a pathogen to establish Johnson et al. (2015). Studies of natural populations have investigated correlations between the spatial patterns of disease and environmental variables, particularly those that can encourage immunologic susceptibility in a population (Buskirk and Ostfeld 1998, Giraudoux et al. 2003). However there may be tradeoffs between species diversity and environmental conditions (Moore and Borer 2012). The interaction of environmental conditions influencing the host or pathogen may operate at scales different than those of species diversity generating asymmetric effects on pathogen transmission (Huang et al. 2016). These effects may also be different for pathogens with a narrow versus large host range. Yet studies investigating relationships between pathogen persistence, environmental factors, and species diversity for pathogens with narrow and wide host ranges are still relatively limited, particularly at the macro-scale and for mammal species in North America.

Studies of host–pathogen systems suggest that the species diversity of communities can influence pathogen prevalence through several mechanisms generally referred to as amplification and dilution effects (Keesing et al. 2006, Ostfeld and Keesing 2012, Salkeld et al. 2013, Johnson et al. 2015, Huang et al. 2016). Dilution effects have been proposed to result primarily from increased species diversity that lowers encounter rates and in turn transmission (Johnson et al.

2015, Huang et al. 2016). Often termed the 'diversity-disease hypothesis', its origins can be traced back to Charles Elton (Elton 2000). Conversely amplification effects of species diversity are predicted to occur when increased diversity of competent hosts leads to an intensification of transmission in a community (Keesing et al. 2006). Empirical studies across a range of host-pathogen systems indicate that high species diversity has been associated with reduced disease occurrence (Knops et al. 1999, Mitchell et al. 2002, Pautasso et al. 2005). However, the role of diversity in multi-host systems may be complex, especially when asymmetric transmission competency among hosts exists, leading some to conclude that the effect of species diversity is idiosyncratic and variable among systems (Keesing et al. 2010, Salkeld et al. 2013). Recently others have proposed that there may be common underlying ecological phenomena that give rise to the perceived idiosyncratic differences in the effect of species diversity. Johnson et al. (2015), Huang et al. (2016), and others have proposed that dilution and amplification may result from non-linear relationships among density and diversity of competent and non-competent hosts, environmental conditions experienced by host and pathogen, scale of both measurement and species interactions, and the metric used to measure the effect (e.g. prevalence versus incidence). To date few studies have attempted to investigate these relationships and additional variables, particularly at broad scales, because the data required are generally unavailable or difficult to collect. A further complication for conducting these studies is observation error.

Detection probabilities (true and false) for both the pathogen and the wildlife host are typically not included in wildlife disease ecology studies (McClintock et al. 2010). Several recent studies have provided evidence that failing to account for observation error can bias even the simplest estimators in a significant way or even change inference about species occupancy (Royle and Link 2006, McClintock et al. 2010, Lahoz-Monfort et al. 2014). Accounting for

observation errors may be even more important when investigating community level processes that are often difficult to detect (Salkeld et al. 2013). In the case of invading species – host or pathogen - this maybe particularly important, especially if the pathogen or host invasion is recent where observation error rates, either true or false detection, can be larger (MacKenzie et al. 2003). For pathogens this may result from individual heterogeneity in immunity, heterogeneity in population immunity and heterogeneity in infection rates across host populations (Pepin et al. 2017). While for invasive host species that by nature are not in an equilibrium state, this may result from heterogeneous distribution of individuals or difficulty in observing the host species due to low densities. Together uncertainty in detection for host and pathogen may obscure effects of ecological processes, and accounting for these errors may provide insight when investigating species diversity-disease relationships.

Wild pigs are one of the most successful invasive mammal species globally and the most abundant free-ranging, exotic ungulate in the United States (Lewis et al. 2017). They are a generalist species, successfully inhabiting a wide range of ecosystems globally and in North America that represent a large gradient of species diversity (e.g. boreal plain in Canada to South Western deserts of the United States) (Brook and van Beest 2014, Lewis et al. 2017). Wild pigs are also a host for over 40 pathogens of concern for human and animal health (Bevins et al. 2014, Miller et al. 2017). The generalist nature of wild pigs and the large number of pathogens they can be a host for makes wild pigs a good study system to investigate species diversity-disease relationships. The processes that facilitate or inhibit introduction and establishment of pathogens in invasive wild pig populations is largely unstudied in North America with most studies reporting apparent prevalence or demographic risk factors (Leiser et al. 2013, Miller et al. 2017, Pedersen et al. 2017). In North America the spatial distribution of pathogens of wild

pigs has only been investigated for a small subset of pathogens primarily of importance for human or domestic animal health. Few have investigated environmental correlates of pathogen prevalence, and none have investigated disease-diversity relationships. However, the well-documented alternative hosts of wild swine pathogens makes them an ideal species in which to investigate complex disease-diversity relationships.

We use a hierarchical Bayesian approach that accounts for imperfect detection probability to investigate the influence of species diversity on the infection probability in wild pigs for pathogens with broad and narrow host ranges. We use these pathogens to investigate if amplification or dilution effects of species diversity might contribute to pathogen prevalence and if environmental conditions that commonly cause stress in mammal populations may contribute to or dampen the effect of species diversity. Because true and false detection probabilities can influence inference and have rarely been included in studies of diversity-disease relationships, we further investigate the potential magnitude of the effect on parameter predictions for the ecological variables of interest. We discuss our results in terms of the diversity-disease hypothesis for pathogen invasion into an invasive species and species that are colonizing new habitats. We highlight the impact of accounting for imperfect detection probabilities on inference. We also discuss how including both ecological processes and imperfect detection can be used to improve predictions of wildlife disease prevalence ultimately improving management of these pathogens.

METHODS

Pathogen Data

We used data collected from January 2007 through December 2015 as part of the US Department of Agriculture, Wildlife Services' National Wildlife Disease Program (NWDP) - a national surveillance program of feral swine. These data have been previously described for various pathogens (Pedersen et al. 2012, Pedersen et al. 2013). Briefly they are collected as part of routine surveillance for a diversity of pathogens of concern for human and animal health. The data include the location (longitude-latitude coordinates), sex, and age of the animal along with serological assay results for a diversity of pathogens. Age class is determined based on lower jaw tooth eruption using an approach commonly used for wild pigs (Matschke 1967) and categorizes animals as juvenile (<2months), subadult (>2 months and ≤ 1 yr), and adult (≥1 yr). Testing for pathogens is limited to serological assays.

We selected two wild pig pathogens, pseudorabies virus (*Suid herpesvirus*) and swine brucellosis (*Brucella suis*), to investigate our hypotheses about dilution and amplification effects. The dilution effect of species diversity has been proposed to reduce disease risk in a community primarily by reducing transmission when low-competence hosts are present (Keesing et al. 2006, Ostfeld and Keesing 2012, Johnson et al. 2015). To investigate potential dilution effects, we selected pseudorabies virus, often termed Aujeszky's disease, which is an economically important disease of domestic swine. Pseudorabies virus is easily transmitted through direct contact via sexual (Romero et al. 2001) or nonsexual (horizontal) transmission (Smith 2012). A broad range of species are known to be susceptible to pseudorabies virus with the pathogen being highly virulent and typically fatal in non-porcine hosts (see Appendix Tables A3.1 and A3.2). Pseudorabies virus apparent prevalence in wild pigs in the United States is estimated to

range from 0% to 61% (Müller et al. 2011, Pedersen et al. 2013). We expected increases in susceptible hosts that have low-competence for this virus to reduce prevalence while accounting for host density and environmental conditions influencing host survival.

In contrast species diversity has been proposed to amplify disease transmission in communities that have a large number of competent host species (Keesing et al. 2006, Huang et al. 2016). That is increasing competent host diversity in a community amplifies transmission and in turn disease prevalence. We used swine brucellosis to investigate potential amplification effects of species diversity. Swine brucellosis is easily transmitted through damaged skin or through mucosal membranes in the respiratory, reproductive, and gastrointestinal tracts (Olsen et al. 2011, Leiser et al. 2013). Routes of transmission in wild pigs are thought to occur through direct contact during intercourse, fighting, or via contact with contaminated aborted fetuses. Swine brucellosis has a large host range and is known to have competent non-porcine maintenance hosts (see supplemental). Swine brucellosis is common in North American wild pigs with apparent prevalence reported to range from 0.3% to 53% (Pedersen et al. 2012, Leiser et al. 2013). With regard to amplification effects, we expected increasing competent-host diversity to be associated with increased prevalence in wild pigs and that environmental conditions influencing host survival would have reduced effects on prevalence due to the differences in host survival relative to environmental conditions.

Scale of analysis

We used hydrologic units from the United States Geological Survey's (USGS) Hydrologic Unit Codes (HUC) as the analysis unit (USGS 2011). Hydrologic units, commonly referred to as watersheds, are a hierarchical classification system that are considered to be ecologically important landscape-level sampling units for studies with large extents (Odum et al.

1971). They have been used as biologically representative sampling units for modeling species occurrence (Collins and Glenn 1990, Peterson et al. 2009) and have previously been used to model invasive wild pig occurrence probability in the United States (McClure et al. 2015). Watersheds represent a discrete set of biotic and abiotic factors and to serve as a ecologically relevant unit for aggregating covariates. We chose a watershed size (HUC8 referred to as subbasin) that was much larger (mean=1,800 km^2) than the mean home range size for wild pigs in the U.S. (~5 km^2) and was expected to be capable of encompassing a population of pigs (McClure et al. 2015). In addition the subbasin scale represented a balance between data density within each subbasin and also being large enough to encompass a population of pigs. Surveillance data were assigned to each subbasin using their location (longitude-latitude coordinates). Hydrologic units have the advantage of being a hierarchical system allowing watersheds to be aggregated or disaggregated while preserving the ecological relationships. Using this characteristic we also considered hydrologic basins (HUC6), and subregions (HUC4) for validation (see Model implementation and performance) and hydrologic regions (HUC2) for observation processes (see Observation model).

Hydrologic subbasin-level environmental data

We evaluated four climatic covariates available from WorldClim (Hijmans et al. 2005) that represent environmental gradients that have been found to be important for limiting the geographic distribution (McClure et al. 2015), the density (Lewis et al. 2017) and the invasion probability (Snow et al. 2016) of wild pigs. The subbasin-level mean for each climatic variable was calculated using methods described in McClure et al. (2015). Pigs are known to have physiological characteristics making them sensitive to extreme temperatures (high and low) (Geisser and Reyer 2005, Acevedo et al. 2006). Pig mortality increases when ambient

temperatures exceed 23°C (Porter and Gates 1969) and when temperatures fall below -4°C (Thompson et al. 1996). To represent these temperature gradients, we used the long term annual mean temperature in the coldest quarter of the year (WorldClim variable BIO11) and the long term annual mean temperature in the driest quarter of the year (WorldClim variable BIO9). We expected increasing temperature in the coldest quarter to be positively associated with pig survival and thus also positively associated with pathogen prevalence. Wild pigs thermo-regulate by accessing water resources (Choquenot and Ruscoe 2003) and require cooling when temperatures exceed 35°C. We used the mean annual temperature of the driest quarter to represent this gradient and expected pig mortality and pathogen prevalence to be negatively correlated as this gradient increased.

Wild pig survival at low temperatures is expected to be exacerbated by precipitation, which in northern climates equates to snow accumulation (Jedrzejewska et al. 1997, Melis et al. 2006, Honda 2009, Danilov and Panchenko 2012). Winter temperature and snow depth have been associated with wild pig occurrence probability (McClure et al. 2015) and wild pig density (Melis et al. 2006, Pedersen et al. 2017). We used the mean amount of precipitation in the coldest quarter (WorldClim variable BIO19) to represent winter precipitation expecting pig mortality to increase and pathogen prevalence to decline with increasing winter precipitation. Conversely, increasing precipitation during the warmest months of the year is expected to decrease the effects of increasing temperature, improving survival (Fraser and Phillips 1989) and has been linked to pig probability of occurrence. To represent this precipitation gradient that is somewhat orthogonal to temperature in the driest quarter, we used precipitation in the driest quarter (WorldClim variable BIO17). We expected wild pig survival and pathogen prevalence to increase as precipitation increased in the driest period of the year.

Subbasin-level host species diversity data

To investigate the potential influence of mammal species diversity on pathogen prevalence, we used Shannon–Weaver species richness index as a proxy for species diversity (Shannon 1949). Species richness is often used as a proxy for species diversity when studying potential dilution or amplification effects of pathogen prevalence (Salkeld et al. 2013). We considered two cases for mammal species richness that have been debated to influence pathogen prevalence in different ways (Johnson et al. 2015) – changes in low-competence hosts (Keesing et al. 2006, Huang et al. 2016) and changes in competent hosts (Huang et al. 2013a). To identify these species we used the results of two meta-analyses by Miller et al. (2017) and Miller et al. (2013) that investigated the transmission potential of 86 pathogens between wild pigs, livestock and wildlife. Based on these studies, we considered competent hosts as those that could be clinically or sub-clinically infected and also demonstrated the ability to shed virus, while non-competent hosts are those that are susceptible but unable to transmit the pathogen. Using these constraints, 28 species from ten families were included as competent hosts for swine brucellosis and 34 non-competent species from 21 families for pseudorabies virus (See supplemental for tables listing species with supporting citations). Because experimental infection studies often use a similar set of species that are easy to work with in the laboratory, we assumed that experimental infection study results were broadly applicable at the taxonomic family level. To develop a measure of species richness, we obtained geographic range data for mammal species from the Nature Serve digital map library of the distributions of the terrestrial mammals of the Western Hemisphere that contains distribution data for over 1,700 species (Patterson et al. 2007). Using these range data, the presence / absence of each species was aggregated to hydrologic

subbasins as described in McClure et al. (2015), and the subbasin-level Shannon–Weaver species richness index was calculated.

Wild pig abundance data

Disease transmission often demonstrates density dependence (Begon et al. 1999), and this relationship can influence intra- and inter-species interactions, influence transmission rates and also alter the effect of species diversity on pathogen prevalence (Johnson et al. 2015). Because we were interested in the influence of species richness on the prevalence of pathogens in wild pigs, we accounted for population density only in the focal species in our analysis. Currently there are no available national scale estimates of wild pig abundance at a resolution (subbasin) useful for our analysis. To account for density dependence of the focal host species (wild pigs), we used the mean subbasin-level relative occurrence probability described by McClure et al. (2015) as a proxy measure of population density. Relative occurrence probability is expected to be proportional to population abundance (Brown 1984) and offered the only available national scale data for our purposes.

Hierarchical model of pathogen infection

We constructed an occupancy model using a hierarchical formulation (Royle and Kéry 2007) for modeling individual infection probability and for estimating subbasin-level prevalence (Figure 3.1). We express the model by its two component processes that are the observations, y_{ij}, conditional on the unobserved state process (i.e., $y_{ij}|z_{ij}$) and, the unobserved or partially observed state process, z_{ij},where *i* indexes the individual pigs sampled for the pathogen and *j* indexes the subbasin that they were located. This formulation allowed us to investigate multiple levels of pathogen infection probability (e.g. individual and subbasin) and allowed us to

investigate the relationship between environmental gradients, species diversity, wild pig host density, and the infection state, z_{ij}, that was the primary focus of our investigation.

State model

The state model relates the probability of infection for each individual to the hypothesized demographic and environmental processes influencing the infection state. The state of individual, z_{ij}, is a Bernoulli process

$$z_{ij} \sim \text{Bernoulli}(\psi_{ij}) \quad \text{Eq. 1}$$

where ψ_{ij} is the probability of infection for individual *i* in subbasin *j* conditional on environmental and demographic processes. z_{ij} has the possible states of 'positive' ($z_{ij} = 1$) or 'negative' ($z_{ij} = 0$). We modeled the relationship of demographic and environmental processes to infection state as a linear process on the logit scale as,

$$\text{logit}(\psi_{ij}) = \beta_{0,j} + \boldsymbol{x'}_{ij}\boldsymbol{\beta} \quad \text{Eq. 2}$$

where $\boldsymbol{\beta}$ is a vector of regression coefficients corresponding to $\boldsymbol{x'}_{ij}$, the transpose for the vector of demographic covariates of sex and age (juvenile, yearling, adult). We used index variables for both sex and age assuming that both are observed without error. Sex was coded relative to males and centered on zero with values -0.5 for males and 0.5 for females. This results in a one unit difference in males and females allowing the predicted posterior regression coefficient to be interpreted as the mean difference between males and females (Gelman and Hill 2006). Age class was coded one for juvenile, two for yearling, and three for adult. A standard normal distribution with precision of 0.01 was used as a prior for both age and sex regression coefficients ($\boldsymbol{\beta}$).

The intercept of Eq. 2, $\beta_{0,j}$, is the influence of subbasin-level factors on the probability of infection common to all individuals in the subbasin and is modeled as

$$\beta_{0,j} \sim \text{normal}\left(\eta_0 + \boldsymbol{w'}_j\boldsymbol{\eta}, \sigma^2_{\beta_0}\right) \quad \text{Eq. 3}$$

where $\boldsymbol{\eta}$ is a vector of regression coefficients corresponding to the subbasin-level predictors and η_0 is the background infection probability common across all subbasins. We used a vaguely informative inverse gamma prior distribution (α=0.5, β=0.5) to model the precision, $1/\sigma^2_{\beta_0}$, of subbasin-level factors contributing to the infection state. A standard normal prior distribution with precision of 0.01 was used for all subbasin-level regression coefficients ($\boldsymbol{\eta}$) and the intercept η_0. We chose not to include uncertainty in the subbasin-level covariates assuming that they are observed without error. The environmental and pig occurrence probability covariates are estimated from other models with unknown error. Similarly the species richness covariate is derived from sixty-two species range estimates that arise from an aggregate of scientific literature sources. Because the error is unknown for these covariates and inclusion of data models that would account for the error would likely be inaccurate, we did not include a data model for these covariates.

Observation model

The observation model specified conditional on the infection state, z_{ij}, is given by

$$y_{ij}|z_{ij} \sim \text{Bernoulli}\left(\left(z_{ij}\rho_k + (1 - z_{ij})(1 - \phi_k)\right)\mathbf{1}_{j\in k}\right) \quad \text{Eq. 4}$$

where ρ_k is the true positive detection probability and $(1 - \phi_k)$ is the false positive detection probability in hydrologic region *k*. An indicator variable, $1_{j\in k}$, is used to identify when subbasin *j* is contained within hydrologic region *k* by,

$$1_{j\in k} = \begin{cases} 1 \; if \; j \in k \\ 0 \text{ otherwise} \end{cases} \quad \text{Eq. 5}$$

This formulation, which is common in epidemiology (McClintock et al. 2010, Christensen et al. 2011) and increasingly common in species occupancy models (Royle and Link 2006, Miller et

al. 2011), provides an estimation of the true state of an individual, in our case infected or not infected, accounting for false positive and false negative errors.

The true positive detection probability, ρ_k (also referred to as true positive rate or sensitivity in some fields), and true negative detection probability (i.e. true negative rate or specificity), ϕ_k, can result from a diversity of processes including diagnostic test error (Branscum et al. 2005), population immunity (Pepin et al. 2017), and differences in detectability of the pathogen given infection status of the animal (Jennelle et al. 2007). The diagnostic tests used for pseudorabies virus and swine brucellosis were developed and validated for domestic animals and performance of diagnostic tests are often different for wildlife (Stallknecht 2007). The beta distribution was used as a prior to account for true positive and true negative detection error arising from diagnostic error and other processes. We used a parameterization common in epidemiological studies (Branscum et al. 2005, Christensen et al. 2011) that is often termed the expert beta. We parameterized ρ_k and ϕ_k with respect to the mean for the diagnostic error using,

$$\rho_k \sim \text{beta}\left(\mu_{\rho_k}\, \sigma_{\rho_k}, \left(1-\mu_{\rho_k}\right)\sigma_{\rho_k}\right) \quad \text{Eq. 6}$$

$$\phi_k \sim \text{beta}\left(\mu_{\phi_k}\, \sigma_{\phi_k}, \left(1-\mu_{\phi_k}\right)\sigma_{\phi_k}\right) \quad \text{Eq. 7}$$

where μ_{ρ_k} and μ_{ϕ_k} are beta distributed hyper-priors for the detection means and σ_{ρ_k} and σ_{ϕ_k} are gamma distributed hyper-priors for the mean detection variances. This parameterization allows the mean and variance for each detection probability to be defined in terms of the confidence in the available data (Christensen et al. 2011). We assumed in the absence of other contributors to observation error that the true positive and true negative detection rates would approach those of the diagnostic test. Deviations from the diagnostic test values would indicate other contributions to detection errors are present. For this purpose, we assumed μ_{ρ_k} and μ_{ϕ_k} were the reported

diagnostic test error rate with 65% confidence, and we assumed that the true value of μ_{ρ_k} and μ_{ϕ_k} was greater than 0.6 with 95% confidence. This parameterization allowed some uncertainty in the true value of μ_{ρ_k} and μ_{ϕ_k} with the null hypothesis that the mean value is the same as the diagnostic test (Appendix Table. A3.3).

Derived subbasin-level quantities

The parameters of the occupancy model of primary interest are the individual-level true infection state probability, ψ_{ij}, the true positive detection probability, ρ_k, the true negative detection probability, ϕ_k and the regression coefficients ($\boldsymbol{\beta}$, $\boldsymbol{\eta}$). To derive the subbasin-level true occurrence probability, which in our model is based on the individual-level true infection probability, we generated realizations from the posterior distribution of ψ_{ij} using,

$$\hat{\pi}_{true_j} = \frac{1}{n_j}\sum_{j=1}^{J}\psi_{ij} \qquad \text{Eq. 8}$$

where $\hat{\pi}_{true_j}$ is the derived true subbasin-level pathogen prevalence conditional on the demographic, environmental, species diversity, and wild pig occurrence probability parameters. Because $\hat{\pi}_{true_j}$ is generated from ψ_{ij}, it represents a subbasin-level posterior prediction that applies to a theoretically infinite population of individuals (Royle and Dorazio 2008) and can be interpreted as the probability of disease for any individual animal sampled from the subbasin (Royle and Kéry 2007).

To understand the influence of including detection probabilities and the resulting influence on subbasin-level derived predictions of true pathogen prevalence, we contrast $\hat{\pi}_{true_j}$, the derived true subbasin-level prevalence, with the standard approach (often termed apparent prevalence) using a metric describing relative bias (%RB) (Jennelle et al. 2007). The bias metric is defined as

$$\%\text{RB} = \frac{\left(\pi_{obs_j} - \hat{\pi}_{true_j}\right) * 100}{\hat{\pi}_{true_j}} \quad \text{Eq. 9}$$

Where π_{obs_j} is the observed apparent prevalence in subbasin *j* defined as

$$\pi_{obs_j} = \frac{1}{n_j}\sum_{j=1}^{J} y_{ij} \quad \text{Eq. 10}$$

where y_{ij} (which when summed) is the observed number of positive animals in subbasin *j*, n_j is the total number of animals tested in subbasin *j*, and $\hat{\pi}_{true_j}$ is the derived true subbasin-level pathogen prevalence.

We were also interested in the ability of the model to accurately predict subbasin-level apparent prevalence so we could evaluate model performance. To derive the subbasin-level predicted apparent prevalence, we simulated new values of $y_{ij}|z_{ij}$ using Eq. 4 and then calculated

$$\hat{\pi}_{pred_j} = \frac{1}{n_j}\sum_{j=1}^{J} y_{ij}|z_{ij} \quad \text{Eq. 11}$$

where $\hat{\pi}_{pred_j}$ is the predicted subbasin-level apparent prevalence conditional on the true positive and true negative detection errors and the individual and subbasin-level processes defined in Eq. 2 and 3.

Model implementation and performance

All environmental and demographic covariates were centered prior to model fitting, and effects were standardized to allow comparison between covariates and models. Posterior distributions of the infection states and parameters of interest were predicted using Markov chain Monte Carlo (MCMC) methods using three chains with diffuse initial conditions (Brooks and Gelman 1998). Each parameter and infection state was predicted by sampling from the posterior

distribution using Gibbs sampling implemented in JAGS (Plummer 2014) and the runjags (Denwood 2016) package in the R computing environment (Team 2011). The MCMC procedure was run until convergence of all model parameters was achieved. Once convergence was assured, posterior inference was based on 20,000 samples from the MCMC chains. Convergence was evaluated by visual inspection of trace plots, the Gelman-Rubin diagnostic (Gelman and Rubin 1992), and the Heidelberg-Welch diagnostic (Heidelberger and Welch 1983). Convergence diagnostics and statistical analysis of the model output was performed using the R coda package (Plummer et al. 2006).

Out-of-sample prediction was used to assess model performance. Subbasins to withhold for model validation were identified using conditioned Latin hypercube sampling (Minasny and McBratney 2006) that allows ancillary data to be used to stratify sampling. We were interested in the predictive abilities of the model across three gradients important for invasive wild pigs in North America. These gradients included: 1) the range of observed apparent prevalence (0 to 1.0), 2) the range of latitudes that pigs occur in North America (N 26° to N 48°), and 3) the length of time wild pigs have been present in a subbasin (<1 year to >100 years). These ancillary data were used to identify approximately 10% of subbasins to use as out-of-sample validation data (Table 3.1).

Posterior predictive evaluations for the ability of the model to predict subbasin-level apparent prevalence were conducted to evaluate the fit of the model to the data (Gelman and Hill 2006, Gelman et al. 2014). Posterior predictive checks use a test statistic calculated from the observed data and from replicated data sets simulated from the posterior predictive distribution. To implement this procedure, we generated replicate data sets of the predicted subbasin-level apparent prevalence (Eq. 11) from the MCMC chains after obtaining convergence. We

calculated Bayesian *P*-values for the mean discrepancy between $\hat{\pi}_{pred_j}$ and π_{obs_j}. Bayesian *P*-values provide a measure of how extreme the predicted data are in comparison to the observed data. Values near 0.5 indicate good fit, while values close to 0 indicate model predictions that are less than the observed data and values close to 1 indicate predictions that are greater than the observed data (Gelman et al. 2014). We also assessed out-of-sample prediction capacity by computing the mean square error (MSE) between observed apparent prevalence (π_{obs_j}) and the predicted apparent prevalence ($\hat{\pi}_{pred_j}$) across the three gradients to indicate discrepancies in dispersion of the predicted data relative to the observed data. Because models often predict better at a courser resolution, we evaluated Bayesian *P*-values at the subbasin (HUC8), basin (HUC6), and subregion (HUC4) hydrologic scales.

RESULTS

Model evaluation

All diagnostics indicated model convergence for the pseudorabies virus and swine brucellosis models for all chains following a 1-million iteration burn-in. Model run times were long ranging from 2-2.5 hours for each 100,000 iterations and required several days to run for convergence. Convergence was easier to achieve for pseudorabies virus than for swine brucellosis, which required two million iterations. Trace plots indicated thorough mixing of all chains, and the upper 97.5% quantile of the Gelman-Rubin diagnostic was less than 1.01 for all parameters in both models after convergence. Chains passed the Heidelberger-Welch test for stationarity and mean half width.

Posterior predictive checks did not indicate significant lack of fit between model predictions and the data (Table 3.2). The mean Bayesian *P*-values for the difference in the mean

observed apparent prevalence and mean predicted apparent prevalence among subbasins ranged from 0.768 to 0.573 pseudorabies virus and 0.869 to 0.694 for swine brucellosis. Bayesian *P*-values approached 0.5 for both pathogens as hydrologic unit became increasingly coarse (subbasin to basin to subregion) indicating our model predictions improved as hydrologic units were aggregated. Bayesian *P*-values greater than 0.5 indicated that the predicted apparent prevalence tended to be less than the observed apparent prevalence.

Comparison of out-of-sample data with model predictions of subbasin-level predicted apparent pathogen prevalence provided confidence that our model could accurately represent the observed apparent pathogen prevalence (Figure. 3.2). Our model accurately predicted subbasin-level observed apparent prevalence in the majority of subbasins, 96.3% for pseudorabies virus and 93.0% for swine brucellosis (Figures 3.2 and 3.3). The models performed well across the range of latitudes considered (26° to 48°) but differed for each pathogen. Latitudes from 26° to 42° had relatively similar amounts of prediction error for swine brucellosis, but an increase in prediction error occurred above 42°. Prediction error for pseudorabies virus was greatest at 26° to 28° latitude, declined through the majority of wild pig range, and then increased some at latitudes above 42°. Prediction errors remained below 0.08 for all latitudes. The prediction error by the length of time pigs have been present in a subbasin had no significant differences for pseudorabies virus and showed some differences for swine brucellosis but remained less than 0.03 across all subbasins (Figure 3.3). In aggregate these assessments indicate that our models had good predictive capacity across the majority of the observed prevalence distribution and across the majority of the wild pig range in the United States providing confidence in our predictions of true apparent prevalence.

Parameter predictions

Generally parameters were predicted with narrow credible intervals relative to the prior distribution and medians that were different than the prior distribution demonstrating that the data informed parameters beyond the information contained in the priors (Table 3.3, 3.4 and Figure 3.4). Derived subbasin-level true pathogen prevalence declined for both pseudorabies virus and swine brucellosis as species diversity increased (Table 3.4 and Figure 3.5). The posterior distribution for the effect of species diversity on pseudorabies virus had a much narrower range when compared to swine brucellosis.

The two pathogens had different responses to environmental gradients associated with host survival. Pseudorabies virus derived true pathogen prevalence was more sensitive to environmental gradients associated with cold temperatures and precipitation with posterior coefficients that did not overlap zero. Pseudorabies virus derived true prevalence declined with increasing precipitation during the coldest quarter of the year. Conversely as the temperature during the coldest quarter increased (i.e. became warmer) pseudorabies virus derived true prevalence increased. All posterior distributions for environmental gradients for swine brucellosis had 95% credible intervals that overlapped zero. However, two environmental gradients, precipitation and temperature in the driest quarter, had greater than 0.92 probability that the posterior was greater than zero. Similarly, temperature in the coldest quarter had 0.95 probability that the posterior was less than zero.

Wild pig probability of occurrence was positively associated with derived true prevalence for both pathogens and did not overlap zero. Age was associated with increased probability of infection for both pathogens, but 95% credible intervals overlapped zero for swine brucellosis.

Females for both pathogens had a similar, higher probability of infection than males, however in both cases the 95% credible interval overlapped zero.

Comparison of derived true prevalence and observed apparent prevalence

Median derived true prevalence among all subbasins for pseudorabies virus was 52% greater than observed apparent prevalence with non-overlapping 95% credible intervals (Table 3.4) indicating that including true positive and true negative detection errors in the model improved prevalence predictions. Similarly swine brucellosis also had a derived true prevalence that was 144% higher than observed apparent prevalence with non-overlapping 95% credible intervals. The distribution of these differences was non-random. For both pathogens, relative bias between derived true prevalence and observed apparent prevalence had an unequal spatial distribution with a gradient from under- to over-estimation from south to north (Figure 3.6).

Predictions of true and false detection

True and false detection varied among hydrologic regions (Figure 3.5) and differed from published values for diagnostic test sensitivity and specificity (Table 3.4). Posterior credible intervals of the mean true detection probability among all hydrologic regions were lower than published diagnostic test values for both pathogens. True negative detection probabilities (i.e. sensitivity) were closer to published diagnostic test values for both pathogens. Pseudorabies virus mean true negative detection probability (i.e. specificity) among all hydrologic regions was within 1% of the published diagnostic test value and the lower 95% credible interval was above 0.95. The mean true negative detection probability among all hydrologic regions for swine brucellosis was above the published diagnostic test value of 0.97.

DISCUSSION

We found support for species richness dilution effects for pseudorabies virus and swine brucellosis after controlling for environmental gradients influencing host survival, host density, and pathogen detection errors. The results for pseudorabies virus align well with currently proposed theory in that increased diversity of non-competent hosts was associated with reduce pathogen prevalence (Keesing et al. 2006, Ostfeld and Keesing 2012, Young et al. 2013). In addition we also observed negative effects of environmental gradients that are associated with reduced host survival. However for swine brucellosis, we did not observe the expected species richness effect. Current species-diversity disease theory would have predicted amplification effects for swine brucellosis because the species richness index used was composed solely of competent species (Johnson et al. 2015, Huang et al. 2016). However, we observed a negative relationship with increasing species richness for swine brucellosis that is contrary to the amplification hypothesis.

There are several potential reasons why we did not observe the expected relationship for swine brucellosis. While our model predicted a negative effect for swine brucellosis, this negative effect had greater variance compared to pseudorabies virus indicating that there may be other processes involved that were not included in our model. These might include non-linear environmental relationships with alternate host survival or infection probability that obscure species-diversity effects on swine brucellosis prevalence for wild pigs. We also only accounted for host density for our focal species, wild pigs, and because swine brucellosis tends to occur at lower prevalence, there may be host density effects for alternate hosts that we did not include. These types of non-linear effects between species-diversity and infection probability have been termed identity effects (Hantsch et al. 2013, Huang et al. 2014) and have been observed for

bovine tuberculosis in Africa (Huang et al. 2014). In the case of bovine tuberculosis in Africa, increasing mammal species richness had a negative effect on disease risk; however in regions with African buffalo, there was a positive effect because African buffalo presence is correlated with species richness (Huang et al. 2013b). Collectively, these relationships highlight the importance of distinguishing non-linear associations between environmental gradients and host density for multi-host pathogens.

For single-host pathogens such as pseudorabies virus, environmental gradients associated with host survival may limit the ability of pathogens to invade populations in more northern climates reducing risks related to disease in these populations. For pseudorabies virus, our model predicts lowered disease prevalence in regions experiencing colder winters with greater precipitation, and while posterior distributions for swine brucellosis contained zero, they were also in the same direction as pseudorabies virus. Presumably this is associated with reduced host survival that might be further aggravated when the host is immunologically compromised by active infection. These effects were greater for pseudorabies virus, which is a life-long persistent infection that commonly worsens under stress or during reproduction. This effect is supported by reported observed apparent prevalence in European wild boar, which generally declines with latitude (Müller et al. 2011).

These environmental effects may be exacerbated by age and sex. The effects of age on infection probability were similar to those previously reported for wild pigs in North America (Pedersen et al. 2012, Pedersen et al. 2013) and also for wild boar in Europe (Ruiz-Fons et al. 2008). However our model did predict different effects of sex on infection probability for both pathogens. We found females had a higher probability of pseudorabies virus infection than males, which is different from two previous studies in North America (Pirtle et al. 1989, Müller

et al. 1998, Pedersen et al. 2013) but similar to findings for wild boar in Spain (Ruiz-Fons et al. 2008). There are several potential reason for the difference from previous studies in North America. Previous studies have not accounted for detection error, which may be different for males and females. These studies also used smaller sample sizes and did not include ecological covariates (e.g. environmental gradients, species diversity, or host density) when estimating effect sizes and hence may have obscured associations between infection probability and sex. Similar to our study, Ruiz-Fons et al. (2008) included environmental factors as covariates, supporting that there may be an interaction between sex and environmental factors in determining infection probability.

Our study has several implications for studies investigating relationships between pathogen prevalence, environmental risk factors, and species diversity. We observed large differences in detection errors that could not be solely explained by reported diagnostic test sensitivity and specificity. Studies that do not account for these types of errors, particularly at the macro-scale, might under estimate pathogen prevalence, and this may in turn influence estimated effect sizes for risk factors. This highlights not only the need to include these types of errors but also to further understand the underlying mechanisms responsible for these processes – both biological and measurement. Improvements to our approach might include explicitly modeling both biological and measurement processes that might contribute to true and false detection errors. This is commonly done in species occupancy models, however is rarely done in epidemiological models where true and false detection is generally assumed to arise solely from the diagnostic testing process (Branscum et al. 2005, Christensen et al. 2011). Of specific interest is investigating if individual and population level immunological processes may be

important in understanding pathogen detection (Pepin et al. 2017) indicating that both biology and measurement may be important in observation processes.

Our study could be extended by including greater resolution in terms of host competency. We assumed that all hosts were equally competent or non-competent for the pathogens. In reality, competency likely occurs along a gradient that has non-linear relationships with host density and other ecological factors influencing host survival. We assumed that infection study results for species were representative of the taxonomic family's host competence. This assumption may have included some species that are not competent due to heterogeneities within taxonomic families. Including greater detail for host competency may highlight host species that are important in the transmission process, either reducing or increasing pathogen transmission. Our model also assumed processes were stationary with respect to time, and there may be important nonlinear and potentially orthogonal effects of species richness on transmission during different seasons or they may change over time as host densities change. This may be particularly important for invasive species that are invading new communities potentially altering species assemblages and transmission processes and also for multi-host pathogens. Our model also did not explicitly include spatial auto-correlation in the model structure so our predictions may be overly optimistic. This may also be influenced by host density as there is an expectation that density of hosts is auto-correlated in space. Including spatial structure of both the focal host, wild pigs, and species richness could be an important extension of this work.

There are practical implications for management of disease risk in wild pig populations from our results. Our finding that environmental gradients are associated with changes in pathogen prevalence and may limit the ability of pathogens to invade populations experiencing stressful conditions may be useful for characterizing disease risk. Populations occurring in more

stressful environments may be at lower risk for disease outbreaks or for pathogens to become established as endemic. This may aid national scale surveillance efforts by allowing more resources to be diverted to areas with greater risk of pathogen establishment. This may also indicate that transmission risk from wild pigs to humans, domestic animals or other wildlife may be reduced for some pathogens in more northern regions of North America.

Our study fills a gap in the current knowledge related to the drivers of macro-scale pathogen prevalence for an important invasive species in North America. There are several implications for studies investigating relationships between pathogen prevalence and species diversity, particularly for multi-host pathogens. Relationships between species-diversity and pathogen prevalence may be obscured if environmental factors are not taken into account. For multi-host pathogens, prevalence-species diversity relationships may also be difficult to untangle if the range of host competency is not adequately represented, although we recognize that this may pose significant limitations for some studies. We also highlight the importance of including potential sources of error in these macro-scale epidemiological models. We found detection probabilities that deviated greatly from diagnostic test error indicating that other sources of error or ecological processes may be important. These may influence accurate observation of pathogens, drive pathogen invasion or persistence processes, and may influence our understanding of driving factors in studies that assume the pathogen is observed without error.

Table 3.1. Data used to fit the Bayesian hierarchical model and data withheld for out-of-sample model validation. Hydrologic subbasins used for out-of-sample validation were restricted to those with greater than 50 individual animals sampled and were identified using conditioned Latin hypercube sampling controlling for observed apparent prevalence, subbasin latitude and the length of time wild swine have been present in the subbasin.

	Pseudorabies virus		Swine brucellosis virus	
	Used for Model	Out-of Sample	Used for Model	Out-of Sample
Subbasins	486	61	496	52
Samples	12,592	5,683	13,796	4,528

Table 3.2. Bayesian *P*-values and 95% posterior quantiles for the lack-of-fit in the mean predicted apparent prevalence ($\hat{\pi}_{pred_j}$) and mean observed apparent prevalence (π_{obs_j}). Bayesian *P*-values are defined as the probability that the test statistic (difference in means) calculated from simulated data is more extreme than the test statistic calculated from observed data. A lack of fit is indicated by values near 1 or 0.

	Mean (95% posterior quantiles)		
	Subbasin (HUC8)	Basin (HUC6)	Subregion (HUC4)
Pseudorabies virus	0.768 (0.733-0.803)	0.604 (0.549-0.659)	0.573 (0.508-0.637)
Swine brucellosis	0.869 (0.836-0.896)	0.716 (0.667-0.754)	0.694 (0.645-0.742)

Table 3.3. Standardized mean, standard deviation (SD), median and quantile (0.025 and 0.975) for posterior distributions of parameters in the models for pseudorabies virus and swine brucellosis. The probability that the posterior does not contain zero, $\Pr(\beta \not\ni 0)$, is also reported.

Parameter	Mean	SD	Median	Quantile		$\Pr(\beta \not\ni 0)$
				0.025	0.975	
Pseudorabies virus						
Wild pig occurrence probability	0.697	0.217	0.689	0.296	1.151	1
Species richness	-0.525	0.063	-0.521	-0.661	-0.414	1
Precipitation driest quarter	0.309	0.284	0.300	-0.226	0.893	0.866
Temperature driest quarter	-0.115	0.341	-0.111	-0.797	0.546	0.632
Precipitation coldest quarter	-0.860	0.380	-0.846	-1.650	-0.147	1
Temperature coldest quarter	0.828	0.256	0.819	0.350	1.364	1
Age	0.583	0.083	0.584	0.420	0.747	1
Sex	0.085	0.135	0.088	-0.192	0.341	0.746
Swine brucellosis						
Wild pig occurrence probability	1.731	0.698	1.630	0.655	3.430	1
Species richness	-1.638	0.426	-1.577	-2.706	-0.974	1
Precipitation driest quarter	0.911	0.637	0.846	-0.149	2.372	0.947
Temperature driest quarter	0.909	0.664	0.884	-0.349	2.295	0.926
Precipitation coldest quarter	-0.785	0.795	-0.712	-2.590	0.593	0.853
Temperature coldest quarter	0.654	0.448	0.618	-0.126	1.640	0.946
Age	0.154	0.235	0.162	-0.328	0.576	0.750
Sex	0.047	0.227	0.060	-0.432	0.482	0.609

Table 3.4. Posterior distributions, medians, and quantiles (0.025 and 0.975) for derived true prevalence, predicted apparent prevalence, sensitivity, and specificity for pseudorabies virus and swine brucellosis.

Parameter	Median	Quantile	
		0.025	0.975
Pseudorabies virus			
Derived true prevalence ($\hat{\pi}_{true}$)	0.203	0.171	0.239
Predicted apparent prevalence ($\hat{\pi}_{pred}$)	0.134	0.119	0.151
Sensitivity (ρ)	0.688	0.602	0.771
Specificity (ϕ)	0.980	0.968	0.989
Swine brucellosis			
Derived true prevalence ($\hat{\pi}_{true}$)	0.105	0.072	0.136
Predicted apparent prevalence ($\hat{\pi}_{pred}$)	0.043	0.037	0.053
Sensitivity (ρ)	0.657	0.546	0.750
Specificity (ϕ)	0.988	0.978	0.994

a) Model specification

Observation model

$y_{ij}|z_{ij} \sim \text{Bernoulli}\left(\left(z_{ij}\rho_k + (1 - z_{ij})(1 - \phi_k)\right)\mathbf{1}_{j\in k}\right)$

True detection

$\rho_k \sim \text{beta}(\mu_{\rho_k}\sigma_{\rho_k}, (1 - \mu_{\rho_k})\sigma_{\rho_k})$

False detection

$\phi_k \sim \text{beta}(\mu_{\phi_k}\sigma_{\phi_k}, (1 - \mu_{\phi_k})\sigma_{\phi_k})$

Process model

$z_{ij} \sim \text{Bernoulli}(\psi_{ij})$

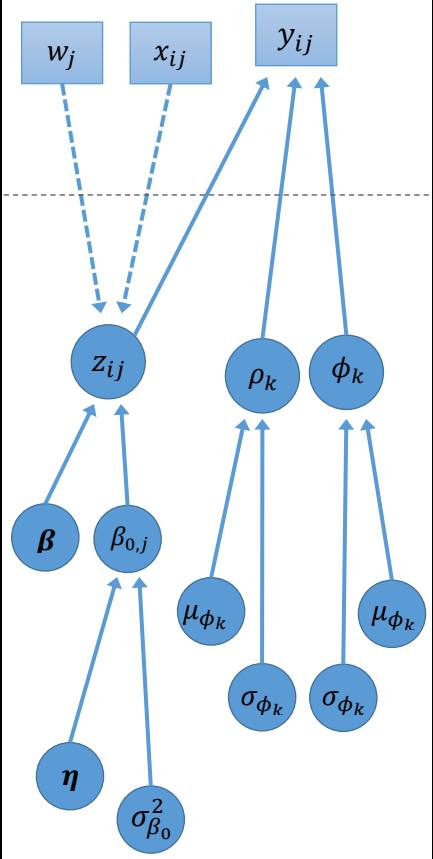
$\text{logit}(\psi_{ij}) = \beta_{0,j} + \boldsymbol{x}'_{ij}\boldsymbol{\beta}$

$\beta_{0,j} \sim \text{normal}(\eta_0 + \boldsymbol{w}'_j\boldsymbol{\eta}, \sigma^2_{\beta_0})$

$\boldsymbol{\beta} \sim \text{normal}(0, 0.001)$
$\boldsymbol{\eta} \sim \text{normal}(0, 0.001)$
$\sigma^2_{\beta_0} \sim \text{gamma}(0.5, 0.5)$

μ_{ρ_k} and μ_{ϕ_k} ~ beta() see appendix Table A3.3 for
σ_{ρ_k} and σ_{ϕ_k} ~ beta() values used

b) Directed acyclic graph



c) Observed quantities

y_{ij} Observed disease state of animal *i* in subbasin *j*

x_{ij} Observed age and sex of animal *i* in subbasin *j*

w_j Subbasin-level species diversity, environmental gradients, and wild pig probability of occurrence

d) Parameter definitions

z_{ij} Observed infection state of animal *i* in subbasin *j*

ρ_k Predicted true detection probability in the *k* hydrologic region

ϕ_k Predicted true negative detection probability in the *k* hydrologic region

μ_{ρ_k} and μ_{ϕ_k} are hyper-priors for the detection means

σ_{ρ_k} and σ_{ϕ_k} are hyper-priors for the detection variances

$\boldsymbol{\eta}$ Predicted coefficients for the subbasin-level environmental factors

$\boldsymbol{\beta}$ Predicted coefficients for the individual-level demographic factors

$\beta_{0,j}$ Predicted intercept for subbasin-level model

$\sigma^2_{\beta_0}$ Variance for subbasin-level species diversity, environmental gradients, and wild pig probability of occurrence

Figure 3.1. Model specification for hierarchical occupancy model used to predict pathogen prevalence and the effects of demographic and ecological processes.

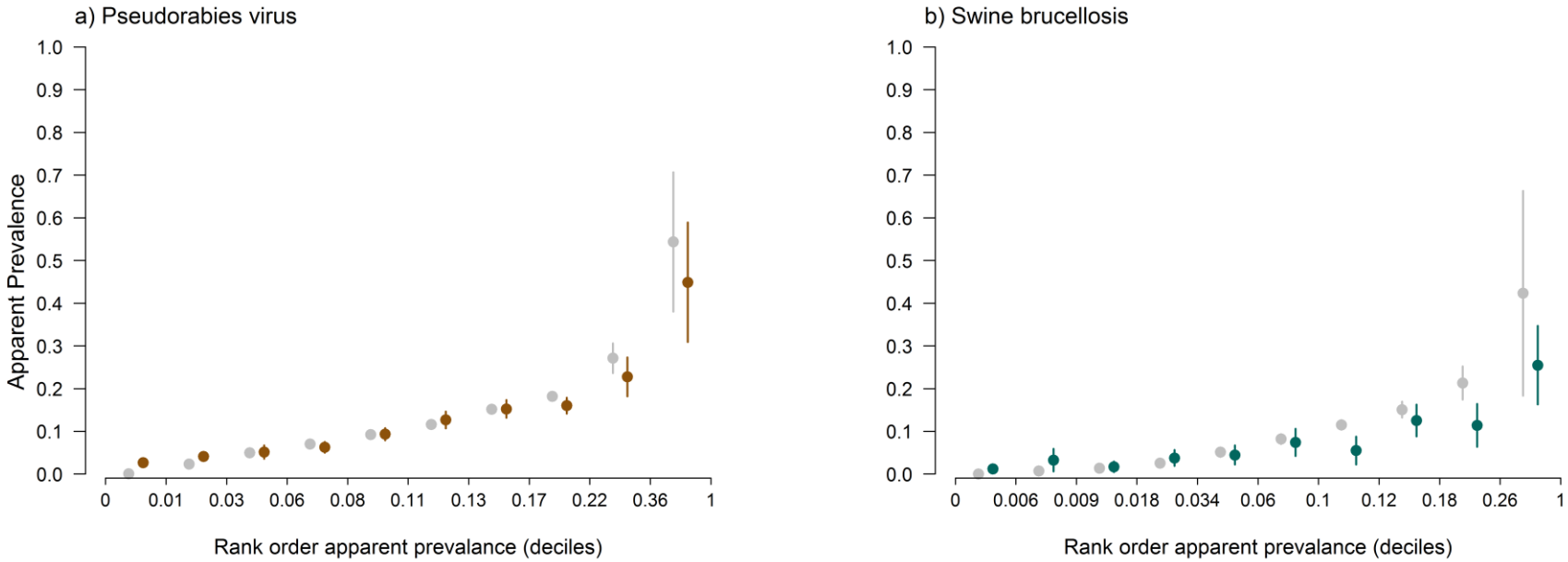


Figure 3.2. Model-predicted apparent prevalence for pseudorabies virus (brown) and swine brucellosis (green) compared with observed apparent prevalence (gray) for out of sample subbasins. Models accurately predicted apparent prevalence across the majority of observed apparent prevalence values. The x-axis is the rank-order of observed apparent prevalence by decile bins for subbasins. Models for both pathogens tended to under predict observed apparent prevalence for the upper 90th% values.

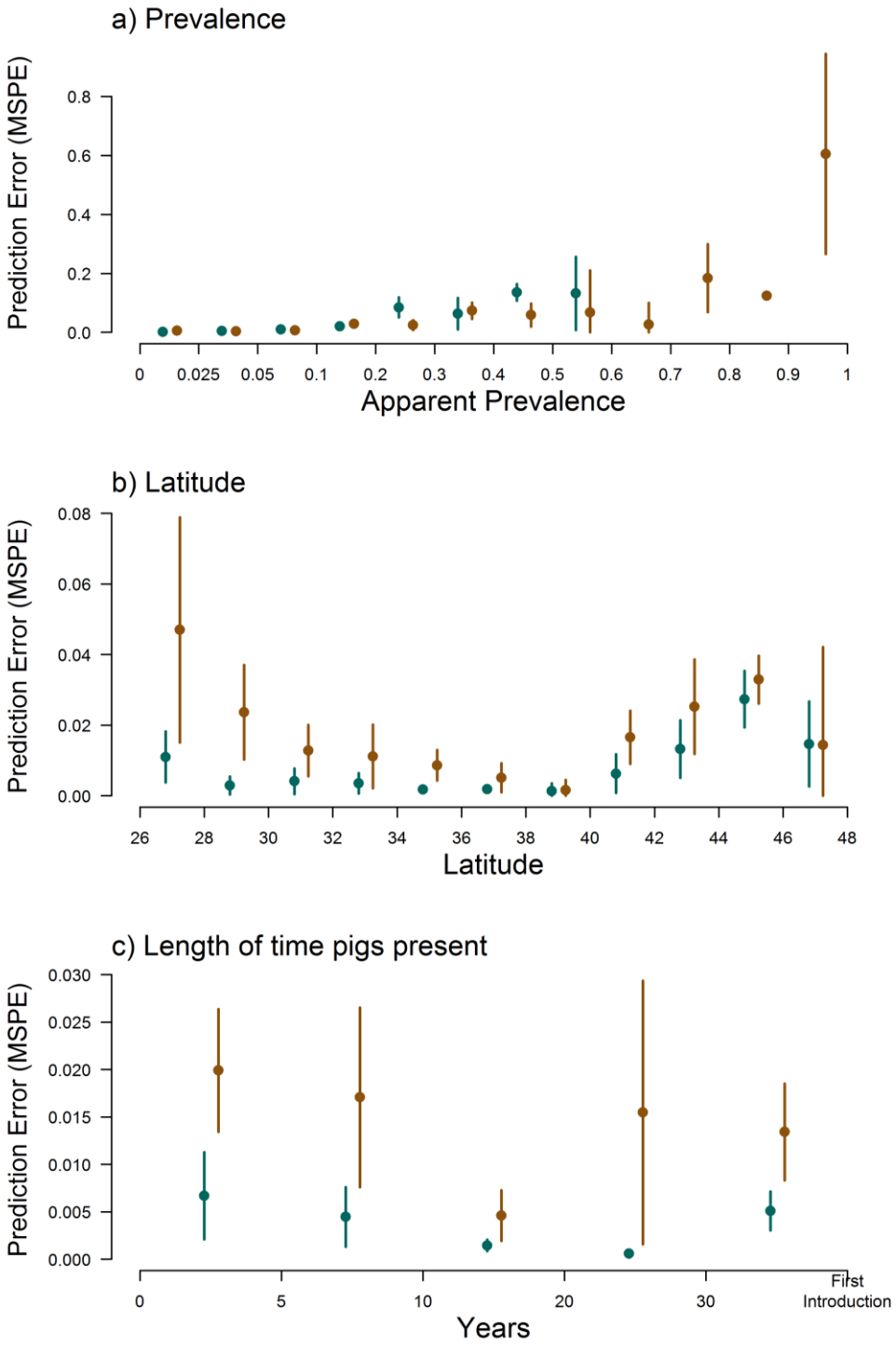


Figure 3.3. Mean square prediction error (MSPE) (y-axis) for model-predicted apparent prevalence for pseudorabies virus (brown) and swine brucellosis (green) across three gradients of importance for invasive pigs in North America. Panel a is the MSPE across the observed range of apparent prevalence; panel b is the MSPE for the range of latitudes that wild pigs occur in North America; panel c is the MSPE for the length time wild pigs have been present in subbasins.

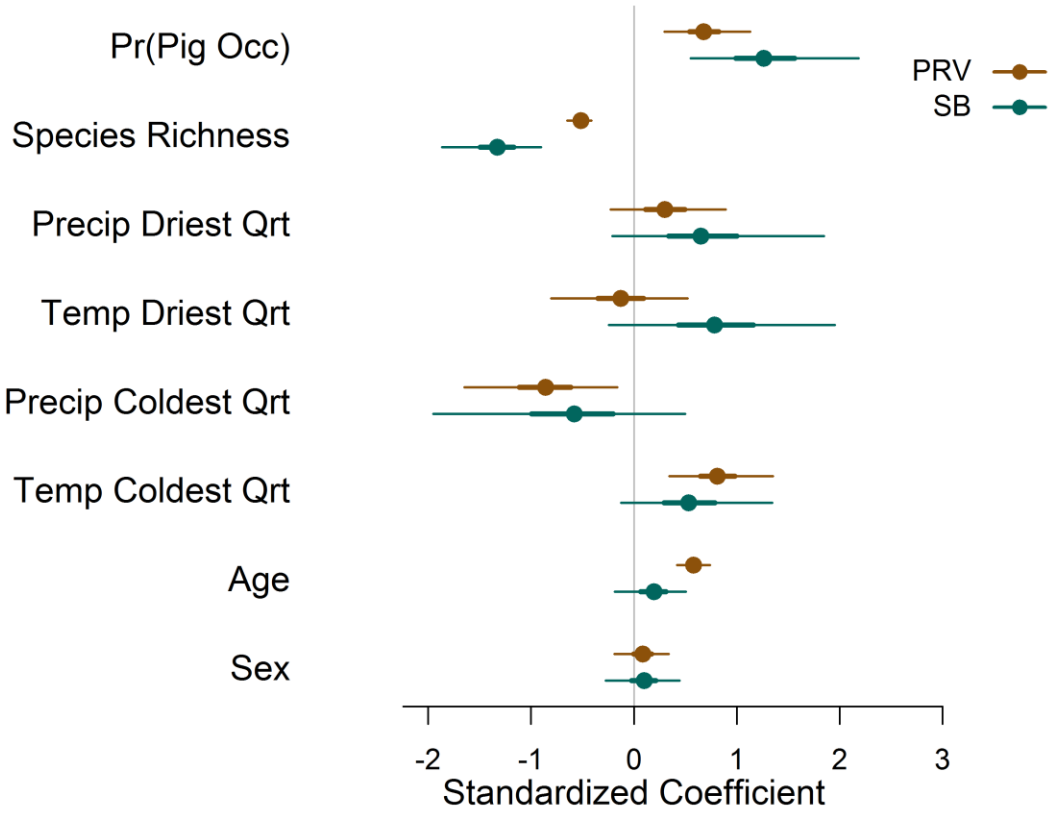


Figure 3.4. Posterior effects for individual and subbasin-level predictors evaluated in our model. Swine brucellosis virus (green) infection probability was more sensitive to extreme environmental conditions when compared to pseudorabies virus (brown). For both pseudorabies virus and swine brucellosis, species richness had a significant negative relationship with infection probability. Age had similar mean importance for both pathogens, although there is greater uncertainty for swine brucellosis. For both pathogens, females had a higher probability of infection relative to males. Probability of wild pig occurrence, which is a proxy for wild pig density, was positively associated with true prevalence for both pathogens.

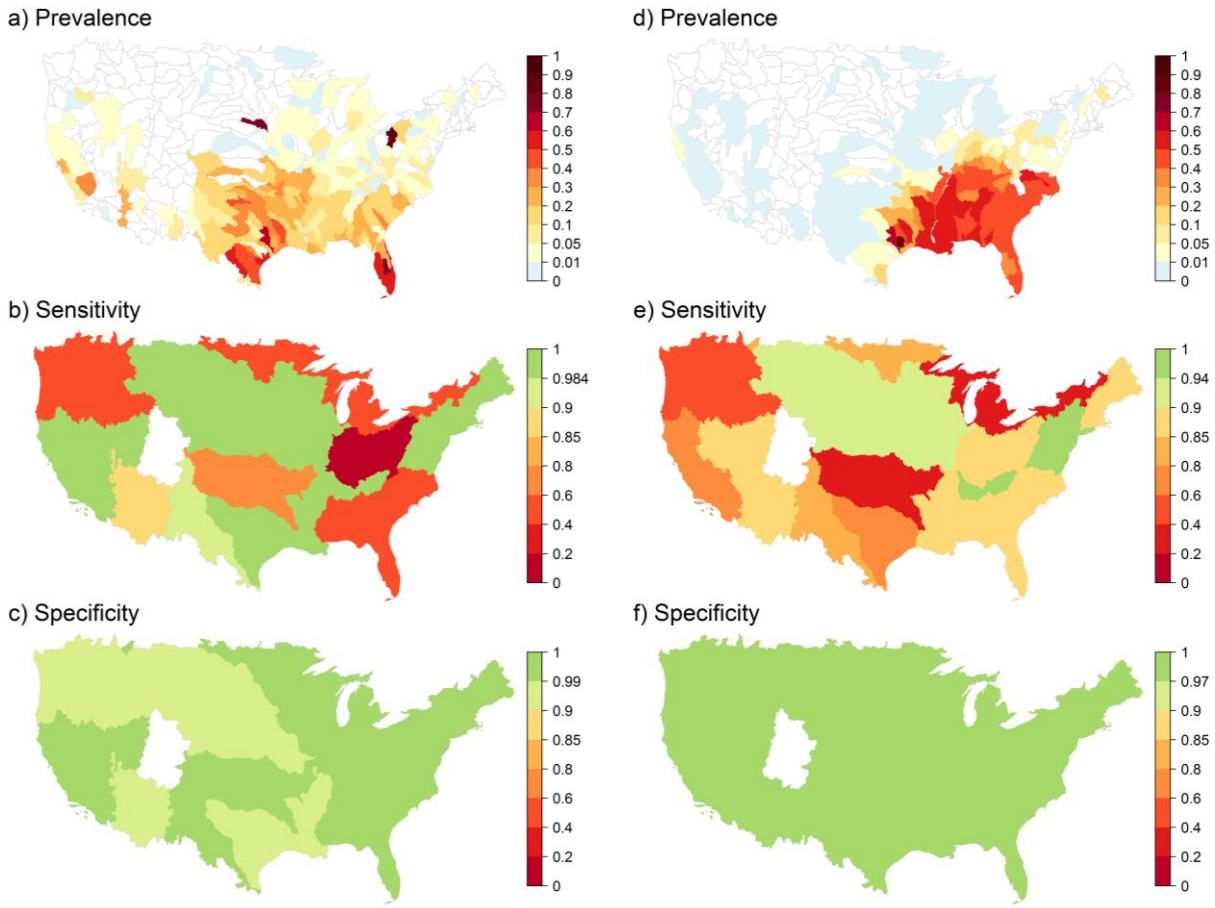


Figure 3.5. Spatial distribution of derived true prevalence, true positive detection (sensitivity), and true negative detection (specificity) for pseudorabies virus (panels a, b, c) and swine brucellosis (panels d, e, f). Derived true prevalence (panels a and d) is shown as the median posterior prevalence at the hydrologic basin scale (HUC6). Blue indicates derived true prevalence that is below 0.01, and white are subbasins without data. True positive and true negative detection probabilities are at the hydrologic region scale (HUC2). Green indicates true positive or true negative detection rates greater than 0.90, and the darkest green color indicates values greater than or equal to the diagnostic test for each pathogen. True negative detection rates were generally closer to diagnostic test values for both pathogens than true positive detection rates among hydrologic regions. Derived true prevalence for both pathogens indicated heterogeneity with pseudorabies virus having greater heterogeneity when compared to swine brucellosis. There was relatively less heterogeneity in the distribution of true negative detection rates.

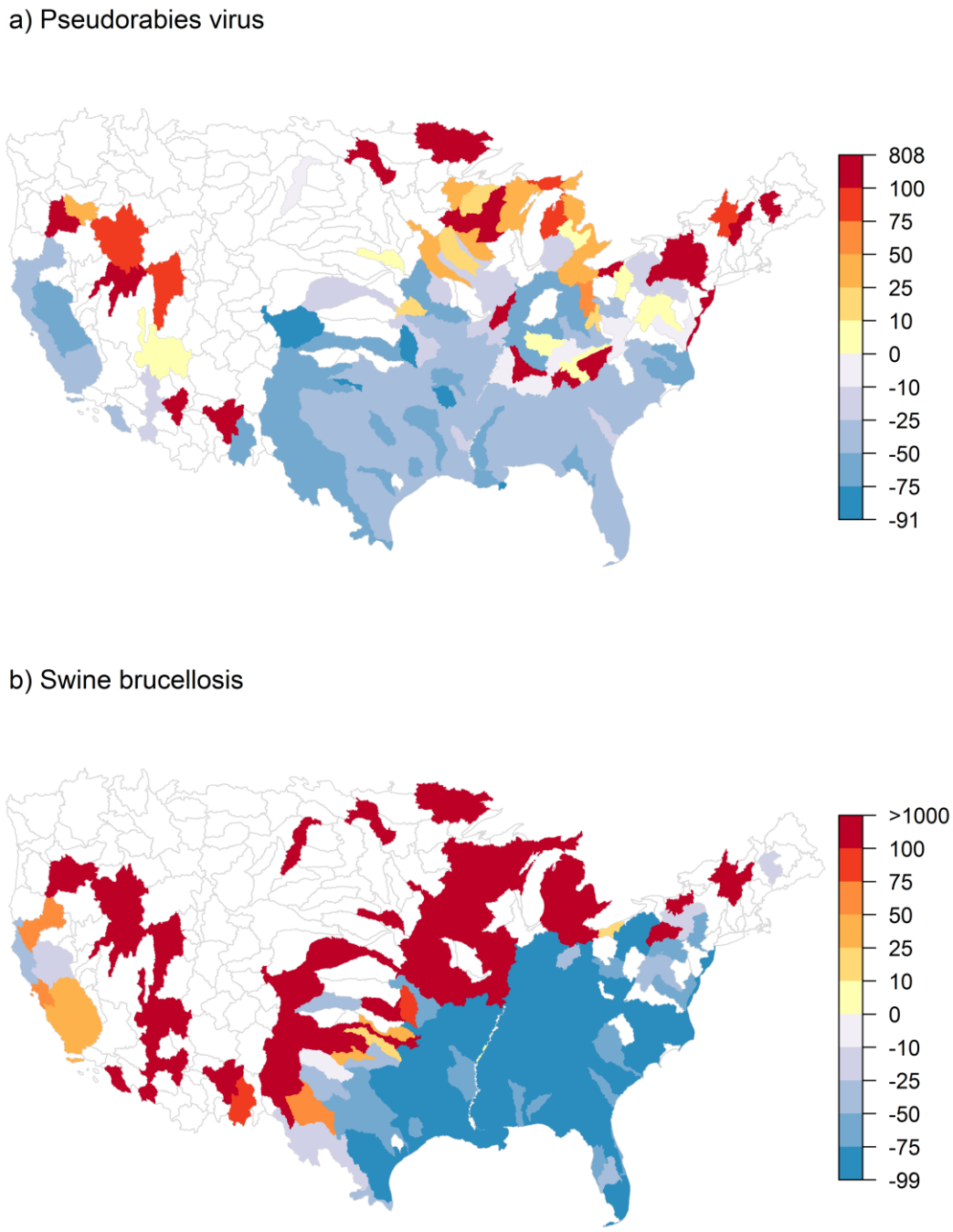


Figure 3.6. Relative bias (%) of observed apparent prevalence (π_{obs_j}) when compared to derived true prevalence ($\hat{\pi}_{true_j}$) for pseudorabies virus and swine brucellosis. Observed apparent prevalence was generally biased high when compared to derived true prevalence along the edge of the distribution of feral swine for both pathogens.

REFERENCES

Acevedo, P., M. A. Escudero, R. Muńoz, and C. Gortázar. 2006. Factors affecting wild boar abundance across an environmental gradient in Spain. Acta Theriologica **51**:327-336.

Banks, M., L. S. Monsalve Torraca, A. G. Greenwood, and D. C. Taylor. 1999. Aujeszky's Disease in Captive Bears. Veterinary Record **145**:362-365.

Barr, S. C., B. E. Eilts, A. F. Roy, and R. Miller. 1986. Brucella-Suis Biotype-1 Infection in a Dog. Journal of the American Veterinary Medical Association **189**:686-687.

Begon, M., S. M. Hazel, D. Baxby, K. Bown, R. Cavanagh, J. Chantrey, T. Jones, and M. Bennett. 1999. Transmission dynamics of a zoonotic pathogen within and between wildlife host species. Proceedings of the Royal Society of London B: Biological Sciences **266**:1939-1945.

Bevins, S. N., K. Pedersen, M. W. Lutman, T. Gidlewski, and T. J. Deliberto. 2014. Consequences Associated with the Recent Range Expansion of Nonnative Feral Swine. BioScience **64**:291-299.

Branscum, A., I. Gardner, and W. Johnson. 2005. Estimation of diagnostic-test sensitivity and specificity through Bayesian modeling. Preventive veterinary medicine **68**:145-163.

Brook, R. K., and F. M. van Beest. 2014. Feral wild boar distribution and perceptions of risk on the central Canadian prairies. Wildlife Society Bulletin **38**:486-494.

Brooks, S. P., and A. Gelman. 1998. General methods for monitoring convergence of iterative simulations. Journal of computational and graphical statistics **7**:434-455.

Brown, J. H. 1984. On the relationship between abundance and distribution of species. The american naturalist **124**:255-279.

Buskirk, J. V., and R. S. Ostfeld. 1998. Habitat heterogeneity, dispersal, and local risk of exposure to Lyme disease. Ecological Applications **8**:365-378.

Caruso, C., A. Dondo, F. Cerutti, L. Masoero, A. Rosamilia, S. Zoppi, V. D'Errico, C. Grattarola, P. L. Acutis, and S. Peletto. 2014. Aujeszky's Disease in Red Fox (Vulpes Vulpes): Phylogenetic Analysis Unravels an Unexpected Epidemiologic Link. Journal of Wildlife Diseases **50**:707-710.

Choquenot, D., and W. A. Ruscoe. 2003. Landscape complementation and food limitation of large herbivores: habitat- related constraints on the foraging efficiency of wild pigs. Journal of Animal Ecology **72**:14-26.

Christensen, R., W. Johnson, A. Branscum, and T. E. Hanson. 2011. Bayesian ideas and data analysis: an introduction for scientists and statisticians. CRC Press.

Collins, S. L., and S. M. Glenn. 1990. A hierarchical analysis of species' abundance patterns in grassland vegetation. American Naturalist **135**:633-648.

Cook, D. R., and J. W. Noble. 1984. Isolation of Brucella Suis from Cattle. Australian Veterinary Journal **61**:263-264.

Cramer, S. D., G. A. Campbell, B. L. Njaa, S. E. Morgan, S. K. Smith Ii, W. R. McLin Iv, B. W. Brodersen, A. G. Wise, G. Scherba, I. M. Langohr, and R. K. Maes. 2011. Pseudorabies Virus Infection in Oklahoma Hunting Dogs. Journal of Veterinary Diagnostic Investigation **23**:915-923.

Crandell, R. A., R. M. Robinson, and P. G. Hannon. 1986. Pseudorabies Infection in Collared Peccaries (Tayassu-Tajacu). Southwestern Veterinarian **37**:193-195.

Cvetnic, Z., S. Spicic, S. Curic, B. Jukic, M. Lojkic, D. Albert, M. Thiébaud, and B. Garin-Bastuji. 2005. Isolation of Brucella Suis Biovar 3 from Horses on Croatia. Veterinary Record **156**:584-585.

Danilov, P., and D. Panchenko. 2012. Expansion and some ecological features of the wild boar beyond the northern boundary of its historical range in European Russia. Russian journal of ecology **43**:45-51.

de Castro, A. M. M. G., T. Brombila, J. G. Bersano, H. S. Soares, S. O. de Souza Silva, A. H. H. Minervino, R. A. Ogata, S. M. Gennari, and L. J. Richtzenhain. 2014. Swine Infectious Agents in Tayassu Pecari and Pecari Tajacu Tissue Samples from Brazil. Journal of Wildlife Diseases **50**:205-209.

Denwood, M. J. 2016. runjags: An R package providing interface utilities, model templates, parallel computing methods and additional distributions for MCMC models in JAGS. Journal of Statistical Software **71**:1-25.

Drew, M. L., D. A. Jessup, A. A. Burr, and C. E. Franti. 1992. Serologic Survey for Brucellosis in Feral Swine, Wild Ruminants, and Black Bear of California, 1977 to 1989. Journal of Wildlife Diseases **28**:355-363.

Elton, C. S. 2000. The ecology of invasions by animals and plants. University of Chicago Press, Chicago, IL.

Ewalt, D. R., J. B. Payeur, J. C. Rhyan, and P. L. Geer. 1997. Brucella suis biovar 1 in naturally infected cattle: a bacteriological, serological, and histological study. Journal of Veterinary Re- search **53**:405-410.

Fort, M., V. Baldone, L. Fuchs, H. Giménez, M. Rojas, J. D. Breccia, and J. Oyhenart. 2012. Experimental Infection of Rabbits (Oryctolagus Cuniculus) with Brucella Suis Biovar 1 Isolated from Wild Hares (Lepus Europaeus). Veterinary microbiology **156**:439-442.

Fraser, D., and P. Phillips. 1989. Lethargy and low water intake by sows during early lactation: a cause of low piglet weight gains and survival? Applied Animal Behaviour Science **24**:13-22.

Geisser, H., and H. U. Reyer. 2005. The influence of food and temperature on population density of wild boar Sus scrofa in the Thurgau (Switzerland). Journal of Zoology **267**:89-96.

Gelman, A., J. B. Carlin, H. S. Stern, and D. B. Rubin. 2014. Bayesian data analysis. Third edition. CRC Press, Boca Raton, FL.

Gelman, A., and J. Hill. 2006. Data analysis using regression and multilevel/hierarchical models. Cambridge University Press, New York, NY.

Gelman, A., and D. B. Rubin. 1992. Inference from iterative simulation using multiple sequences. Statistical science **7**:457-472.

Giraudoux, P., P. Craig, P. Delattre, G. Bao, B. Bartholomot, S. Harraga, J.-P. Quéré, F. Raoul, Y. Wang, and D. Shi. 2003. Interactions between landscape changes and host communities can regulate Echinococcus multilocularis transmission. Parasitology **127**:S121-S131.

Goyal, S. M., R. Drolet, and P. King. 1986. Pseudorabies in Free-Ranging Raccoons. Journal of the American Veterinary Medical Association **189**:1163-1164.

Gyuranecz, M., K. Erdélyi, L. Makrai, L. Fodor, B. Szépe, Á. R. Mészáros, A. Dán, L. Dencso, E. Fassang, and L. Szeredi. 2011. Brucellosis of the European Brown Hare (Lepus Europaeus). Journal of Comparative Pathology **145**:1-5.

Hantsch, L., U. Braun, M. Scherer-Lorenzen, and H. Bruelheide. 2013. Species richness and species identity effects on occurrence of foliar fungal pathogens in a tree diversity experiment. Ecosphere **4**:1-12.

Hara, M., T. Shimizu, S. Nemoto, M. Fukuyama, T. Ikeda, A. Kiuchi, K. Tabuchi, Y. Nomura, K. Shirota, Y. Une, and R. Ishizaki. 1991. A Natural Case of Aujeszky's Disease in the Cat in Japan. Journal of Veterinary Medical Science **53**:947-949.

Heidelberger, P., and P. D. Welch. 1983. Simulation run length control in the presence of an initial transient. Operations Research **31**:1109-1144.

Hellmann, E., and H. U. Sprenger. 1978. Brucella Suis Infections in the Dog. Berliner und Munchener Tierarztliche Wochenschrift **91**:385-387.

Hijmans, R. J., S. E. Cameron, J. L. Parra, P. G. Jones, and A. Jarvis. 2005. Very high resolution interpolated climate surfaces for global land areas. International journal of climatology **25**:1965-1978.

Honda, T. 2009. Environmental factors affecting the distribution of the wild boar, sika deer, Asiatic black bear and Japanese macaque in central Japan, with implications for human-wildlife conflict. Mammal Study **34**:107-116.

Huang, Z., F. Van Langevelde, A. Estrada-Peña, G. Suzán, and W. De Boer. 2016. The diversity–disease relationship: evidence for and criticisms of the dilution effect. Parasitology **143**:1075-1086.

Huang, Z., C. Xu, F. Van Langevelde, H. Prins, K. B. Jebara, and W. De Boer. 2014. Dilution effect and identity effect by wildlife in the persistence and recurrence of bovine tuberculosis. Parasitology **141**:981-987.

Huang, Z. Y., W. F. de Boer, F. van Langevelde, V. Olson, T. M. Blackburn, and H. H. Prins. 2013a. Species' life-history traits explain interspecific variation in reservoir competence: a possible mechanism underlying the dilution effect. PLoS One **8**:e54341.

Huang, Z. Y., W. F. de Boer, F. van Langevelde, C. Xu, K. B. Jebara, F. Berlingieri, and H. H. Prins. 2013b. Dilution effect in bovine tuberculosis: risk factors for regional disease occurrence in Africa. Proceedings of the Royal Society of London B: Biological Sciences **280**:20130624.

Jedrzejewska, B., W. Jedrzejewski, A. N. Bunevich, L. Milkowski, and Z. A. Krasinski. 1997. Factors shaping population densities and increase rates of ungulates in Bialowieza Primeval Forest (Poland and Belarus) in the 19th and 20th centuries. Zeszyty Problemowe Postepow Nauk Rolniczych **42**:399-451.

Jennelle, C. S., E. G. Cooch, M. J. Conroy, and J. C. Senar. 2007. State-specific detection probabilities and disease prevalence. Ecological Applications **17**:154-167.

Jin, M. L., H. C. Chen, C. Liu, M. L. Luo, M. Z. Wu, D. Z. Guo, and C. F. Ye. 1992. Isolation of Pseudorabies Virus from Dairy Calf and Buffalo. Journal of Huazhong Agricultural University **11**:186-188.

Johnson, P. T., R. S. Ostfeld, and F. Keesing. 2015. Frontiers in research on biodiversity and disease. Ecology letters **18**:1119-1133.

Keesing, F., L. K. Belden, P. Daszak, A. Dobson, C. D. Harvell, R. D. Holt, P. Hudson, A. Jolles, K. E. Jones, and C. E. Mitchell. 2010. Impacts of biodiversity on the emergence and transmission of infectious diseases. Nature **468**:647-652.

Keesing, F., R. D. Holt, and R. S. Ostfeld. 2006. Effects of species diversity on disease risk. Ecology letters **9**:485-498.

Kimman, T. G., G. J. Binkhorst, T. S. van den Ingh, J. M. Pol, A. L. Gielkens, and M. E. Roelvink. 1991. Aujeszky's Disease in Horses Fulfils Koch's Postulates. Veterinary Record **128**:103-106.

Kimman, T. G., and J. T. Van Oirschot. 1986. Pathology of Aujeszky's Disease in Mink. Veterinary Pathology **23**:303-309.

Knops, J. M., D. Tilman, d. N. Haddad, S. Naeem, C. Mitchell, J. Haarstad, M. Ritchie, K. Howe, P. Reich, and E. Siemann. 1999. Effects of plant species richness on invasion dynamics, disease outbreaks, insect abundances and diversity. Ecology letters **2**:286-293.

Kormendy, B., and G. Nagy. 1982. The Supposed Involvement of Dogs Carrying Brucella-Suis in the Spread of Swine Brucellosis. Acta veterinaria academiae scientiarum hungaricae **30**:3-7.

Lahoz- Monfort, J. J., G. Guillera- Arroita, and B. A. Wintle. 2014. Imperfect detection impacts the performance of species distribution models. Global Ecology and Biogeography **23**:504-515.

Leiser, O. P., J. L. Corn, B. S. Schmit, P. S. Keim, and J. T. Foster. 2013. Feral swine brucellosis in the United States and prospective genomic techniques for disease epidemiology. Veterinary microbiology **166**:1-10.

Lewis, J. S., M. L. Farnsworth, C. L. Burdett, D. M. Theobald, M. Gray, and R. S. Miller. 2017. Biotic and abiotic factors predicting the global distribution and population density of an invasive large mammal. Scientific Reports **7**:44152.

Lord, V. R., and R. D. Lord. 1991. Brucella suis infections in collared peccaries in Venezuela. Journal of Wildlife Diseases **27**:477-481.

Lucero, N. E., S. M. Ayala, G. J. Escobar, and N. R. Jacob. 2008. Brucella Isolated in Humans and Animals in Latin America from 1968 to 2006. Epidemiology and Infection **136**:496-503.

MacKenzie, D. I., J. D. Nichols, J. E. Hines, M. G. Knutson, and A. B. Franklin. 2003. Estimating site occupancy, colonization, and local extinction when a species is detected imperfectly. Ecology **84**:2200-2207.

Marcaccini, A., M. López Peña, M. I. Quiroga, R. Bermúdez, J. M. Nieto, and N. Alemañ. 2008. Pseudorabies Virus Infection in Mink: A Host-Specific Pathogenesis. Veterinary Immunology and Immunopathology **124**:264-273.

Matschke, G. H. 1967. Aging European wild hogs by dentition. The Journal of Wildlife Management **31**:109-113.

Mayor, P., Y. Le Pendu, D. A. Guimaraes, J. V. da Silva, H. L. Tavares, M. Tello, W. Pereira, M. Lopez-Béjar, and F. Jori. 2006. A health evaluation in a colony of captive collared peccaries (Tayassu tajacu) in the eastern Amazon. Research in veterinary science **81**:246-253.

McClintock, B. T., J. D. Nichols, L. L. Bailey, D. I. MacKenzie, W. Kendall, and A. B. Franklin. 2010. Seeking a second opinion: uncertainty in disease ecology. Ecology letters **13**:659-674.

McClure, M. L., C. L. Burdett, M. L. Farnsworth, M. Lutman, D. M. Theobald, P. D. Riggs, D. A. Grear, and R. S. Miller. 2015. Modeling and mapping the probability of occurrence of invasive wild pigs across the contiguous United States. In review.

McLean, R. G., J. B. Katz, D. S. Maehr, C. B. Cropp, L. J. Kirk, A. J. McKeirnan, and J. F. Evermann. 1994. Isolation of Pseudorabies (Aujeszky's Disease) Virus from a Floria Panther. Journal of Wildlife Diseases **30**:180-184.

Melis, C., P. A. Szafrańska, B. Jędrzejewska, and K. Bartoń. 2006. Biogeographical variation in the population density of wild boar (Sus scrofa) in western Eurasia. Journal of Biogeography **33**:803-811.

Miller, D. A., J. D. Nichols, B. T. McClintock, E. H. C. Grant, L. L. Bailey, and L. A. Weir. 2011. Improving occupancy estimation when two types of observational error occur: non- detection and species misidentification. Ecology **92**:1422-1428.

Miller, R. S., M. L. Farnsworth, and J. L. Malmberg. 2013. Diseases at the Livestock-Wildlife Interface: Status, Challenges, and Opportunities in the United States. Preventive veterinary medicine **110**:119-132.

Miller, R. S., S. J. Sweeney, C. Slootmaker, D. A. Grear, P. D. Salvo, D. Kiser, and S. A. Shwiff. 2017. Cross-species transmission potential between wild pigs, livestock, poultry, wildlife, and humans: implications for disease risk management in North America. Scientific Reports **In Submission**.

Minasny, B., and A. B. McBratney. 2006. A conditioned Latin hypercube method for sampling in the presence of ancillary information. Computers & Geosciences **32**:1378-1388.

Mitchell, C. E., D. Tilman, and J. V. Groth. 2002. Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. Ecology **83**:1713-1726.

Mocsári, E., J. Szolnoki, R. Glávits, and L. Zsák. 1989. Horizontal Transmission of Aujeszky's Disease Virus from Sheep to Pigs. Veterinary microbiology **19**:245-252.

Mocsári, E., C. Tóth, M. Meder, E. Sághy, and R. Glávits. 1987. Aujeszky's Disease of Sheep: Experimental Studies on the Excretion and Horizontal Transmission of the Virus. Veterinary microbiology **13**:353-359.

Moore, S. M., and E. T. Borer. 2012. The influence of host diversity and composition on epidemiological patterns at multiple spatial scales. Ecology **93**:1095-1105.

Mor, S. M., A. K. Wiethoelter, A. Lee, B. Moloney, D. R. James, and R. Malik. 2016. Emergence of Brucella Suis in Dogs in New South Wales, Australia: Clinical Findings and Implications for Zoonotic Transmission. BMC Veterinary Research **12**:199.

Müller, T., E. Hahn, F. Tottewitz, M. Kramer, B. Klupp, T. Mettenleiter, and C. Freuling. 2011. Pseudorabies virus in wild swine: a global perspective. Archives of Virology **156**:1691-1705.

Müller, T., J. Teuffert, K. Ziedler, C. Possardt, M. Kramer, C. Staubach, and F. Conraths. 1998. Pseudorabies in the European wild boar from eastern Germany. Journal of Wildlife Diseases **34**:251-258.

Norton, J. H., and A. D. Thomas. 1979. Brucella Suis Infection in Pregnant Cattle. Australian Veterinary Journal **55**:525-527.

Odum, E. P., H. T. Odum, and J. Andrews. 2001. Fundamentals of ecology, 3rd Edition, Saunders, Philadelphia

Olsen, S., B. Garin-Bastuji, J. Blasco, A. Nicola, and L. Samartino. 2011. Swine Brucellosis.*in* J. J. Zimmerman, L. A. Karriker, A. Ramirez, K. J. Schwartz, and G. W. Stevenson, editors. Diseases of Swine, 10th Edition,. Blackwell, USA.

Ostfeld, R. S., and F. Keesing. 2012. Effects of host diversity on infectious disease. Annual Review of Ecology, Evolution, and Systematics **43**:157-182.

Palic, D. 1985. Experimental Infection of Red Foxes with Pseudorabies Virus.*in* International Conference on Wildlife Diseases. Statens Veterinaermedicinska Anstalt, Uppsala, Sweden.

Paolicchi, F. A., H. R. Terzolo, and C. M. Campero. 1993. Isolation of Brucella-Suis from the Semen of a Ram. Veterinary Record **132**:67-67.

Patterson, B., G. Ceballos, W. Sechrest, M. Tognelli, T. Brooks, L. Luna, P. Ortega, I. Salazar, and B. Young. 2007. Digital distribution maps of the mammals of the Western Hemisphere.*in* NatureServe, editor. NatureServe, Arlington, Virginia, USA.

Pautasso, M., O. Holdenrieder, and J. Stenlid. 2005. Susceptibility to fungal pathogens of forests differing in tree diversity. Pages 263-289 *in* M. Scherer-Lorenzen, C. Körner, and E.-D. Schulze, editors. Forest diversity and function. Springer Berlin, Germany.

Pedersen, K., S. N. Bevins, J. A. Baroch, J. C. Cumbee Jr, S. C. Chandler, B. S. Woodruff, T. T. Bigelow, and T. J. DeLiberto. 2013. Pseudorabies in feral swine in the United States, 2009-2012. Journal of Wildlife Diseases **49**:709-713.

Pedersen, K., S. N. Bevins, B. S. Schmit, M. W. Lutman, M. P. Milleson, C. T. Turnage, T. T. Bigelow, and T. J. Deliberto. 2012. Apparent prevalence of swine brucellosis in feral swine in the United States. Human – Wildlife Interactions **6**:38-47.

Pedersen, K., R. S. Miller, T. D. Anderson, K. L. Pabilonia, J. R. Lewis, R. L. Mihalco, C. Gortázar, and T. Gidlewski. 2017. Limited Antibody Evidence of Exposure to Mycobacterium bovis in Feral Swine (Sus scrofa) in the USA. Journal of Wildlife Diseases **53**:30-36.

Pepin, K. M., S. L. Kay, B. D. Golas, S. S. Shriner, A. T. Gilbert, R. S. Miller, A. L. Graham, S. Riley, P. C. Cross, and M. D. Samuel. 2017. Inferring infection hazard in wildlife

populations by linking data across individual and population scales. Ecology letters **20**:275-292.

Peterson, A. T., M. Papes, and D. A. Kluza. 2009. Predicting the potential invasive distributions of four alien plant species in North America. Weed Science **51**:863-868.

Pirtle, E. C., M. E. Roelke, and J. Brady. 1986. Antibodies against Pseudorabies Virus in the Serum of a Florida Black Bear Cub. Journal of the American Veterinary Medical Association **189**:1164.

Pirtle, E. C., J. M. Sacks, V. F. Nettles, and I. Rollor. 1989. Prevalence and transmission of pseudorabies virus in an isolated population of feral swine. Journal of Wildlife Diseases **25**:605-607.

Platt, K. B., D. L. Graham, and R. A. Faaborg. 1983. Pseudorabies: Experimental Studies in Raccoons (Procyon Lotor) Wit Different Virus Strains. Journal of Wildlife Diseases **19**:297-301.

Plummer, M. 2014. JAGS: A program for analysis of Bayesian graphical models using Gibbs sampling, 2003. URL http://sourceforge. net/projects/mcmc-jags.

Plummer, M., N. Best, K. Cowles, and K. Vines. 2006. CODA: convergence diagnosis and output analysis for MCMC. R news **6**:7-11.

Porter, W. P., and D. M. Gates. 1969. Thermodynamic equilibria of animals with environment. Ecological monographs **39**:227-244.

Portugal, M. A., A. Nesti, W. Giorgi, E. N. França, and B. S. de Oliveira Jr. 1971. Brucellosis in Equins Caused by Brucella Suis. Arquivos do Instituto Biologico **38**:125-132.

Power, E. P., M. O'Connor, W. J. Donnelly, and C. E. Dolan. 1990. Aujeszky's Disease in a Cow. Veterinary Record **126**:13-15.

Quiroga, M. I., M. López-Peña, S. Vázquez, and J. M. Nieto. 1997. Distribution of Aujeszky's Disease Virus in Experimentally Infected Mink (Mustela Vison). Dtw. Deutsche Tierärztliche Wochenschrift **104**:147-150.

R Development Core Team. 2011. R package version 3.1-97. R Foundation for Statistical Computing, Vienna. R Foundation for Statistical Computing, Vienna, Austria.

Rademacher, G., G. Dirksen, and O. C. Straub. 1991. Outbreaks of Aujeszkys Disease in Cattle Herds in Bavaria. Tierarztliche Umschau **46**:513-520.

Ramamoorthy, S., M. Woldemeskel, A. Ligett, R. Snider, R. Cobb, and S. Rajeev. 2011. Brucella Suis Infection in Dogs, Georgia, USA. Emerging Infectious Diseases **17**:2386-2387.

Raymond, J. T., R. G. Gillespie, M. Woodruff, and E. B. Janovitz. 1997. Pseudorabies in Captive Coyotes. Journal of Wildlife Diseases **33**:916-918.

Reddy, P. R., and P. V. Rao. 1984. A Note on Isolation of Brucella Suis from Foetus of a She Goat. Kerala journal of veterinary science **15**:173-174.

Romero, C. H., P. N. Meade, J. E. Shultz, H. Y. Chung, E. P. Gibbs, E. C. Hahn, and G. Lollis. 2001. Venereal transmission of pseudorabies viruses indigenous to feral swine. Journal of Wildlife Diseases **37**:289-296.

Royle, J. A., and R. M. Dorazio. 2008. Hierarchical modeling and inference in ecology: the analysis of data from populations, metapopulations and communities. Academic Press, San Diego, CA.

Royle, J. A., and M. Kéry. 2007. A Bayesian state- space formulation of dynamic occupancy models. Ecology **88**:1813-1823.

Royle, J. A., and W. A. Link. 2006. Generalized site occupancy models allowing for false positive and false negative errors. Ecology **87**:835-841.

Ruiz-Fons, F., D. Vidal, J. Vicente, P. Acevedo, I. G. Fernández-de-Mera, V. Montoro, and C. Gortázar. 2008. Epidemiological risk factors of Aujeszky's disease in wild boars (Sus scrofa) and domestic pigs in Spain. European Journal of Wildlife Research **54**:549-555.

Sakkubai, P. R., and S. C. Ramachandran. 1992. Pseudorabies (Aujeszky's Disease) in Horses. Centaur (Mylapore) **8**:75-78.

Salkeld, D. J., K. A. Padgett, and J. H. Jones. 2013. A meta- analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. Ecology letters **16**:679-686.

Schmidt, S. P., and J. P. Kluge. 1992. The Anatomical Location of Neural Structures Most Optimally Sampled for Pseudorabies (Aujeszky's Disease) in Sheep. Journal of Veterinary Diagnostic Investigation **4**:206-208.

Schmidt, S. P., E. C. Pirtle, W. A. Hagemoser, and D. P. Wages. 1987. A Necrotizing Pneumonia in Lambs Caused by Pseudorabies Virus Aujeszky's Disease Virus. Canadian Journal of Veterinary Research **51**:145-149.

Schultze, A. E., R. K. Maes, and D. C. Taylor. 1986. Pseudorabies and Volvulus in Black Bear. Journal of the American Veterinary Medical Association **189**:1165-1166.

Shannon, C. E. 1949. W. Weaver The mathematical theory of communication. Urbana: University of Illinois Press **29**.

Smith, G. 2012. Preferential sexual transmission of pseudorabies virus in feral swine populations may not account for observed seroprevalence in the USA. Preventive veterinary medicine **103**:145-156.

Snow, N. P., M. A. Jarzyna, and K. C. VerCauteren. 2016. Interpreting and Predicting the Spread of Invasive Wild Pigs. Journal of Applied Ecology.

Stallknecht, D. 2007. Impediments to wildlife disease surveillance, research, and diagnostics. Pages 445-461 Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances and Consequences of Cross-Species Transmission. Springer.

Steinrigl, A., S. Revilla-Fernández, J. Kolodziejek, E. Wodak, Z. Bagó, N. Nowotny, F. Schmoll, and J. Köfer. 2012. Detection and Molecular Characterization of Suid Herpesvirus Type 1 in Austrian Wild Boar and Hunting Dogs. Veterinary microbiology **157**:276-284.

Stěrba, F. 1982. Pathomorphological Changes in Brucellosis in Hares. Veterinarni Medicina **27**:437-448.

Stěrba, F. 1984. Brucellosis in Rabbits Induced by an Experimental Infection with Brucella Suis Germs. Veterinarni Medicina **29**:231-244.

Szyfres, B., J. González Tomé, and T. Palacio Mendieta. 1968. Isolation of Brucella Suis from the European Hare (Lepus Auropaeus) in Argentinea. Boletin de la Oficina Sanitaria Panamericana **65**:441-445.

Tae, H., S. Shallom, R. Settlage, G. N. Hawkins, L. G. Adams, and H. R. Garner. 2012. Complete Genome Sequence of Brucella Suis Vbi22, Isolated from Bovine Milk. Journal of Bacteriology **194**:910.

Thanappa, P. M., S. Nedunchelliyan, and N. Raghavan. 1990. Brucellosis in a Dog Caused by Brucella Suis Biovar I in Madras. Cheiron **19**:97-98.

Thawley, D. G., and J. C. Wright. 1982. Pseudorabies Virus Infection in Raccoons: A Review. Journal of Wildlife Diseases **18**:113-116.

Thiry, E., D. Addie, S. Belak, C. Boucraut-Baralon, H. Egberink, T. Frymus, T. Gruffydd-Jones, K. Hartmann, M. J. Hosie, A. Lloret, H. Lutz, F. Marsilio, K. Mostl, M. G. Pennisi, A. D. Radford, U. Truyen, and M. C. Horzinek. 2013. Aujeszky's Disease/Pseudorabies in Cats: Abcd Guidelines on Prevention and Management. Journal of Feline Medicine and Surgery **15**:555-556.

Thompson, P., J. Brewer, and E. Brewer. 1996. Swine care handbook. National Pork Board, Des Moines **34**.

Thorpe, B. D., R. Sidwell, D. Johnson, K. Smart, and D. Parker. 1965. Tularemia in the wildlife and livestock of the Great Salt Lake Desert Region, 1951 through 1964. American Journal of Tropical Medicine and Hygiene **14**:622-637.

Tworek, R., and D. Serokowa. 1956. [Isolation of Brucella Suis from a Hare]. Przegląd epidemiologiczny **10**:369-370.

USGS. 2011. Watershed Boundary Dataset.*in* U.S. Geological Survey Gap Analysis Program (USGS GAP), editor., National Land Cover v2.

van den Ingh, T. S. G. A. M., G. J. Binkhorst, T. G. Kimman, J. Vreeswijk, J. M. A. Pol, and J. T. van Oirschot. 1990. Aujeszky's Disease in a Horse. Journal of Veterinary Medicine, Series B **37**:532-538.

Verpoest, S., A. B. Cay, O. Bertrand, M. Saulmont, and N. De Regge. 2014. Isolation and Characterization of Pseudorabies Virus from a Wolf (Canis Lupus) from Belgium. European Journal of Wildlife Research **60**:149-153.

Xiao, F. 1985. An outbreak of pseudorabies on a raccoon farm. Chinese Journal of Veterinary Science and Technology (Zhongguo Shouyi Keji) **5**:24-25.

Yildirim, S., C. Özkan, Z. Yener, M. Çetin, and S. Kozat. 2017. Immunohistochemical Diagnosis of Pseudorabies (Aujeszky's Disease) in a Cow in Van, Turkey. Kafkas Universitesi Veteriner Fakultesi Dergisi **23**:173-176.

Young, H., R. H. Griffin, C. L. Wood, and C. L. Nunn. 2013. Does habitat disturbance increase infectious disease risk for primates? Ecology letters **16**:656-663.

Zanin, E., I. Capua, C. Casaccia, A. Zuin, and A. Moresco. 1997. Isolation and Characterization of Aujeszky's Disease Virus in Captive Brown Bears from Italy. Journal of Wildlife Diseases **33**:632-634.

Zhang, L., C. Zhong, J. Wang, Z. Lu, L. Liu, W. Yang, and Y. Lyu. 2015. Pathogenesis of Natural and Experimental Pseudorabies Virus Infections in Dogs Herpes Viruses. Virology Journal **12**:44.

CHAPTER 4

NATIVE AND INVASIVE POPULATIONS DEMONSTRATE DIFFERENCES IN LIFE HISTORY STRATEGIES THAT BUFFER ENVIRONMENTAL CONDITIONS

INTRODUCTION

Transient processes play a key role in our ecological understanding (Hastings 2004) and often explain higher order processes of energy flux and loss that drive ecological systems (Rip and McCann 2011, Gellner et al. 2016). Short term transient population dynamics are common in vertebrates, particularly invasive vertebrates, and can fundamentally influence the ability of a species to colonize new areas (McMahon and Metcalf 2008, Iles et al. 2016). A central theme in ecology that also bears on transient dynamics is the impact of variability in environmental conditions on population dynamics (Lande et al. 2003). Transient dynamics by their nature are directly influenced by deviations in population structure (e.g. age structure) from equilibrium as mediated by vital rates. These age structure deviations that manifest as transient dynamics are driven by both exogenous and endogenous factors, yet the linkage between transient population dynamics and environmental drivers are rarely studied. These relationships are fundamental to an improved ecological understanding of transient population dynamics, which is particularly relevant to invasive vertebrates and their management.

Globally wild pigs are one of the most successful invasive mammal species and the most abundant free-ranging, exotic ungulate in the United States (Lowe et al. 2000, Bevins et al. 2014, Lewis et al. 2017). Wild pigs are found on every content except Antarctica, and wild pig populations are both extending their native range (Massei et al. 2015, Vetter et al. 2015) and increasing their invasive range (McClure et al. 2015, Vetter et al. 2015). Their success as an

invasive species has been hypothesized to be a function of their generalist traits and their high fecundity (Bieber and Ruf 2005). On their native range, wild pigs evolved as pulsed resource consumers in temperate forests of Europe where hard mast species are common (Gamelon et al. 2013), and their population dynamics appear to rapidly respond to changes in environmental conditions (Bieber and Ruf 2005, Focardi et al. 2008, Sabrina et al. 2009). The ability to rapidly respond over a short period to changing environmental conditions may be an important trait facilitating invasion success (Sol and Lefebvre 2000).

The success of wild pigs can be unpacked into several traits that have been hypothesized to interact with environmental drivers to make wild pigs a particularly adaptable species on their native range. Reproductive maturity is primarily linked to body weight allowing juveniles to take advantage of pulsed forage resources, often reproducing at less than one year of age (Bieber and Ruf 2005, Geisser and Reyer 2005, Csányi 2014) resulting in increased population growth when resources are plentiful. High fecundity (Bywater et al. 2010) and short gestation periods relative to their body size (Gethöffer et al. 2007) in combination with generally high juvenile survival as a result of close knit matrilineal family groups has been hypothesized to buffer populations from changing environmental conditions contributing to rapid population growth (Bieber and Ruf 2005, Focardi et al. 2008). Winter severity has been proposed to regulate populations by reducing the ability of wild pigs to root for forage resources acting as a population regulator (Bieber and Ruf 2005, McClure et al. 2015). Taken in total, these characteristics support the idea that wild pigs are highly fecund generalists that can take advantage of plentiful pulsed natural forage resources that often occur over short periods on their native range. Increasing populations of wild boar in Europe have been hypothesized to be a result of increasing annual temperature due to climate change resulting in lower periodicity and increased forage production that has in

turn increased wild boar fecundity (Vetter et al. 2015, Frauendorf et al. 2016). However, testing of these hypotheses has relied on phenomenological observational studies, and they have not been investigated for invasive populations. The effect of environmental drivers are generally thought to be increased for invasive populations and may be important over short-time periods important for species establishment (Sol and Lefebvre 2000).

Asymptotic population dynamics that have traditionally been used to investigate the influence of environmental conditions on age structure and vital rates assume stable population growth and stable age structure (Caswell 2014). However, when populations experience heterogeneous environments and extrinsic disruptions to age structure, the assumptions of asymptotic population dynamics are rarely met (Bierzychudek 1999, Hastings 2004, Ezard et al. 2010). Recent work to disentangle asymptotic and transient dynamics has found that transients can account for >50% of overall variation in population dynamics within and among populations (Ellis and Crone 2013, Koons et al. 2016, McDonald et al. 2016). In addition there are gaps in our current understanding of the relationship between vital rates, age structure deviations and population dynamics over short time horizons (Koons et al. 2016) that may reduce or increase the effect of changes in environmental conditions (Gaillard and Yoccoz 2003). In the case of invasive species that are pioneering previously unoccupied habitats, the population is inherently in an unstable demographic condition but life-history strategies may be flexible enough to absorb deviations from equilibrium state (Koons et al. 2005, Iles et al. 2016). A focus on transient population dynamics allows for the study of changes in demographic structure in non-equilibrium populations and their relative contribution to overall population dynamics. However, analyses of transient population dynamics are rarely applied to mammal populations, particularly to understand the effect of environmental conditions on populations (Koons et al.

2016, McDonald et al. 2016) and the implications for invasive mammal species such as wild pigs.

Here we use relatively recent methodological advances in the analysis of transient population dynamics to examine how environmental conditions correlate with short-term transient population dynamics, particularly population growth rates (Ellis and Crone 2013, Koons et al. 2016, McDonald et al. 2016). We use a novel database of wild pig matrices describing survival and fecundity for 16 native and invasive populations occurring on four continents to determine indices describing population transient dynamics. Using these data we investigate the vital rates and age classes that contribute most to transient population growth under different environmental conditions experienced by native and invasive populations. Because others have previously found that survival of younger age classes can be important for short-term population growth (Koons et al. 2016) we hypothesized that survival of younger age classes contributes most to transient population growth. Based on the observational studies in the native rage, we also hypothesized that environmental conditions that increase survival then reduce the amount of transient dynamics and that increases in forage availability will increase transient population growth. To evaluate hypothesized differences in invasive and native life-history strategies, we estimated the relative contribution of age structure, survival rates and fecundities to variation in transient population growth. To investigate hypotheses about the relationship between environmental gradients and transient dynamics, we then use maximum likelihood methods to estimate the effect of environmental conditions on transient dynamics for native and invasive populations. We discuss these results in the context of invasive species and describe how these results can be used to better understand population regulators important for determining risk of establishment in new areas and for designing control tools that are effective.

METHODS

Our goal was to evaluate hypotheses about the contribution of environmental conditions to the persistence of transient dynamics; investigate the contribution of survival, fecundity and age structure to transient dynamics; and contrast any differences for native and invasive populations. To accomplish this, first we calculated the ratio of transient growth rate relative to the realized growth rate as a proxy to transient dynamics using demographic matrix models and investigated differences among invasive and native populations in this ratio. Second, we calculated age specific contributions of survival, fecundity and age structure to variation in transient population growth for invasive and native populations to investigate hypotheses about differences in life-history strategies. Lastly, we used our proxy of transient dynamics (the ratio of transient growth rate relative to the realized growth rate) as a response variable in statistical models to investigate hypotheses concerning the effect of environmental variables on the amount of transient dynamics experienced by invasive and native populations.

Demographic matrix model

A standard approach to modeling age structured demographic dynamics is to combine age-specific vital rates, such as survival and fecundity, into a transition matrix that can be used to project age-specific abundances through time and estimate population growth (Caswell 2014). We used a transition matrix in a time-variant population model that is commonly used to model age structured dynamics and project abundance of individuals $n_{i,t}$ expected in age class i at time t,

$$\boldsymbol{n_{t+1}} = \boldsymbol{A n_t} \qquad \text{Eq. 1}$$

where $\boldsymbol{A}$ is a transition matrix containing the vital rates for survival (σ_i) and fecundity (γ_i) for individuals in age class i, and $\boldsymbol{n}$ is the vector of abundances in each age class representing age

structure. When the matrix $\boldsymbol{A}$ is constant in time, the population growth rate (as $t \to \infty$) can be estimated by the dominant eigenvalue (λ_{max}) of the matrix $\boldsymbol{A}$ and the asymptotic stable age structure from the corresponding eigenvector. Populations may not exhibit asymptotic dynamics for a variety of reasons, e.g. fluctuating environmental conditions, harvest, and of particular relevance here, invasion of new habitat. In these situations, the age structure is not at the asymptotic distribution, so the observed population growth rate in each time step can be written as $\lambda_{\ t} = log\left(\sum_i n_{i,t+1} / \sum_i n_{i,t}\right)$. Thus, λ is composed of both asymptotic and transient contributions.

Model for transient response

The transient response, called *reactivity* (Neubert and Caswell 1997, Townley et al. 2007), is the single time-step transient amplification or attenuation of the population due to deviations of $\boldsymbol{n_t}$ from the asymptotic stable age structure of $\boldsymbol{A}$ and can be represented as,

$$reactivity_t = \frac{\sum_i \frac{n_{i,t+1}}{\lambda_{max}}}{\sum_i n_{i,t}} \qquad \text{Eq. 2}$$

where the standardization of $\boldsymbol{A}$ by λ_{max} removes the asymptotic population trend from the overall realized dynamics and growth rate λ such that as the population approaches asymptotic stable age structure, $reactivity_t$ goes to one (see Neubert and Caswell 1997 and also Townley et al. 2007).

Mathematically the combination of asymptotic and transient dynamics that generate the observed population growth, λ, between t and $t+1$ can be represented multiplicatively,

$$\lambda_t = \lambda_{max} * reactivity_t$$

and is often log-transformed to represent the dynamics as additive components (Ellis and Crone 2013, McDonald et al. 2016),

$$\log(\lambda_{realized,t}) = |log(\lambda_{max})| + |\log(reactivity_t)| \quad \text{Eq. 3}$$

Eq 3 introduces $\lambda_{realized,t}$, which adds absolute values so that the strength of antagonistic asymptotic and transient effects with opposite signs may be differentiated providing a measure of the total realized dynamics (McDonald et al. 2016). To estimate the relative contribution of transient dynamics, we rearranged Eq. 3 to define the ratio of transient growth rate relative to the realized growth rate on the log scale (McDonald et al. 2016):

$$\boldsymbol{\rho}_t = \frac{|\log(reactivity_t)|}{\log(\lambda_{realized,t})} \quad \text{Eq. 4}$$

$\boldsymbol{\rho}_t$ is a summary statistic proxy for the amount of transient dynamics, which for brevity we refer to as the ratio of transient dynamics.

Vital rate data

To model age structured population dynamics we collected data describing age specific vital rates for wild pigs (*Sus scrofa sp.*) from the scientific literature and from COMADRE Animal Matrix Database (Salguero-Gómez et al. 2016). Studies considered were limited to those reporting complete data describing age specific vital rates for female survival and fecundity for the population. The proportion of females reproducing annually by age class was not available for most studies so we assumed that all females reproduced once per year. We mostly considered three age classes, juveniles (j) <1 year, yearlings (y) ≥ 1 year and < 2 years, and adults (a) ≥ 2 year, although two studies had only two age classes. We included a non-zero

value for adult survival that represents the probability of remaining in the adult age class for all ages greater than 2 years. Our transition matrices are female only assuming that sex ratios are equal, which is an appropriate assumption for most wild pig populations (FernáNdez-Llario et al. 1999, Bieber and Ruf 2005). The beginning of female reproduction primarily depends on body weight with the average threshold weight required for onset of reproduction being 27kg (Sabrina et al. 2009), and females have been documented to reproduce at 6-months (Cellina 2008). As a result, fecundity for the juvenile age class is included in our transition matrices. Vital rate data (see Table A4.1) from 14 studies on four continents were used to build 16 age structured transition matrices of the form:

$$\boldsymbol{A} = \begin{bmatrix} \sigma_j\gamma_j & \sigma_y\gamma_y & \sigma_a\gamma_a \\ \sigma_j & 0 & 0 \\ 0 & \sigma_y & \sigma_a \end{bmatrix} \quad \text{Eq. 5}$$

Initial age structure

By definition, transient dynamics are sensitive to age structure (see Eq 2 and 3), but initial age structure is often unknown for wildlife populations (Skalski et al. 2010). To address uncertainty in age structure, we generated 1000 potential initial conditions for each age class using Latin hypercube sampling (Stein 1987) and normalized such that $\sum_i n_{i,0} = 1$.

Simulations and generation of transient measures

For each matrix, we simulated 1000 population trajectories using the Latin hypercube initial conditions resulting in 16,000 trajectories across all matrices. Initial investigation indicated that transient dynamics approached zero near the tenth time step for both native and invasive populations, thus we limited our investigation to the first ten time steps, where each

time step represents one year. For each population trajectory we calculated $\lambda_{realized,t}$ and $\boldsymbol{\rho}_t$ (Eq 3 and 4) for each time step.

Sensitivity of transient population growth

To determine the vital rates and age classes most important for influencing transient population growth, we calculated sensitivities (Koons et al. 2016; Caswell 2014). To implement this approach, we placed the parameters comprising $\boldsymbol{A}$ (e.g. σ_i and γ_i) and each age structure component, $n_{i,t}$, into a vector $\boldsymbol{\Theta}_{k,t}$ where *k* indexes native or invasive populations. Following Caswell (2007), the sensitivities of the realized population growth, $\lambda_{realized,t}$, to change in each parameter are generated by calculating $\partial\lambda_{realized,k,t}/\partial\boldsymbol{\Theta}_{k,t}$. These sensitivities are used with the covariance among the elements of $\boldsymbol{\Theta}_{k,t}$ to obtain a first-order approximation of variation in $\lambda_{realized,k,t}$ resulting from changes in the vital rates and changes in age structure,

$$var\left(\lambda_{realized,k,t}\right) \approx \sum_{k,l}\sum_{k,m} cov\left(\theta_{k,l}\theta_{k,m}\right)\frac{\partial\lambda_{realized,k,t}}{\partial\theta_{k,l}}\,\frac{\partial\lambda_{realized,k,t}}{\partial\theta_{k,m}}\Bigg|_{\overline{\theta_{k,lm}}} \qquad \text{Eq. 6}$$

where $\theta_{k,l}$ and $\theta_{k,m}$ are pairs of elements in $\boldsymbol{\Theta}_{k,t}$ and the sensitivities are evaluated at the mean of $\theta_{k,lm}$ across invasive and native populations. Each term in Eq 6 is the contribution to variance in $\lambda_{realized,k,t}$ resulting from the covariance among each pair of elements in $\boldsymbol{\Theta}_{k,t}$ that include vital rates and age structure (Koons et al. 2016). A measure of the total contribution for each element in $\boldsymbol{\Theta}_{k,t}$ (e.g. $\sigma_{k,i}, \gamma_{k,i}, n_{k,i}$) to $var\left(\lambda_{realized,k,t}\right)$ can be obtained by summing over the covariances using (Horvitz et al. 1997),

$$\chi_{\theta_k} \approx \sum_{k} cov\left(\theta_{k,l}\theta_{k,m}\right)\frac{\partial\lambda_{realized,k,t}}{\partial\theta_{k,l}}\,\frac{\partial\lambda_{realized,k,t}}{\partial\theta_{k,m}}\Bigg|_{\overline{\theta_{k,lm}}} \qquad \text{Eq. 7}$$

χ_{θ_k} provides a measure of the contribution to the realized dynamics of each vital rate, each component of age structure or sets of these, (Koons et al. 2016). We report the median percent contribution, $\tilde{x}$, to $var(\lambda_{realized,k,t})$ across generations and initial conditions for native and invasive populations. We were interested in the potential differences in the contribution of age class to variation in realized dynamics for invasive and native populations so we summed χ_{θ_k} across age classes (i.e. $\chi_{\sigma_{k,i}} + \chi_{\gamma_{k,i}} + \chi_{n_{k,i}}$). We were also interested in potential differences between native and invasive populations in age specific vital rates (i.e. $\sum_i \chi_{\sigma_{k,i}}$, $\sum_i \chi_{\gamma_{k,i}}$) and age structure, n_i (i.e. $\sum_i \chi_{n_{k,i}}$). Because trade-offs between vital rates (i.e. σ_i, γ_i) and age structure (n_i) have recently been proposed to be important in driving transient dynamics, we calculated the Spearman rank correlation between contribution of age structure, $\chi_{n_{k,i}}$ and contribution of age specific vital rates, $\chi_{\sigma_{k,i}}$ or $\chi_{\gamma_{k,i}}$, to variation in realized population growth rates for each population.

Statistical Methods

The role of environmental conditions in transient dynamics

Differences in the vital rates and the ratio of transient dynamics ($\boldsymbol{\rho}_t$) between native and invasive populations were evaluated using repeated measures Analysis of Variance (ANOVA) (Girden 1992). To determine if differences in the amount of transient dynamics ($\boldsymbol{\rho}_t$) and in the contribution of parameters to $\lambda_{realized,t}$ (χ_{θ_k}) were significant and if there were significant differences in the vital rates themselves, we evaluated two location statistics: Welch's t-test of the mean (indicated as *μ p-value)* and Wilcoxon rank sum test of the medians (indicated as $\tilde{x}$ *p-value*) also with a Benjamini and Hochberg correction for multiple testing to evaluate differences in the central tendency for native and invasive populations. We considered two criteria for

significance: for $\boldsymbol{\rho}_t$ we considered transients within 2% of zero as non-significant, and for χ_{θ_k} we evaluated differences from zero.

To determine the role of environmental conditions in the persistence of transient dynamics, we considered the relative ratio of transient dynamics, $\boldsymbol{\rho}_t$, as a response variable together with environmental covariates strongly linked to the hypothesized mechanisms thought to control the amount of transients (i.e., availability of forage, variability in precipitation and temperature, precipitation during the coldest and warmest seasons, and annual mean temperature) using restricted maximum likelihood (REML) methods with generation as a repeated-measure factor. We considered if the population was native or invasive as a fixed explanatory variable and allowed the intercepts to vary for invasive and native populations. We considered only fixed effects of environmental covariates in our analysis. For two covariates, that have previously been found to have non-linear effects on wild pig density we considered linear and non-linear forms for these predictors. To determine the contribution each environmental covariate made to transient dynamics we calculated the proportion of variance explained for each predictor using adjusted r-squared. We calculated adjusted r-squared using all combinations of predictors and then applied a hierarchical partitioning algorithm to determine the independent contribution of each predictor to the variance of transient dynamics (Chevan and Sutherland 1991, Mac Nally 2000). Statistical models were implemented using the Mixed GAM Computation Vehicle (mgcv) package (Wood 2011), hierarchical partitioning of the variance was implemented using the Hierarchical Partitioning (hier.part) package (Walsh et al. 2013), and matrix models including transient sensitivity analysis was implemented in the R computing environment (R 2016).

Environmental covariates

We used model covariates that are hypothesized to influence survival and fecundity based on known life history traits and ecological requirements of wild pigs. We identified covariates that best represented factors describing forage resources that have been linked to wild pig fecundity and environmental conditions that influence survival (Table A4.2). We used a geographic information system (GIS) to develop covariates for each study population using two publicly available global datasets: BioClim for abiotic covariates (Hijmans et al. 2005) and the Global Consensus Land Cover for biotic predictors (Tuanmu and Jetz 2014). The published latitude and longitude for each study population was assumed to represent the center of the study site. There is uncertainty in this location and the geographic extent the population may have occupied; to address this we used a 40km radius relevant for wild pig density (Lewis et al. 2017) to represent the average environmental conditions the population experienced. The mean for each environmental predictor within this 40km radius was generated for each population (e.g. matrix) and standardized prior to model fitting.

Pigs have physiological characteristics making them sensitive to high and low temperatures (Geisser and Reyer 2005, Acevedo et al. 2006). Pig mortality increases when ambient temperatures exceed 23°C with exposure to full sun and when ambient temperatures exceed 35°C (Porter and Gates 1969) with exposure to partial sun. Pigs require cooling when temperatures exceed 35°C and juvenile pig mortality increases when temperatures fall below -4°C (Thompson et al. 1996). To represent these temperature extremes we used the long term annual mean temperature (BioClim variable BIO1) and temperature annual range (variability) (BIO7). We expected increasing annual mean temperature to reduce the amount of transient

dynamics (negative relationship) and increased variation in temperature seasonality to increase transient dynamics (positive relationship).

Wild pig reproductive success and survival at low temperatures is influenced by the amount of precipitation in the coldest period of the year (Jedrzejewska et al. 1997, Melis et al. 2006, Honda 2009, Danilov and Panchenko 2012), and has been linked to wild pig occurrence probability (McClure et al. 2015), and wild pig density (Melis et al. 2006, Lewis et al. 2017). The mean amount of precipitation in the coldest quarter (BIO19) was used to represent winter precipitation for the studies. We expected transient dynamics to increase as the amount of winter precipitation increased. In addition, wild pigs thermo-regulate by accessing water resources (Choquenot and Ruscoe 2003), and reduced access to water causes increased juvenile mortality (Fraser and Phillips 1989). We used the mean amount of precipitation in the warmest quarter of the year (summer) (BIO18) to represent this limitation. We predicted that transient dynamics would decline as the amount of summer precipitation increased. Increased annual variability in precipitation has been associated with increased variation in wild pig body mass that indirectly effects survival and reproductive success (Mysterud et al. 2007) and is expected to increase the effects of summer and winter precipitation. We used precipitation seasonality (BIO15) that is a measure of the variation in annual precipitation to represent these potential associations. We predicted an increase in transient dynamics (positive relationship) as the annual variation in precipitation increased.

Litter size and increased reproductive activity has been associated with availability of crops (Frauendorf et al. 2016) and hard mast (i.e., nuts such as acorns) (Sabrina et al. 2009, Salinas et al. 2015, Vetter et al. 2015). Both cultivated land and hard mast forests have been associated with increased wild pig population growth rates (Osada et al. 2015, Salinas et al.

2015, Vetter et al. 2015) however they are expected to influence populations differently. Cultivated land provides a constant forage resource at the expense of reduced habitat (Morelle and Lejeune 2015), while hard mast forests provide a forage resource that may vary considerably but also provides security cover and thermo-regulation (Canu et al. 2015). Because of these potential mechanistic differences we expected cultivated land and hard mast forests to influence transient dynamics differently. We predicted that cultivated land would increase transient dynamics by increasing fecundity but that this might only occur below some threshold due to the reduced amount of habitat. Similarly we predicted that hard mast producing forests would increase transient dynamics by increasing fecundity but the effect would be less than cultivated land. The proportion of each study site occupied by cultivated land and deciduous forests (a surrogate for hard mast forests) were used. Previous studies have found non-linear effects on wild pig density for cultivated land and deciduous forests (Lewis et al. 2017). To evaluate this for transient dynamics, we considered both linear and nonlinear forms for these two predictors.

RESULTS

Differences in vital rates

Differences in vital rates (i.e. σ_i, γ_i) among invasive and native populations were limited. After adjusting for multiple tests using Benjamini and Hochberg correction, differences were detected for three vital rates at α=0.1 using the Welch t-test (Figure 4.1, Table A4.3). These included juvenile fecundity (adjusted p-value = 0.097, unadjusted p-value = 0.048), yearling fecundity (adjusted p-value = 0.073, unadjusted p-value = 0.024), and juvenile survival (adjusted p-value = 0.073, unadjusted p-value = 0.016). Analysis of variance found differences for yearling survival (adjusted p-value = 0.052, unadjusted p-value = 0.017) and yearling fecundity

(adjusted p-value = 0.052, unadjusted p-value = 0.01) at α=0.1 level of significance. There were no differences in the median among the vital rates found using the Wilcoxon rank sum test.

Differences in transient dynamics

The amount of transient dynamics represented as the ratio of transient dynamics ($\boldsymbol{\rho}_t$) differed among invasive and native populations across all time steps while taking uncertainty in initial age structure into account (*F-Statistic* = 636.7, p-value = $<2.0\text{x}10^{-16}$) (Figure 4.2C). The ratio of transient dynamics for invasive populations declined from a median of 12.1% (95% CI: 11.4-12.9%) in the first generation to $1.6\text{x}10^{-4}$% (95% CI: $1.4\text{x}10^{-4}$-$1.8\text{x}10^{-4}$ %) in the fifth generation. Native populations had greater transient dynamics in all generations relative to invasive populations with the median ratio of transient dynamics in the first generation being 29.0% (95% CI: 28.4-30.0%) and in the fifth generation declining to a median of $3.0\text{x}10^{-3}$ % (95% CI: $2.7\text{x}10^{-3}$-$3.2\text{x}10^{-3}$ %). Transients declined faster for invasive populations, and the median was not significantly different from zero in the third generation (*adjusted p-value* = 1). Transients in native populations declined more slowly, and the median was not significantly different from zero in the fifth generation (*adjusted p-value* = 1).

Contribution of vital rates and age structure to realized population dynamics

The total contribution of juveniles, yearlings, and adults to realized dynamics (e.g. $\chi_{\sigma_{k,i}} + \chi_{\gamma_{k,i}} + \chi_{n_{k,i}}$) differed for invasive and native populations (Figure 4.3A). For native populations, all age classes contributed similar amounts to the variation in realized population growth except in the first generation where adults contributed more ($\tilde{x}$ = 44.9%) and juveniles contributed the least ($\tilde{x}$ = 16.5%). Invasive populations demonstrated greater differences in total age specific contributions where, after the first generation, juveniles contributed to the majority ($\tilde{x}$ = 90.5% - 92.1%) of variation. Yearlings contributed a small but significant amount after the

first generation ($\tilde{x}$ = 6.8% - 9.0%). For invasive populations, total adult contribution to variation in transient growth was not different from zero ($\tilde{x}$ *= 0.006;* $\tilde{x}$ *adj. p-value* = 0.999) beyond the second generation.

The contribution summed across all age classes of survival ($\sum_i \chi_{\sigma_{k,i}}$), fecundity ($\sum_i \chi_{\gamma_{k,i}}$) and age structure ($\sum_i \chi_{n_{k,i}}$) also demonstrated differences (Figure 4.3B, C). Age structure contributed the majority to variation in transient population growth across all generations for native populations ($\tilde{x}$ = 49.8% - 57.8%) (Figure 4.3C). This differed for invasive populations where age structure accounted for the majority of variation in the first generation ($\tilde{x}$ = 54.3%) however this declined ($\tilde{x}$ = 41.0%) by the second generation and survival accounted for slightly greater variation in all subsequent generations ($\tilde{x}$ = 43.0%) (see supplemental Figure S1). Fecundity contributed the least amount in both native and invasive populations.

The contribution of age specific vital rates to variation in transient growth ($\chi_{\sigma_{k,i}}$ or $\chi_{\gamma_{k,i}}$) differed between invasive and native populations (see Figure S1 of the supplemental). Juvenile survival ($\tilde{x}$ = 12.6% - 43.9%) and fecundity ($\tilde{x}$ = 6.8% - 13.1%) contributed most (combined $\tilde{x}$ = 19.4% - 57.5%) in invasive populations. Yearling survival contribution ($\tilde{x}$ = 0.4% - 1.5%) was small but significantly different from zero (μ *p-value* < 5.9×10^{-131}; $\tilde{x}$ *p-value* < 2.8×10^{-118}) in all generations. Adult survival contribution was not different from zero (μ *p-value* = 1; $\tilde{x}$ *p-value* = 1) beyond the first generation. Yearling ($\tilde{x}$ = 0.2% - 3.2%) and adult ($\tilde{x}$ = 0.4% - 8.3%) fecundity contribution was small but significantly greater than zero for all generations (μ *p-value* < 1.27×10^{-95}; $\tilde{x}$ *p-value* < 2.2×10^{-31}). Contribution of vital rates to variation in transient growth for native populations was more variable. Adult survival contributed most in the first generation ($\tilde{x}$ = 21.0%) but declined in subsequent generations ($\tilde{x}$ = 2.6% - 3.2%) with yearling ($\tilde{x}$ = 6.8% - 10.1%) and juvenile ($\tilde{x}$ = 11.9% - 12.0%) contributions being greater. Juvenile

fecundity ($\tilde{x}$ = 9.4% - 11.8%) contributed most to transient growth in native populations with adult contribution not significantly different from zero after the first generation (μ *p-value* = 1; $\tilde{x}$ *p-value* = 1).

Spearman rank correlation between the contribution of age structure components ($\chi_{n_{k,i}}$) and age specific vital rates ($\chi_{\sigma_{k,i}}$ or $\chi_{\gamma_{k,i}}$) differed for invasive and native populations (Figure 4.4). Native populations tended to have positive correlations that were greater than invasive populations. For native populations the contribution of yearling and adult survival had the strongest positive correlations with the contribution of the size of the yearling class, while the contribution of juvenile survival had large variation in correlation with the contribution of the size of the juvenile class. Invasive populations tended to have either a negative correlation or positive correlation less than native populations between the contribution of age structure and the contribution of vital rates. Invasive populations had the largest negative correlation for both the contribution of fecundity and the contribution of survival with the contribution of the size of the juvenile age class, indicating a trade-off. In invasive populations the negative trade-off between the contribution of juvenile age class via age structure, survival and fecundity indicated that reductions in survival or fecundity were buffered by the size of the juvenile age class.

Association of environmental conditions and transient dynamics

Statistical models investigating the association between $\boldsymbol{\rho}_t$ and environmental conditions had an adjusted $R^2 = 0.503$ and explained 76.1% of the total deviance when compared with an intercept only model (Zuur et al. 2009). The autocorrelation between successive time periods was small with the first-order autoregressive correlation estimated to be 0.29. A random intercept for native and invasive populations was significant and was included in the final model used to estimate effects of environmental factors.

Annual mean temperature (β*=0.53, SE=0.07*) and annual range in temperature (β*=1.93, SE=0.12*) were positively associated with the ratio of transient dynamics ($\boldsymbol{\rho}_t$) (Figure 4.5). However, annual mean temperature explained a relatively small amount of the variance in the ratio of transient dynamics for native (3%) and invasive (2%) populations. Similarly the explanatory power of annual range in temperature for variance of $\boldsymbol{\rho}_t$ was also small for native (1%) and invasive (4%) populations. Precipitation seasonality was negatively associated (β*=-5.26, SE=0.34*) with the ratio of transient dynamics and explained a greater amount of variation in $\boldsymbol{\rho}_t$ for native (16%) than invasive (12%) populations. Precipitation during the coldest quarter of the year was positively associated (β*=2.00, SE=0.12*) with transient dynamics while precipitation in the warmest quarter was negatively associated (β*=-7.26, SE=0.53*). Precipitation during the coldest quarter explained a larger amount of the variance in the ratio of transient dynamics for invasive populations (21%) than native populations (12%). Precipitation in the warmest quarter explained generally a small amount of variance in the ratio of transient dynamics in invasive (8%) and native (2%) populations.

Cultivated land and deciduous forest were both positively associated with the ratio of transient dynamics (see Supplemental Figure S2). Cultivated land had a non-linear association with the ratio of transient dynamics that explained different amounts of variance for invasive (14%) and native (32%) populations. The highest transient dynamics occurred when cultivated land was a relatively small percentage of available land cover for native populations (8%) and invasive populations (1%). The ratio of transient dynamics declined with increasing cultivated land for both native and invasive populations and approached zero when the proportion of the study site cultivated was greater than 0.3. Deciduous forest demonstrated a positive linear association (β*=1.31, SE=0.16*) that over all explained a similar amount of variance in the ratio

of transient dynamics for native (4%) and invasive (6%) populations. However the response of invasive populations to increasing deciduous forest was more rapid than native populations, with the largest effect on transient dynamics for both populations occurring when the percentage of the study site with deciduous forest was greater than 50%.

DISCUSSION

This study quantified variation in key vital rates across the global geographical distribution of an important invasive species. We found consistent differences in the way vital rates and age structure in invasive and native populations contribute to transient dynamics despite small differences in the vital rates themselves. Native pig populations appear to be more sensitive to deviations from stable age structure having consistently larger changes in transient dynamics relative to invasive populations. These dynamics appear, in part, to be linked with environmental conditions that have been found in previous studies to regulate demography. Consistent with studies that have found positive relationships between reproductive fitness in wild boar and forage resources (Geisser and Reyer 2005, Honda 2009, Morelle and Lejeune 2015, Frauendorf et al. 2016), we found a positive relationship between forage resources and transient dynamics. Relationships were different for deciduous and cultivated land with cultivated land influencing transient dynamics only when relatively small amounts were present. There may be a trade-off with cultivated land that is typically more open and does not provide security cover but does provide high energy forage. This may explain the relatively large magnitude of transient response to cultivated land. In contrast deciduous forests provide both security and forage resources and had a consistent positive linear relationship with transient

dynamics. These environmental conditions that had the largest influence on transient growth appear to be most directly related to fecundity.

A common empirical finding for invasive species is that invasion success is correlated with fecundity (Iles et al. 2016). Highly fecund species are generally better equipped to overcome demographic stochasticity in the establishment phase (Allele effects) than less fecund species (Kanarek and Webb 2010). However in our study, fecundity was not the primary demographic parameter of importance for explaining transient dynamics. For invasive populations, juvenile survival and age structure had the largest influence on variation in realized population growth rate. While juvenile survival is indirectly related to fecundity, this result suggests that invasive species success cannot solely be predicted by fecundity and that there may be complex interactions between vital rates and age structure. We found large differences in the patterns of trade-offs between the contribution of age structure and the contribution of vital rates (see Figure 4.4). For invasive populations the trade-off between the contribution of juvenile age class and the contribution of both survival and fecundity were in opposite directions indicating that reductions in survival or fecundity were buffered by the size of the juvenile age class. This trade-off between age structure and vital rates did not occur in native populations. This may explain why invasive populations were less sensitive to deviations from asymptotic age structure, and hence potentially environmental conditions, when compared with native populations. The negative co-variation of the contributions of age specific vital rates and the size of an age class may be an important mechanism for buffering invasive populations from perturbations such as changing environmental conditions (Gamelon et al. 2015) or harvest. Unstable population structure can strongly affect population dynamics (Koons et al. 2005), but negative co-variation with vital rates has been found to nullify its impact (Gamelon et al. 2015,

Koons et al. 2016). Reduced sensitivity of invasive populations to environmental conditions or perturbations (e.g. harvest) may aid in invasion success for wild pigs. Strong differences between native and invasive populations may indicate underlying differences resulting from different selection pressures acting on the vital rates or introgression in invasive populations that has altered the vital rates of invasive populations.

The demographic buffering hypothesis proposes selection should favor reduced variance in vital rates that contribute most to fitness and has been observed in birds, large mammals and some plant species (Pfister 1998, Gaillard and Yoccoz 2003, Morris and Doak 2004). More recently Koons et al. (2016) suggested that this expectation is incorrect for short-term population fluctuations and that accounting for changing population structure and the interaction with vital rates may result in greater variability for those parameters contributing most to population growth. Consistent with these recent findings, we did not observe reduced variation in vital rates that contributed most to variation in realized population growth. In our study the vital rates that had the largest influence on population growth rates where those that had the greatest variability and those that contributed little to overall population growth rates had the smallest variability (see Figure 4.3 and supplemental Figure S1). This pattern was accentuated for invasive populations. Ellis and Crone (2013) proposed that when transient responses are in the opposite direction of demographic variation there may be alternative mechanisms reducing the effect of environmental variability at a population level and that the degree of ‘‘transient’’ buffering may depend on the correlation of vital rates. While we did not investigate environmental stochasticity explicitly in our study, we did observe differences between native and invasive populations with respect to vital rate and age structure contributions to population growth. Native populations had a tendency for contributions of vital rates and age structure to move in opposite directions. That

is the contribution of vital rates declined in successive generations and the contribution of age structure alone became most important. However the opposite was true for invasive populations where the contributions of age specific vital rates and age structure moved in the same direction increasing in successive generations. This may indicate that invasive and native populations have different mechanisms for buffering environmental variability. These differences cannot be attributed to invasive populations being relatively new because all of the invasive populations we examined were established during European or Polynesian colonization and had been in existence for many hundreds of years (Mayer and Brisbin 1991, Choquenot et al. 1996).

The differences we observed between invasive and native populations may be attributed to differences in both the genetic variation of invasive populations and selection pressures. Introgression from domestic populations into wild populations appears to have increased invasive potential for a diversity of mammals (Senn et al. 2010), fish (Hubbs 1955, Scribner et al. 2000, Nentwig 2007), and plant species (Rieseberg et al. 1999, Whitney et al. 2006). Invasive wild pig populations are typically composed of domestic-wild hybrids (Giuffra et al. 2000, Larson et al. 2005) with domestic introgression that is greater (Gongora et al. 2004) than native populations (Canu et al. 2016). There is evidence that introgression from domestic pigs, which are under artificial directional selection for large litters and rapid growth, increases wild pig fitness (Fulgione et al. 2016) allowing invasive pigs to reproduce earlier and with larger litters. This is consistent with our findings that fecundity of younger age classes was significantly greater for invasive populations than for native populations. In addition juvenile fecundity was important in contributing to transient growth rates. This increased fitness may result in different mechanisms that buffer perturbations facilitating invasion success. In addition, invasive species (Nentwig 2007) including wild pigs (Tabak et al. 2016), are frequently-moved often over long

distances and experience a diversity of environments. This frequent movement along with increased genetic diversity (Prentis et al. 2008) may result in selection of traits that are less sensitive to environmental conditions because populations have had to repeatedly and abruptly adapt to a diversity of environments across multiple continents. Repeated introductions and movement that result in genetic bottlenecks have been observed to influence biological invasions positively (Golani et al. 2007) and has been attributed to invasive species success for European rabbits (Zenger et al. 2003) and sunflowers (Rieseberg et al. 1999).

Our findings support the use of transient analyses as an approach to guide short-term management decisions (Buhnerkempe 2011, Stott et al. 2012, Ellis and Crone 2013, Koons et al. 2016). The observed trade-offs between juvenile age class, survival, and fecundity may have unexpected consequences when managing invasive populations and implementing population control. Empirical work by Sabrina et al. (2009) found that high harvest of wild pig populations resulted in increased juvenile reproduction regardless of the environmental conditions – reproduction occurred under both good and poor conditions. In light of our findings for invasive wild pig populations, any changes to age structure (e.g. harvest, culling) that shift populations toward greater juvenile densities may unintentionally increase the population growth rate. Given these findings, approaches to population control must be selected carefully, and our results suggest shifting population age structure to be adult skewed would least influence population growth rates. Our findings indicate that recreational hunting or programs using public harvest to control wild pig populations may inadvertently be increasing population growth rates because these methods often focus on older aged animals. We also found that invasive populations recovered from perturbations quickly, meaning that control efforts that infrequently remove animals may have little to no effect on population growth rates. The rapid recovery from

perturbations may also indicate that invasive wild pig populations can overcome environmental variability allowing successful invasion of habitats thought to be of poor quality or at low risk for invasion.

Biological invasions are a key driving force in current global change and are likely to become increasingly important (Mooney and Cleland 2001). Our study has broad applications for studying invasive species and the mechanisms that give rise to their population dynamics. While we did not find support for the demographic buffering hypothesis, there is the suggestion of a new hypothesis that could be generalized for invasive species suggesting that tradeoffs in sensitivity between vital rates and age structure can buffer populations. In addition there may be surprising potential for strong contributions of juvenile classes and a smaller direct role for fecundity than previously thought. These tradeoffs may be particularly important for invasive species that have experienced domestic introgression. The innovative methodological approach used here allows not only strong tests of current hypotheses regarding controls on population dynamics, but more detailed insights into the tradeoffs between vital rates and age structure that form novel hypotheses regarding how populations may be buffered to environmental variability and perturbation. While relevant to all species, these insights are particularly useful for understanding the response of invasive species and hence developing appropriate, ecologically informed control methods using "best science" practices.

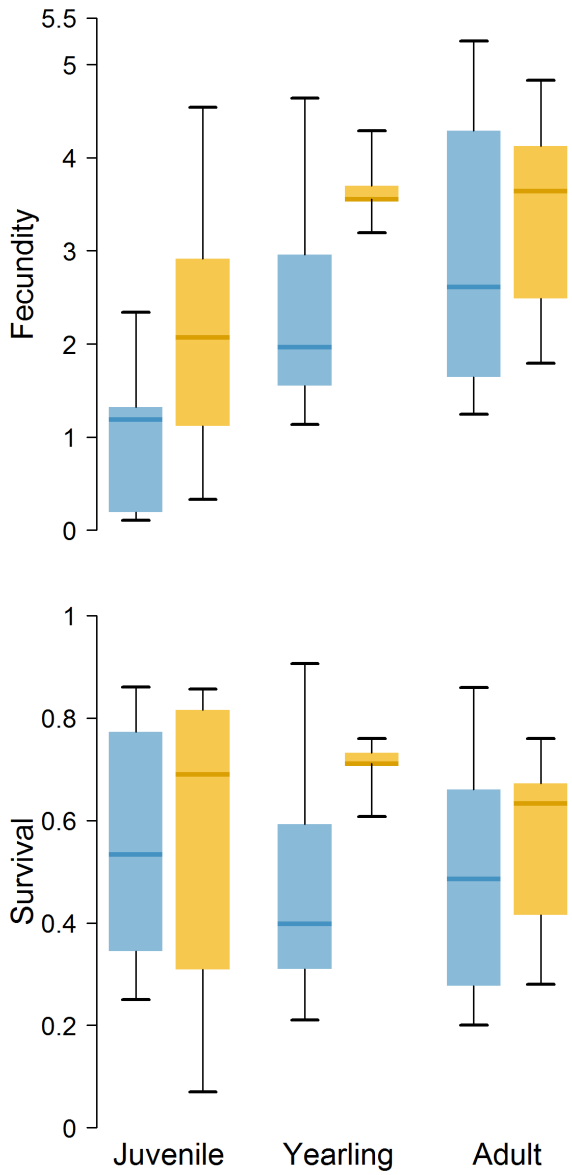


Figure 4.1. Vital rates for native (blue) and invasive (yellow) wild pig populations used in our study. Juvenile and yearling pigs in invasive populations have higher fecundity ($\sigma_i \gamma_i$) compared to native populations. Adult pigs do not exhibit differences in fecundity. Survival (σ_i) had relatively large variance across native and invasive populations. Yearling survival for invasive populations was higher on average than native populations. Whiskers indicate the minimum and maximum of the data; boxes are the interquartile range; median is indicated by a solid line; and if present outliers are indicated by a circle.

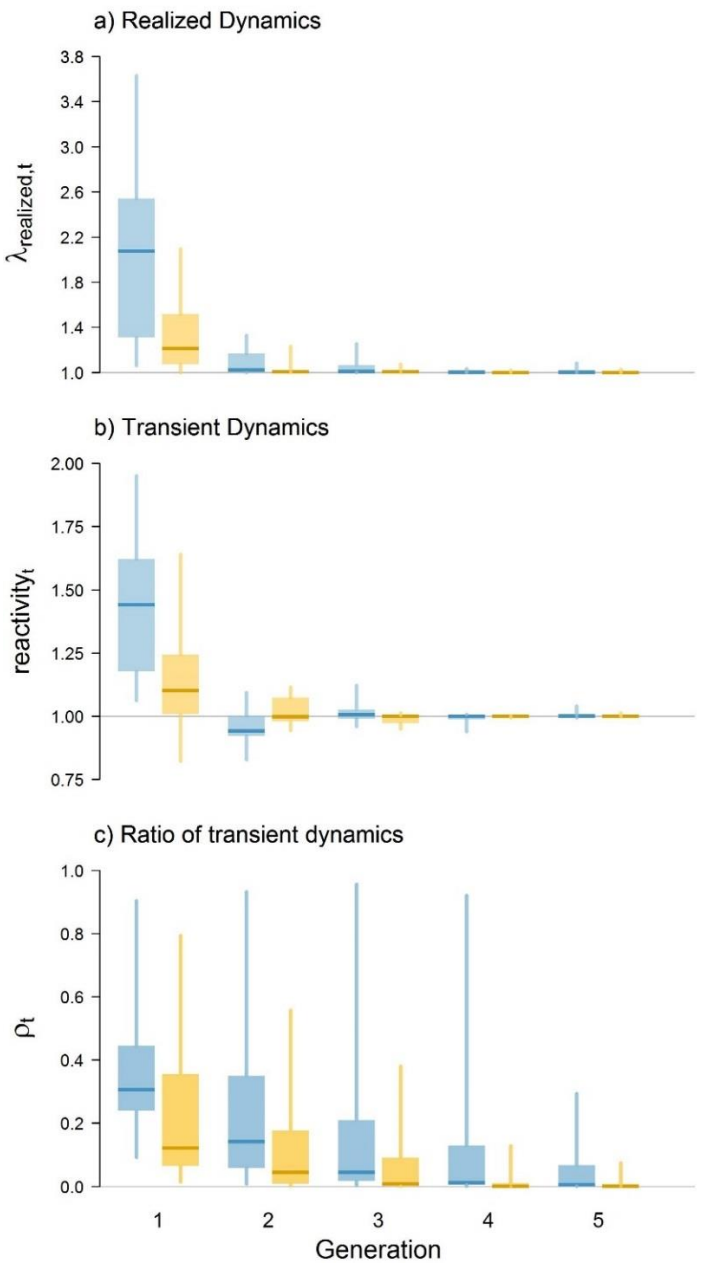


Figure 4.2. Transient population dynamics for invasive (yellow) and native (blue) wild pig populations included in our study. Invasive populations move more rapidly towards stable population dynamics and transient dynamics are less persistent. Boxplot whiskers indicate full range of dynamics; boxes identify interquartile range; and median is marked as a dark line. Panel a is the realized population dynamics ($\lambda_{realized,t}$) for the first five generations. The values are scaled so that a value of 1 indicates that population growth is at asymptotic lambda. Panel b is the transient dynamics ($reactivity_t$) for where a value of 1 indicates no reactivity, meaning that there is no contribution of age structure to realized population dynamics. Panel C is the ratio of transient growth rate relative to the realized growth rate ($\boldsymbol{\rho}_t$). For clarity in presentation outliers are not plotted separately and whiskers indicate the entire range of the data.

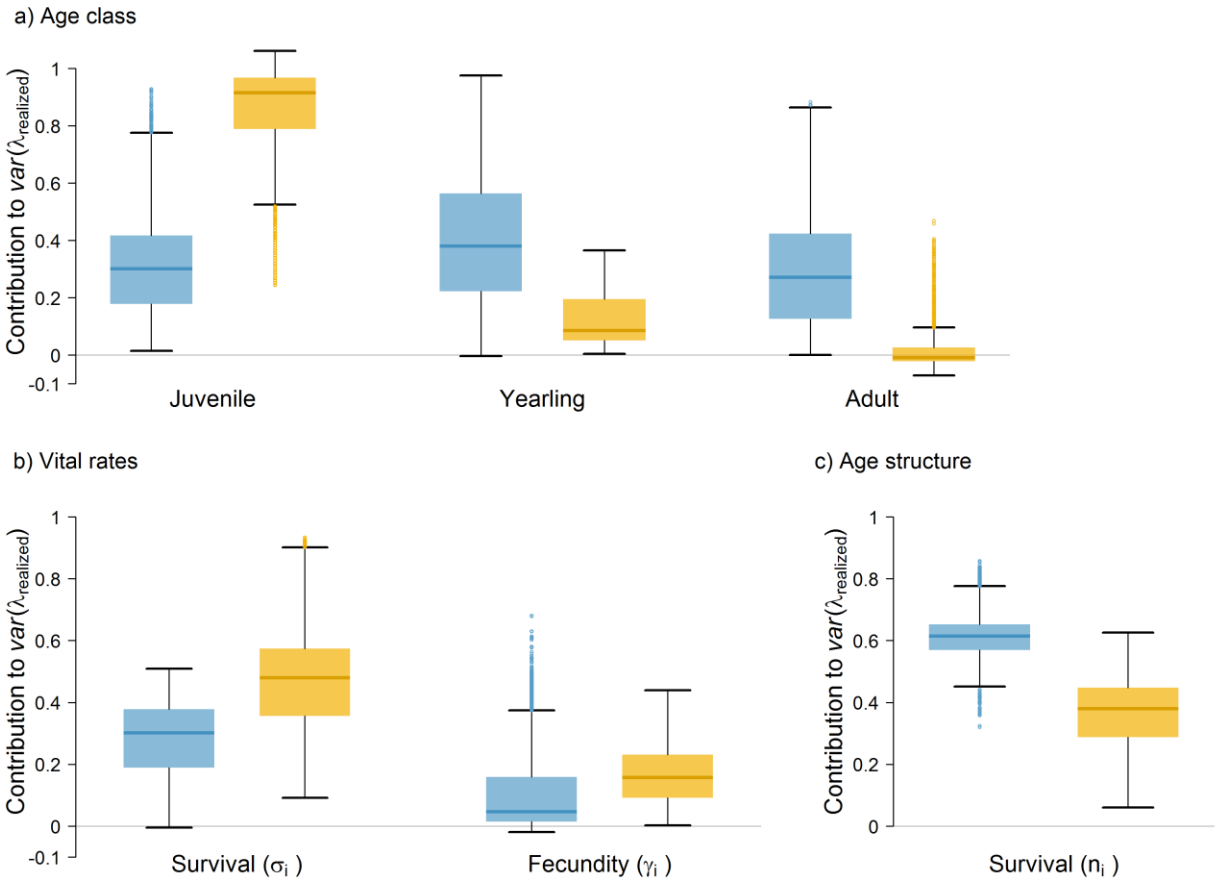


Figure 4.3. Contribution of age class (i.e. $\chi_{\sigma_{k,i}} + \chi_{\gamma_{k,i}} + \chi_{n_{k,i}}$) (panel a), vital rates (i.e. $\sum_i \chi_{\sigma_{k,i}}$, $\sum_i \chi_{\gamma_{k,i}}$) (panel b), and age structure (i.e. $\sum_i \chi_{n_{k,i}}$) (panel c) to variation in realized growth rate ($\lambda_{realized}$) for the third generation (other generations available in supplemental). In panel a, juveniles in invasive populations (yellow) contribute significantly more to $\lambda_{realized}$ when compared to native populations (blue). For native populations, juveniles, yearlings, and adults contribute nearly equal amounts. In panel b survival has greater contribution to $\lambda_{realized}$ relative to native populations. Comparison of panel b and c shows that the contribution of age structure was most important for native populations. Survival and age structure contributed most for invasive populations with fecundity being least important. For invasive populations parameters that contributed most to population growth also had the greatest variability.

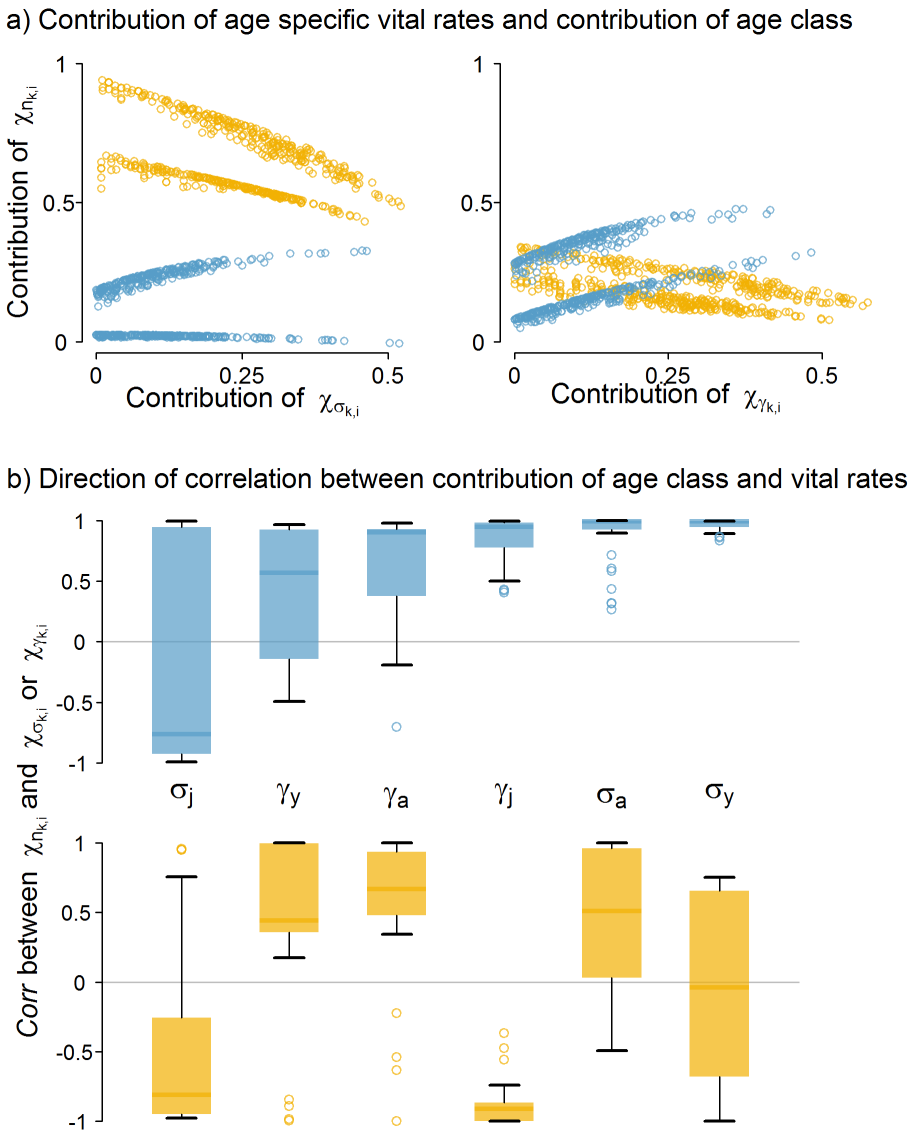


Figure 4.4. Relationship between the contribution of age structure and the contribution of age specific vital rates to variation in realized growth rate, $\lambda_{realized,t}$. Panel a illustrates the relationship between the contribution of age structure ($\chi_{n_{k,i}}$), survival ($\chi_{\sigma_{k,i}}$), fecundity ($\chi_{\gamma_{k,i}}$) for juveniles in two native (blue) and two invasive (yellow) populations that have opposite directions. Panel b describes the direction of the relationship in the contribution of age structure, survival, and fecundity for all populations in our study measured using Spearman's rank correlation. Boxplots represent distribution of correlations for native and invasive populations for the generations two through five (each generation is available in supplemental). Native populations (blue) are ordered by median direction of relationship and have largely positive or neutral trade-offs between age structure and vital rates indicating that demographic buffering is not important. The corresponding vital rates for invasive populations (yellow) have generally larger variation. There is a negative trade-off for juvenile survival and fecundity indicating that reductions in survival or fecundity were buffered by juvenile age structure.

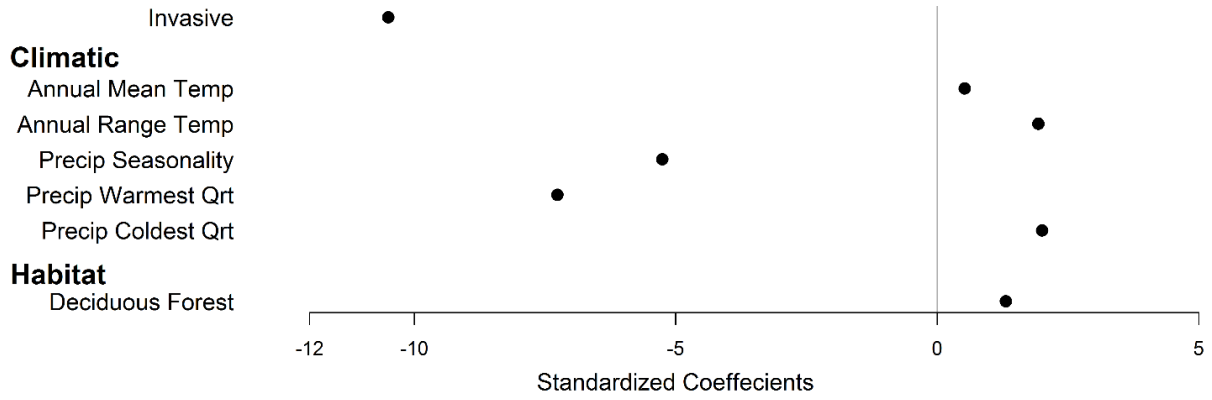


Figure 4.5. Standardized restricted maximum likelihood parameter estimates for linear effects of environmental variables on the ratio of transient dynamics ($\boldsymbol{\rho_t}$). Environmental conditions related to forage availability, deciduous forests, tended to increase transient dynamics while climatic conditions influencing survival had mixed effects.

REFERENCES

Acevedo, P., M. A. Escudero, R. Muńoz, and C. Gortázar. 2006. Factors affecting wild boar abundance across an environmental gradient in Spain. Acta Theriologica **51**:327-336.

Barrett, R. H. 1971. Ecology of the feral hog in Tehama County, California. University of California, Berkeley.

Bevins, S. N., K. Pedersen, M. W. Lutman, T. Gidlewski, and T. J. Deliberto. 2014. Consequences Associated with the Recent Range Expansion of Nonnative Feral Swine. BioScience **64**:291-299.

Bieber, C., and T. Ruf. 2005. Population dynamics in wild boar Sus scrofa: ecology, elasticity of growth rate and implications for the management of pulsed resource consumers. Journal of Applied Ecology **42**:1203-1213.

Bierzychudek, P. 1999. Looking backwards: assessing the projections of a transition matrix model. Ecological Applications **9**:1278-1287.

Boitani, L., P. Trapanese, L. Mattei, and D. Nonis. 1995. Demography of a wild boar (Sus scrofa, L.) population in Tuscany, Italy. Gibier faune sauvage **12**:109-132.

Buhnerkempe, M. 2011. The utility of transient sensitivity for wildlife management and conservation: bison as a case study. Biological Conservation **144**:1808-1815.

Bywater, K. A., M. Apollonio, N. Cappai, and P. A. Stephens. 2010. Litter size and latitude in a large mammal: the wild boar Sus scrofa. Mammal Review **40**:212-220.

Canu, A., M. Scandura, E. Merli, R. Chirichella, E. Bottero, F. Chianucci, A. Cutini, and M. Apollonio. 2015. Reproductive phenology and conception synchrony in a natural wild boar population. Hystrix, the Italian Journal of Mammalogy **26**:77-84.

Canu, A., S. T. Vilaça, L. Iacolina, M. Apollonio, G. Bertorelle, and M. Scandura. 2016. Lack of polymorphism at the MC1R wild-type allele and evidence of domestic allele introgression across European wild boar populations. Mammalian Biology-Zeitschrift für Säugetierkunde **81**:477-479.

Caswell, H. 2007. Sensitivity analysis of transient population dynamics. Ecology letters **10**:1-15.

Caswell, H. 2014. Matrix population models. Wiley Online Library.

Cellina, S. 2008. Effects of supplemental feeding on the body condition and reproductive state of wild boar Sus scrofa in Luxembourg. University of Sussex Brighton, Dudelange, Luxembourg.

Chevan, A., and M. Sutherland. 1991. Hierarchical partitioning. The American Statistician **45**:90-96.

Choquenot, D., J. McIlroy, and T. Korn. 1996. Managing Vertebrate Pests: Feral Pigs. Page 163 *in* Bureau of Resource Sciences, editor. Australian Government Publishing Service, Canberra.

Choquenot, D., and W. A. Ruscoe. 2003. Landscape complementation and food limitation of large herbivores: habitat- related constraints on the foraging efficiency of wild pigs. Journal of Animal Ecology **72**:14-26.

Csányi, S. 2014. Wild boar population dynamics and management in Hungary. Journal of Mountain Ecology **3**.

Danilov, P., and D. Panchenko. 2012. Expansion and some ecological features of the wild boar beyond the northern boundary of its historical range in European Russia. Russian journal of ecology **43**:45-51.

Diong, C. H. 1982. Population biology and management of the feral pig (Sus scrofa L.) in Kipahulu Valley, Maui. Dissertation. University of Hawai'i, Manoa Honolulu.

Ellis, M. M., and E. E. Crone. 2013. The role of transient dynamics in stochastic population growth for nine perennial plants. Ecology **94**:1681-1686.

Ezard, T. H., J. M. Bullock, H. J. Dalgleish, A. Millon, F. Pelletier, A. Ozgul, and D. N. Koons. 2010. Matrix models for a changeable world: the importance of transient dynamics in population management. Journal of Applied Ecology **47**:515-523.

FernáNdez-Llario, P., J. Carranza, and P. Mateos-Quesada. 1999. Sex allocation in a polygynous mammal with large litters: the wild boar. Animal Behaviour **58**:1079-1084.

Focardi, S., J.-M. Gaillard, F. Ronchi, and S. Rossi. 2008. Survival of wild boars in a variable environment: unexpected life-history variation in an unusual ungulate. Journal of Mammalogy **89**:1113-1123.

Fraser, D., and P. Phillips. 1989. Lethargy and low water intake by sows during early lactation: a cause of low piglet weight gains and survival? Applied Animal Behaviour Science **24**:13-22.

Frauendorf, M., F. Gethöffer, U. Siebert, and O. Keuling. 2016. The influence of environmental and physiological factors on the litter size of wild boar (Sus scrofa) in an agriculture dominated area in Germany. Science of the Total Environment **541**:877-882.

Fulgione, D., D. Rippa, M. Buglione, M. Trapanese, S. Petrelli, and V. Maselli. 2016. Unexpected but welcome. Artificially selected traits may increase fitness in wild boar. Evolutionary Applications.

Gabor, T. M., E. C. Hellgren, R. A. Bussche, and N. J. Silvy. 1999. Demography, sociospatial behaviour and genetics of feral pigs (Sus scrofa) in a semi- arid environment. Journal of Zoology **247**:311-322.

Gaillard, J.-M., and N. G. Yoccoz. 2003. Temporal variation in survival of mammals: a case of environmental canalization? Ecology **84**:3294-3306.

Gamelon, M., A. Besnard, J. M. Gaillard, S. Servanty, E. Baubet, S. Brandt, and O. Gimenez. 2011. High hunting pressure selects for earlier birth date: wild boar as a case study. Evolution **65**:3100-3112.

Gamelon, M., M. Douhard, E. Baubet, O. Gimenez, S. Brandt, and J.-M. Gaillard. 2013. Fluctuating food resources influence developmental plasticity in wild boar. Biology letters **9**:20130419.

Gamelon, M., J. M. Gaillard, O. Gimenez, T. Coulson, S. Tuljapurkar, and E. Baubet. 2015. Linking demographic responses and life history tactics from longitudinal data in mammals. Oikos **125**:395-404.

Gamelon, M., J. M. Gaillard, S. Servanty, O. Gimenez, C. Toïgo, E. Baubet, F. Klein, and J. D. Lebreton. 2012. Making use of harvest information to examine alternative management scenarios: a body weight- structured model for wild boar. Journal of Applied Ecology **49**:833-841.

Geisser, H., and H. U. Reyer. 2005. The influence of food and temperature on population density of wild boar Sus scrofa in the Thurgau (Switzerland). Journal of Zoology **267**:89-96.

Gellner, G., K. S. McCann, and A. Hastings. 2016. The duality of stability: towards a stochastic theory of species interactions. Theoretical Ecology:1-9.

Gethöffer, F., G. Sodeikat, and K. Pohlmeyer. 2007. Reproductive parameters of wild boar (Sus scrofa) in three different parts of Germany. European Journal of Wildlife Research **53**:287-297.

Giles, J. 1980. The ecology of feral pigs in western New South Wales. University of Sydney, Sydney.

Girden, E. R. 1992. ANOVA: Repeated measures. Sage.

Giuffra, E., J. Kijas, V. Amarger, Ö. Carlborg, J.-T. Jeon, and L. Andersson. 2000. The origin of the domestic pig: independent domestication and subsequent introgression. Genetics **154**:1785-1791.

Golani, D., E. Azzurro, M. Corsini-Foka, M. Falautano, F. Andaloro, and G. Bernardi. 2007. Genetic bottlenecks and successful biological invasions: the case of a recent Lessepsian migrant. Biology letters **3**:541-545.

Gongora, J., P. Fleming, P. B. Spencer, R. Mason, O. Garkavenko, J.-N. Meyer, C. Droegemueller, J. H. Lee, and C. Moran. 2004. Phylogenetic relationships of Australian and New Zealand feral pigs assessed by mitochondrial control region sequence and nuclear GPIP genotype. Molecular Phylogenetics and Evolution **33**:339-348.

Hanson, L. B., M. S. Mitchell, J. B. Grand, D. B. Jolley, B. D. Sparklin, and S. S. Ditchkoff. 2009. Effect of experimental manipulation on survival and recruitment of feral pigs. Wildlife Research **36**:185-191.

Hastings, A. 2004. Transients: the key to long-term ecological understanding? Trends in Ecology & Evolution **19**:39-45.

Hijmans, R. J., S. E. Cameron, J. L. Parra, P. G. Jones, and A. Jarvis. 2005. Very high resolution interpolated climate surfaces for global land areas. International journal of climatology **25**:1965-1978.

Honda, T. 2009. Environmental factors affecting the distribution of the wild boar, sika deer, Asiatic black bear and Japanese macaque in central Japan, with implications for human-wildlife conflict. Mammal Study **34**:107-116.

Horvitz, C., D. W. Schemske, and H. Caswell. 1997. The relative "importance" of life-history stages to population growth: prospective and retrospective analyses. Pages 247-271 Structured-population models in marine, terrestrial, and freshwater systems. Springer.

Hubbs, C. L. 1955. Hybridization between fish species in nature. Systematic zoology **4**:1-20.

Iles, D. T., R. Salguero- Gómez, P. B. Adler, and D. N. Koons. 2016. Linking transient dynamics and life history to biological invasion success. Journal of Ecology **104**:399-408.

Jedrzejewska, B., W. Jedrzejewski, A. N. Bunevich, L. Milkowski, and Z. A. Krasinski. 1997. Factors shaping population densities and increase rates of ungulates in Bialowieza Primeval Forest (Poland and Belarus) in the 19th and 20th centuries. Zeszyty Problemowe Postepow Nauk Rolniczych **42**:399-451.

Kanarek, A. R., and C. T. Webb. 2010. Allee effects, adaptive evolution, and invasion success. Evolutionary Applications **3**:122-135.

Koons, D. N., J. B. Grand, B. Zinner, and R. F. Rockwell. 2005. Transient population dynamics: Relations to life history and initial population state. Ecological Modelling **185**:283-297.

Koons, D. N., D. T. Iles, M. Schaub, and H. Caswell. 2016. A life- history perspective on the demographic drivers of structured population dynamics in changing environments. Ecology letters **19**:1023-1031.

Lande, R., S. Engen, and B.-E. Saether. 2003. Stochastic population dynamics in ecology and conservation. Oxford University Press on Demand.

Larson, G., K. Dobney, U. Albarella, M. Fang, E. Matisoo-Smith, J. Robins, S. Lowden, H. Finlayson, T. Brand, and E. Willerslev. 2005. Worldwide phylogeography of wild boar reveals multiple centers of pig domestication. Science **307**:1618-1621.

Lewis, J. S., M. L. Farnsworth, C. L. Burdett, D. M. Theobald, M. Gray, and R. S. Miller. 2017. Biotic and abiotic factors predicting the global distribution and population density of an invasive large mammal. Scientific Reports **7**.

Lowe, S., M. Browne, S. Boudjelas, and M. De Poorter. 2000. 100 of the world's worst invasive alien species: a selection from the global invasive species database. Page 12. The Invasive Species Specialist Group a specialist group of the Species Survival Commission of the World Conservation Union,.

Mac Nally, R. 2000. Regression and model-building in conservation biology, biogeography and ecology: the distinction between–and reconciliation of–'predictive'and 'explanatory'models. Biodiversity and Conservation **9**:655-671.

Massei, G., P. Genov, B. Staines, and M. Gorman. 1997. Mortality of wild boar, Sus scrofa, in a Mediterranean area in relation to sex and age. Journal of Zoology **242**:394-400.

Massei, G., J. Kindberg, A. Licoppe, D. Gačić, N. Šprem, J. Kamler, E. Baubet, U. Hohmann, A. Monaco, and J. Ozoliņš. 2015. Wild boar populations up, numbers of hunters down? A review of trends and implications for Europe. Pest management science **71**:492-500.

Mayer, J., and I. Brisbin. 1991. Wild pigs in the United States: their life history, morphology and current status. University of Georgia Press. Athens, Georgia, USA.

McClure, M. L., C. L. Burdett, M. L. Farnsworth, M. Lutman, D. M. Theobald, P. D. Riggs, D. A. Grear, and R. S. Miller. 2015. Modeling and mapping the probability of occurrence of invasive wild pigs across the contiguous United States. PloS one **10**.

McDonald, J. L., I. Stott, S. Townley, and D. J. Hodgson. 2016. Transients drive the demographic dynamics of plant populations in variable environments. Journal of Ecology **104**:306-314.

McMahon, S. M., and C. Metcalf. 2008. Transient sensitivities of non-indigenous shrub species indicate complicated invasion dynamics. Biological Invasions **10**:833-846.

Melis, C., P. A. Szafrańska, B. Jędrzejewska, and K. Bartoń. 2006. Biogeographical variation in the population density of wild boar (Sus scrofa) in western Eurasia. Journal of Biogeography **33**:803-811.

Mooney, H. A., and E. E. Cleland. 2001. The evolutionary impact of invasive species. Proceedings of the National Academy of Sciences **98**:5446-5451.

Morelle, K., and P. Lejeune. 2015. Seasonal variations of wild boar Sus scrofa distribution in agricultural landscapes: a species distribution modelling approach. European Journal of Wildlife Research **61**:45-56.

Moretti, M. 2014. Birth distribution, structure and dynamics of a hunted mountain population of wild boars (Sus scrofa L.), Ticino, Switzerland. Journal of Mountain Ecology **3**.

Morris, W. F., and D. F. Doak. 2004. Buffering of life histories against environmental stochasticity: accounting for a spurious correlation between the variabilities of vital rates and their contributions to fitness. The American Naturalist **163**:579-590.

Mysterud, A., P. Tryjanowski, M. Panek, N. Pettorelli, and N. C. Stenseth. 2007. Inter-specific synchrony of two contrasting ungulates: wild boar (Sus scrofa) and roe deer (Capreolus capreolus). Oecologia **151**:232-239.

Neet, C. 2014. Population dynamics and management of Sus scrofa in western Switzerland: a statistical modelling approach. Journal of Mountain Ecology **3**.

Nentwig, W. 2007. Biological invasions. Springer Science & Business Media.

Neubert, M. G., and H. Caswell. 1997. Alternatives to resilience for measuring the responses of ecological systems to perturbations. Ecology **78**:653-665.

Osada, Y., T. Kuriyama, M. Asada, H. Yokomizo, and T. Miyashita. 2015. Exploring the drivers of wildlife population dynamics from insufficient data by Bayesian model averaging. Population Ecology **57**:485-493.

Pfister, C. A. 1998. Patterns of variance in stage-structured populations: evolutionary predictions and ecological implications. Proceedings of the National Academy of Sciences **95**:213-218.

Porter, W. P., and D. M. Gates. 1969. Thermodynamic equilibria of animals with environment. Ecological monographs **39**:227-244.

Prentis, P. J., J. R. Wilson, E. E. Dormontt, D. M. Richardson, and A. J. Lowe. 2008. Adaptive evolution in invasive species. Trends in plant science **13**:288-294.

R Core Team. 2016. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria.

Rieseberg, L. H., M. J. Kim, and G. J. Seiler. 1999. Introgression between the cultivated sunflower and a sympatric wild relative, Helianthus petiolaris (Asteraceae). International Journal of Plant Sciences **160**:102-108.

Rip, J., and K. McCann. 2011. Cross- ecosystem differences in stability and the principle of energy flux. Ecology letters **14**:733-740.

Sabrina, S., G. Jean- Michel, T. Carole, B. Serge, and B. Eric. 2009. Pulsed resources and climate- induced variation in the reproductive traits of wild boar under high hunting pressure. Journal of Animal Ecology **78**:1278-1290.

Salguero- Gómez, R., O. R. Jones, C. R. Archer, C. Bein, H. Buhr, C. Farack, F. Gottschalk, A. Hartmann, A. Henning, and G. Hoppe. 2016. COMADRE: a global data base of animal demography. Journal of Animal Ecology **85**:371-384.

Salinas, R. A., W. H. Stiver, J. L. Corn, S. Lenhart, C. Collins, M. Madden, K. C. Vercauteren, B. B. Schmit, E. Kasari, and A. ODOI. 2015. An individual- based model for feral hogs in great smoky mountains national park. Natural Resource Modeling **28**:18-36.

Scribner, K. T., K. S. Page, and M. L. Bartron. 2000. Hybridization in freshwater fishes: a review of case studies and cytonuclear methods of biological inference. Reviews in Fish Biology and Fisheries **10**:293-323.

Senn, H. V., N. H. Barton, S. J. Goodman, G. M. Swanson, K. A. Abernethy, and J. M. Pemberton. 2010. Investigating temporal changes in hybridization and introgression in a predominantly bimodal hybridizing population of invasive sika (Cervus nippon) and native red deer (C-elaphus) on the Kintyre Peninsula, Scotland. Molecular Ecology **19**:910-924.

Singer, F. J., and B. B. Ackerman. 1981. Food availability, reproduction, and condition of European wild boar in Great Smoky Mountains National Park. US Department of Interior, National Park Service, Southeast Region [and] Uplands Field Research Laboratory, Great Smoky Mountains National Park.

Skalski, J. R., K. E. Ryding, and J. Millspaugh. 2010. Wildlife demography: analysis of sex, age, and count data. Academic Press, Seattle, Washington.

Sol, D., and L. Lefebvre. 2000. Behavioural flexibility predicts invasion success in birds introduced to New Zealand. Oikos **90**:599-605.

Stein, M. 1987. Large sample properties of simulations using Latin hypercube sampling. Technometrics **29**:143-151.

Stott, I., D. J. Hodgson, and S. Townley. 2012. Beyond sensitivity: nonlinear perturbation analysis of transient dynamics. Methods in Ecology and Evolution **3**:673-684.

Tabak, M. A., A. J. Piaggio, R. S. Miller, R. A. Sweitzer, and H. B. Ernest. 2016. Linking anthropogenic factors with the movement of an invasive species. In Submission.

Thompson, P., J. Brewer, and E. Brewer. 1996. Swine care handbook. National Pork Board, Des Moines **34**.

Townley, S., D. Carslake, O. KELLIE- SMITH, D. McCarthy, and D. Hodgson. 2007. Predicting transient amplification in perturbed ecological systems. Journal of Applied Ecology **44**:1243-1251.

Tuanmu, M. N., and W. Jetz. 2014. A global 1- km consensus land- cover product for biodiversity and ecosystem modelling. Global Ecology and Biogeography **23**:1031-1045.

Vetter, S. G., T. Ruf, C. Bieber, and W. Arnold. 2015. What is a mild winter? Regional differences in within-species responses to climate change. PloS one **10**:e0132178.

Walsh, C., R. Mac Nally, and M. C. Walsh. 2013. The hier. part package. Hierarchical Partitioning. R project for statistical computing.

Whitney, K. D., R. A. Randell, and L. H. Rieseberg. 2006. Adaptive introgression of herbivore resistance traits in the weedy sunflower Helianthus annuus. The American Naturalist **167**:794-807.

Wood, S. N. 2011. Fast stable restricted maximum likelihood and marginal likelihood estimation of semiparametric generalized linear models. Journal of the Royal Statistical Society: Series B (Statistical Methodology) **73**:3-36.

Zenger, K., B. Richardson, and A. M. Vachot- Griffin. 2003. A rapid population expansion retains genetic diversity within European rabbits in Australia. Molecular Ecology **12**:789-794.

Zuur, A., E. Ieno, N. Walker, A. Saveliev, and G. Smith. 2009. Mixed effects models and extensions in ecology with R. Gail M, Krickeberg K, Samet JM, Tsiatis A, Wong W, editors. New York, NY: Spring Science and Business Media.

CHAPTER 5

GENERAL CONCLUSIONS

In this dissertation, I have explored ecological mechanisms underlying population growth, pathogen prevalence, and emerging policy for a globally important invasive species. As outlined in the introduction, the primary aims of this dissertation were to study the interaction between societal and biological drivers and the relative contribution of these to environmental policy. Understanding these interactions requires both investigation of the latent biological processes that give rise to environmental policy and also the societal perceptions of these biological processes.

First, I investigated the relative contribution of invasive species range expansion and social discourse in generating national invasive species policy. This investigation found that two biological processes, population growth, specifically range expansion and disease risk, are often associated with the emergence of wildlife-agricultural policy. Specifically concerns from livestock agriculture related to economically important pathogens appeared to be the primary determinant of policy at the wildlife-livestock interface. Using a meta-analysis and network methods, I identified 34 economically important pathogens that can be transmitted among wild pigs, livestock, humans, and wildlife indicating that invasive wild pigs may pose risks for disease transmission among a diversity of species. I am actively expanding this line of inquiry to investigate how differences in state level regulations related to human movement of wild pigs may have contributed to range expansion and how proxy data, such as Twitter, can be used to map the social value placed on wild pigs. These data describing state regulations and social values are being used as covariates to estimate the probability of introduction and persistence of

wild pigs using dynamic occupancy models. The objective is to compete ecological factors such as habitat suitability against anthropogenic processes associated with human movement of pigs to determine which is more important in wild pig range expansion. The broader objective is to develop methods that explicitly link biological processes and societal processes do describe processes important for invasive species studies.

To elucidate biological processes important for invasive species in order to better understand policy opportunities and policy consequences, I investigated hypothesis of ecological processes important for short-term population dynamics that contribute to invasive species population growth. Analysis of short-term transient population growth demonstrated consistent differences in the way vital rates and age structure in invasive and native populations contribute to short-term population growth. Contrary to the demographic buffering hypothesis, vital rates that had the largest influence on population growth also had the greatest variability. Invasive population's demonstrated trade-offs between juvenile age structure and vital rates indicating that the sensitivity between vital rates and age structure may be an important contributor to invasive species population dynamics. The lack of support for the demographic buffering hypotheses indicates that there may be a new hypothesis that could be generalized for invasive species suggesting that tradeoffs in sensitivity between vital rates and age structure can buffer populations. These findings have may have unexpected consequences when managing invasive populations and implementing population control. Changes to age structure (e.g. harvest, culling) that shift populations toward greater juvenile densities may unintentionally increase the population growth rate. Given these findings, approaches to population control must be selected carefully and our results suggest shifting population age structure may be an important component when implementing population control. I am currently developing simulation studies

that use data describing the age distribution of harvested pigs to explicitly investigate how commonly used control methods may alter population growth rates and how these deviate from optimal control. In addition to investigate how domestic introgression of invasive species may influence transient dynamics, I have started collecting data describing the amount of domestic introgression in populations with vital rate data available and plan to incorporate these data into the models developed during this dissertation. Investigating domestic introgression of invasive species using transient dynamics may offer additional insights into selection pressures that facilitate invasive species success.

Disease risks were also an important driver of emerging wildlife-agricultural policy. Pathogen prevalence was associated with environmental gradients affecting host survival and by changes in mammal host species richness. A single-host pathogen was most sensitive to changes in both environmental conditions and species richness relative to a multi-host pathogen. For the single-host pathogen pseudorabies virus, I found support for dilution effects, but I did not find support for amplification effects in swine brucellosis when controlling for environmental factors, host density and observation error. However, there may be non-linear relationships among population growth, host species density, and host competency. Considering host competency as a continuum and explicitly including the range of competency may provide greater insight into the role of species diversity in multi-host pathogen systems. Amplification in pathogen transmission resulting from increasing species diversity may depend on the range of host competency or on the composition of different host competencies in a community. Additionally, detection probability demonstrated differences that could not be attributed to measurement error alone (i.e. diagnostic test error). In future investigations of these relationships, I plan to investigate population and ecological processes that might contribute to detection probability

providing insight into mechanisms that might influence pathogen invasion and persistence. Using the hierarchical formulation in a dynamic occupancy framework might be a useful approach to explicitly link ecological processes to pathogen invasion and persistence.

My time as a PhD student has truly been a transformative experience. I intentionally sought to think deeply about policy processes from a political science perspective and also from an ecological lens. While challenging at times, this has altered the way I approach ecological problems allowing me to think much more broadly about how ecological processes and anthropogenic drivers are linked. In addition I have developed a broader and more quantitative approach to conceptualizing ecological lines of inquiry. Collectively this dissertation has broad applications for studying invasive species and the mechanisms that give rise to their population and disease dynamics offering a framework from which I can explore policy solutions and ecological drivers of disease and population dynamics. Additionally, incorporating data such as congressional policy activity, media, and social media such as Twitter that are not traditionally used in ecology may offer additional opportunities to investigate more directly how social processes shape both policy issues and biological systems. Doing this in a rigorous quantitative framework also offers opportunities to rigorously test new hypothesis and propose untested alternative hypotheses furthering overall understanding of these systems.

APPENDIX

Table A1.1. Susceptibility of seven species to OIE listed swine pathogens and the known status (present/absent) and reported prevalence in North American wild pigs. The table presents the results of the host susceptibility classification for 45 pathogens known to impact swine. In addition, the known status (present / absent) of the pathogen in North American wild pigs along with the reported prevalence range are included. If the pathogen was historically present but has been eradicated from the United States the year of eradication is noted. Wild pigs are included specifically to identify gaps in available scientific data for differences in susceptibility between domestic swine and wild pigs.

	Present in North America[1]	Wild pig Prevalence[2]	Study	Wild Swine	Domestic Swine	Cattle	Sheep	Goats	Poultry[3]	Cervids[4]	Horse	Humans	Citations
Bacterial													
Anthrax[5]	Yes			C	C	C	C	C	C	C	C	C	(Turnbull et al. 1992, Coetzer et al. 2004, Spickler 2007, Williams and Barker 2008, Spickler 2010, Zimmerman et al. 2012)
Bovine tuberculosis	Yes	2-85%	(Smith 1968, Essey et al. 1981)	C	C	C	C	C	U	C	C	C	(Williams and Barker 2008, Spickler 2009b, Zimmerman et al. 2012, Miller et al. 2013)
Brucellosis (bovine)	Yes	35%	(Stoffregen et al. 2007)	C	C	C	C	C		C	C	C	(Williams and Barker 2008, Spickler 2009a, Zimmerman et al. 2012)
Brucellosis (swine)[6]	Yes	0-68.8%	(Zygmont et al. 1982, Drew et al. 1992, Van Der Leek et al.	C	C	SC	O	O			C	C	(Coetzer et al. 2004, Spickler 2010)

[1] Year indicates date of eradication.
[2] Prevalence data shown only for North America.
[3] Includes only domestic chickens, ducks, geese, or turkeys.
[4] Includes only deer or elk that are commonly farmed in the U.S.
[5] Swine may develop chronic, persistent *Bacillus anthracis* infections of the oropharynx, and rarely of the intestinal tract.
[6] *(biovars 1, 2, and 3 affect swine; 1 and 3 are in the U.S. while 2 is in Europe. Biovar 1 is endemic in U.S. feral swine)*

			1993a, Gresham et al. 2002, Stoffregen et al. 2007, Pedersen et al. 2012)										
Brucellosis (melitensis)	1999				C	C	C	C			C	C	(Coetzer et al. 2004, Spickler 2010, Zimmerman et al. 2012)
Hemorrhagic septicemia	Yes				C	C	C	C		C	C		(Coetzer et al. 2004, Spickler 2009c, 2010)
Leptospirosis**	Yes	8-87%	(New Jr et al. 1994, Chatfield et al. 2013)	C	C	C	C	C		C	C	C	(Williams and Barker 2008, Zimmerman et al. 2012, Spickler 2013)
Paratuberculosis (Johne's disease)	Yes			C	C	C	C	C	U	C	EX	U	(Spickler 2007, Williams and Barker 2008, Zimmerman et al. 2012)
Q Fever	Yes	50%	(Randhawa et al. 1977)	A	A	C	C	C	A	A	A	C	(Spickler 2007)
Tularemia	Yes	1.3%	(Hartin et al. 2007)	C	C	C	C	C	U	A	C	C	(Morner 1992, Coetzer et al. 2004, Williams and Barker 2008, Spickler 2010)
Viral													
African swine fever	N/R			C	C								(Coetzer et al. 2004, Spickler 2010)
Pseudorabies virus	Yes	7-61%	(Van der Leek et al. 1993b, Gresham et al. 2002, Corn et al. 2004, Müller et al. 2011)	C	C	C	C	C*	U	C*	O*		(Coetzer et al. 2004, Williams and Barker 2008, Spickler 2010, Zimmerman et al. 2012)
Influenza (avian)	Yes	1-14.4%	(Hall et al. 2008, Feng et al. 2014)	C	C	A	SC	SC	C	SC	C	C	(Guo et al. 1992, Guo et al. 1995, Cook 2005, Olsen et al. 2006, Kalthoff et al. 2008, Lipatov et al. 2008)
Influenza (equine)	Yes				C	EX			A	U	C	C	(Morens and Taubenberger 2010, Spickler 2014)

Bluetongue	Yes					SC	C	C		C		O	(Spickler 2010)
Bovine viral diarrhea virus	Yes	0%	(New Jr et al. 1994)		C	C	SC	SC		SC			(Passler et al. 2007, Duncan et al. 2008, Williams and Barker 2008, Zimmerman et al. 2012)
Classical swine fever	2015	0%	(Nettles et al. 1989)	C	C								(Coetzer et al. 2004, Williams and Barker 2008, Spickler 2010)
Crimean-Congo hemorrhagic fever	N/R				SC	SC	SC	SC			SC	C	(Spickler 2010)
Eastern equine encephalomyelitis	Yes	16.5%	(Brody and Murray Jr 1959, Elvinger et al. 1996)	C	C*	C*	C*	C*	C	C*	C	C*	(Tate et al. 2005, Schmitt et al. 2007, Spickler 2008)
Epizootic hemorrhagic disease	Yes					C	EX			C			(Spickler 2010, Ruder et al. 2012, Breard et al. 2013)
Foot and mouth disease	1947			C	C	C	C	C		C		C	(Coetzer et al. 2004, Spickler 2010)
Heartwater	N/R					C	C	C		EX			(Dardiri et al. 1987, Spickler 2010)
Infectious bovine rhinotracheitis	Yes				C	C		C		A			(Williams and Barker 2008)
Japanese encephalitis	N/R				C	SC	SC	SC	SC		C*	C*	(Emord and Morris 1984, Kumar 1999, Coetzer et al. 2004, Tate et al. 2005, Schmitt et al. 2007, Spickler 2010, Zimmerman et al. 2012)
Malignant catarrhal fever**	Yes				C*	C*	SC	SC		C*			(Williams and Barker 2008, Spickler 2010, Zimmerman et al. 2012)
Nipah virus encephalitis	N/R				C		U	C			C	C	(Coetzer et al. 2004, Spickler 2010, Zimmerman et al. 2012)
Peste des petits ruminants virus	N/R				EX*	SC*	C	C		EX			(Coetzer et al. 2004, Aitken 2008, Spickler 2010)

Porcine epidemic diarrhea virus**	Yes				C								(2005, Stevenson et al. 2013)
Porcine reproductive and respiratory syndrome**	Yes	1-3%	(Saliki et al. 1998, Corn et al. 2009, Wyckoff et al. 2009)	C	C								(Williams and Barker 2008, Corn et al. 2009)
Rabies	Yes				C	C	C	C		C	C	C	(Coetzer et al. 2004, Spickler 2010)
Rift Valley fever	N/R				A	C	C	C	A			C	(Scott 1963, Spickler 2010)
Rinderpest	N/R				C	C	C	C					(Barrett and Rossiter 1999, Rossiter et al. 2001)
Swine vesicular disease	N/R				C								(Coetzer et al. 2004, Spickler 2010)
Transmissible gastroenteritis	Yes	0%	(Woods et al. 1990, Saliki et al. 1998)	C	C								(Williams and Barker 2008)
Venezuelan equine encephalomyelitis	1971				C	C	C*	C*	SC		C	C	(Coetzer et al. 2004, Spickler 2010)
Vesicular stomatitis	Yes	0-100%[7]	(Stallknecht et al. 1985, Stallknecht et al. 1986, Stallknecht et al. 1993)	C	C	C	C	C	A, EX	A	C	C	(Webb et al. 1987, Coetzer et al. 2004, Williams and Barker 2008, Spickler 2010)
West Nile virus	Yes	16.1-32.1%	(Gibbs et al. 2006)	SC*	SC*	SC*	C*	SC*	C	C*	C*	C*	(Miller et al. 2005, Van der Meulen et al. 2005, Kramer et al. 2007, Nemeth and Bowen 2007, Spickler 2010)
Parasitic													
Trichinellosis	Yes	13.3%	(Sandfoss et al. 2011)	C	C						SC	C	(Murrell et al. 1987, Gajadhar et al. 1997, Coetzer et al. 2004)
Echinococcosis	Yes			C, IH	C, IH	C, IH	C, IH	C, IH		C, IH	C, IH	C, IH	(Leiby et al. 1970, Storandt and Kazacos 1993, Storandt

[7] Endemic in feral swine living on Ossabaw Island in Georgia.

													et al. 2002, Thompson et al. 2006, Spickler 2010, 2011)
Leishmaniasis	N/R				SC*	C	C	C			C	C	(Coetzer et al. 2004, Spickler 2010)
New world screwworm	1990				C	C	C	C	C	C	C	C	(Coetzer et al. 2004, Spickler 2010)
Old world screwworm	N/R				C	C	C	C	C	C	C	C	(Coetzer et al. 2004, Spickler 2010)
Porcine cysticercosis	Yes	42-59.2%	(Corn et al. 2009, Baker et al. 2011, Sandfoss et al. 2012)	C, IH	C, IH		O, IH			O, IH		C, DH, IH	(Coetzer et al. 2004, Spickler 2010)
Surra (T. evansi)	N/R				C	C	O	O		C	C	O	(Coetzer et al. 2004, Spickler 2010)
Trypanosomiasis (tsetse transmitted)	N/R				C	C	C	C		U	C	C	(Spickler 2010)

Table A3.1. Species susceptible to pseudorabies virus that were used to calculate the Shannon diversity index.

Family	Wild Species	Domestic Species	Susceptibility	Citation
Bovidae	*B. bison, O. americanus, O. canadensis*	*B. taurus, C. aegagrus, O. aries*	Clinical	(Mocsári et al. 1987, Schmidt et al. 1987, Mocsári et al. 1989, Power et al. 1990, Rademacher et al. 1991, Jin et al. 1992, Schmidt and Kluge 1992, Yildirim et al. 2017)
Canidae	*C. latrans, C. lupus, C. rufus, U. cinereoargenteus, U. littoralis, V. macrotis, V. velox, V. vulpes*		Clinical	(Palic 1985, Raymond et al. 1997, Cramer et al. 2011, Steinrigl et al. 2012, Caruso et al. 2014, Verpoest et al. 2014, Zhang et al. 2015)
Felidae	*L. pardalis, L. canadensis, L. rufus, P. onca, P. concolor, P. yagouaroundi,*		Clinical	(Hara et al. 1991, McLean et al. 1994, Thiry et al. 2013)
Procyonidae	*B. astutus, P. lotor,*		Clinical	(Thawley and Wright 1982, Platt et al. 1983, Xiao 1985, Goyal et al. 1986)
Mustelidae	*G. gulo, L. Canadensis, M. americana, M. pennanti, M. erminea, M. frenata, M. nigripes, M. nivalis, N. narica, N. vison, T. taxus,*		Clinical	(Kimman and Van Oirschot 1986, Quiroga et al. 1997, Marcaccini et al. 2008)
Ursidae	*U. americanus, U. arctos, U. arctos,*		Clinical	(Pirtle et al. 1986, Schultze et al. 1986, Zanin et al. 1997, Banks et al. 1999)
Equidae		*E. ferus*	Clinical	(van den Ingh et al. 1990, Kimman et al. 1991, Sakkubai and Ramachandran 1992)
Tayassuidae	*P. tajacu*		Clinical and Sub-clinical	(Crandell et al. 1986, de Castro et al. 2014)

Table A3.2. Species susceptible to *Brecella suis* biovar 1 or 2 that were used to calculate the Shannon diversity index.

Family	Wild Species	Domestic Species	Susceptibility	Citation
Bovidae	*B. bison, O. americanus, O. canadensis*	*B. taurus, C. aegagrus, O. aries*	Sub-clinical	(Norton and Thomas 1979, Cook and Noble 1984, Reddy and Rao 1984, Drew et al. 1992, Paolicchi et al. 1993, Ewalt et al. 1997, Lucero et al. 2008, Tae et al. 2012)
Leporidae	*B. idahoensis, L. alleni, L. americanus, L. californicus, L. callotis, L. townsendii, S. aquaticus, S. audubonii, S. bachmani, S. cognatus, S. floridanus, S. nuttallii, S. obscurus, S. palustris, S. robustus, S. transitionalis*		Clinical and Sub-clinical	(Tworek and Serokowa 1956, Thorpe et al. 1965, Szyfres et al. 1968, Stěrba 1982, 1984, Gyuranecz et al. 2011, Fort et al. 2012)
Canidae	*C. latrans, C. lupus, C. rufus, U. cinereoargenteus, U. littoralis, V. macrotis, V. velox, V. vulpes*		Clinical and Sub-clinical	(Hellmann and Sprenger 1978, Kormendy and Nagy 1982, Barr et al. 1986, Thanappa et al. 1990, Lucero et al. 2008, Ramamoorthy et al. 2011, Mor et al. 2016)
Equidae		*E. ferus*	Clinical and Sub-clinical	(Portugal et al. 1971, Cvetnic et al. 2005)
Tayassuidae	*P. tajacu*		Clinical and Sub-clinical	(Lord and Lord 1991, Mayor et al. 2006)

Table A3.3. Hyperprior distributions used to parameterize the informative 'expert' beta distribution for sensitivity and specificity for both pathogens.

Parameter	Definition	Distribution
Pseudorabies virus		
μ_{ρ_k}	Mean for true positive detection rate assumed with 65% confidence to be 0.984	beta(2.089, 1.018)
σ_{ρ_k}	Variance for mean of true positive detection rate	gamma(1.0031, 0.001)
μ_{ϕ_k}	Mean for true negative detection rate assumed with 65% confidence to be 0.99	beta(2.076, 1.01)
σ_{ϕ_k}	Variance for mean of true negative detection rate	gamma(1.003, 0.001)
Swine brucellosis		
μ_{ρ_k}	Mean for true positive detection rate assumed with 65% confidence to be 0.94	beta(2.2, 1.077)
σ_{ρ_k}	Variance for mean of true positive detection rate	gamma(1.0032, 0.001)
μ_{ϕ_k}	Mean for true negative detection rate assumed with 65% confidence to be 0.97	beta(2.121, 1.035)
σ_{ϕ_k}	Variance for mean of true negative detection rate	gamma(1.0031, 0.001)

Table A4.1. Studies reporting vital rates for wild pigs used in our analysis.

Study	Country / State	Invasive /Native	Survival			Fecundity		
			Juvenile	Yearling	Adult	Juvenile	Yearling	Adult
(Barrett 1971)	California	Invasive	0.86	0.71	0.42	5.30	5.20	6.93
	California	Invasive	0.86	0.71	0.42	2.58	5.00	5.05
(Hanson et al. 2009)	Georgia	Invasive	0.23	-	0.28	4.80	-	6.40
(Giles 1980)	New South Wales	Invasive	0.07	0.61	0.63	4.75	5.85	6.93
	New South Wales	Invasive	0.77	0.73	0.66	4.67	5.86	7.38
(Singer and Ackerman 1981)	Tennessee	Invasive	0.39	0.76	0.76	2.93	4.20	4.79
(Gabor et al. 1999)	Texas	Invasive	0.69	-	0.69	3.00	-	5.60
(Gamelon et al. 2011)	France	Native	0.77	0.40	0.28	0.17	4.37	5.93
(Gamelon et al. 2012)	France	Native	0.86	0.49	0.86	0.23	4.86	6.12
(Bieber and Ruf 2005)	Germany	Native	0.52	0.60	0.71	4.50	6.50	6.80
	Germany	Native	0.25	0.31	0.58	3.50	4.50	6.30
	Germany	Native	0.33	0.40	0.66	4.00	5.50	6.50
(Diong 1982)	Hawaii	Native	0.35	0.28	0.23	3.50	5.80	6.80
(Boitani et al. 1995)	Italy	Native	0.63	0.59	0.54	1.90	4.99	5.03
(Neet 2014)	Switzerland	Native	0.55	0.91	0.20	2.17	5.12	6.23
(Moretti 2014)	Switzerland	Native	0.78	0.21	0.43	2.40	5.40	5.80

Table A4.2. Environmental conditions investigated in our statistical models for invasive and native populations. The predicted relationship indicates our hypothesized relationship between the amount of transients and the environmental condition.

Predictor	Data Source	Predicted Relationship	Supporting Citation for Prediction
Climatic			
Annual Mean Temperature (BIO1)	BioClim World Climate Data (Hijmans et al. 2005)	Negative	(Jedrzejewska et al. 1997, Sabrina et al. 2009, Frauendorf et al. 2016)
Temperature Annual Range (BIO7)		Positive	(Geisser and Reyer 2005, McClure et al. 2015)
Precipitation Seasonality (BIO15)		Positive	(Massei et al. 1997, Geisser and Reyer 2005, Sabrina et al. 2009)
Precipitation of Warmest Quarter (BIO18)		Negative	(Sabrina et al. 2009, Frauendorf et al. 2016)
Precipitation of Coldest Quarter (BIO19)		Positive	(Geisser and Reyer 2005)
Habitat			
Deciduous Broadleaf Trees	Global Consensus Land Cover (Tuanmu and Jetz 2014)	Positive; linear	(Geisser and Reyer 2005, Honda 2009, Frauendorf et al. 2016)
Cultivated and Managed Vegetation		Positive; nonlinear	(Geisser and Reyer 2005, Honda 2009, Morelle and Lejeune 2015)

Table A4.3. Differences in the reported vital rates for invasive and native populations.

Lower Level Vital Rate		ANOVA			Welch t-test			Wilcoxon rank sum test		
		F	P-value	Adjusted P-value	t	P-value	Adjusted P-value	F	P-value	Adjusted P-value
Fecundity	Juvenile	2.668	0.141	0.281	-1.779	0.048	0.097	24	0.796	0.943
	Yearling	7.886	0.017	0.052	-2.195	0.024	0.073	32	0.12	0.286
	Adult	0.176	0.681	0.817	-0.406	0.346	0.415	17	0.943	0.943
Survival	Juvenile	0.003	0.959	0.959	0.055	0.478	0.478	16	0.056	0.286
	Yearling	10.096	0.01	0.052	-2.431	0.016	0.073	31	0.143	0.286
	Adult	0.25	0.625	0.817	-0.483	0.318	0.415	19	0.916	0.943

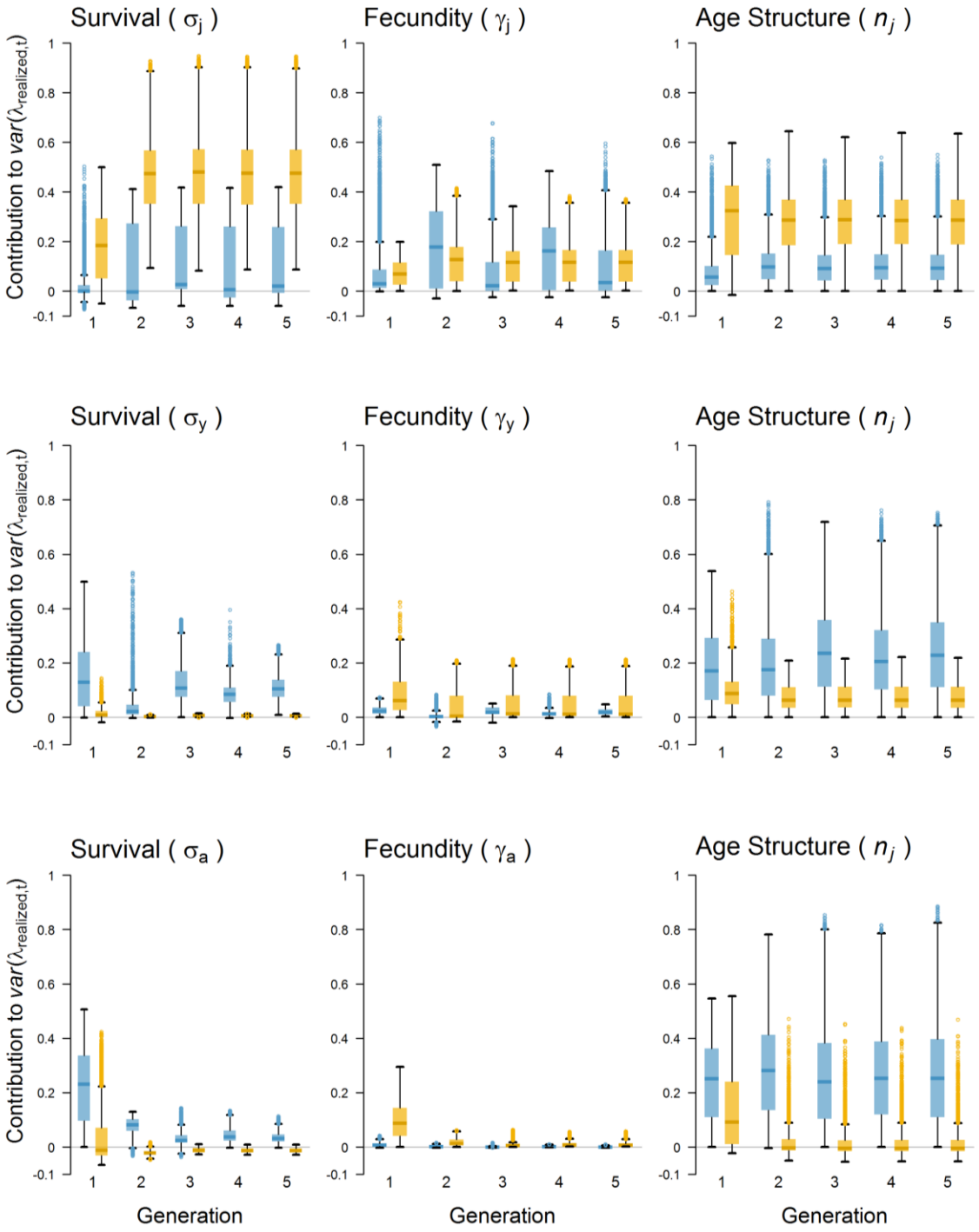


Figure A4.1. The contribution (χ_{θ_k}) of age specific vital rates, survival ($\chi_{\sigma_{k,i}}$), fecundity ($\chi_{\gamma_{k,i}}$) and age structure ($\chi_{n_{k,i}}$), to variation in realized growth rate ($\lambda_{realized,t}$) for the first five generations. The contribution to the variance in $\lambda_{realized,t}$ results from the covariance (Eq. 7) among each pair of parameters that includes survival, fecundity, and age structure.

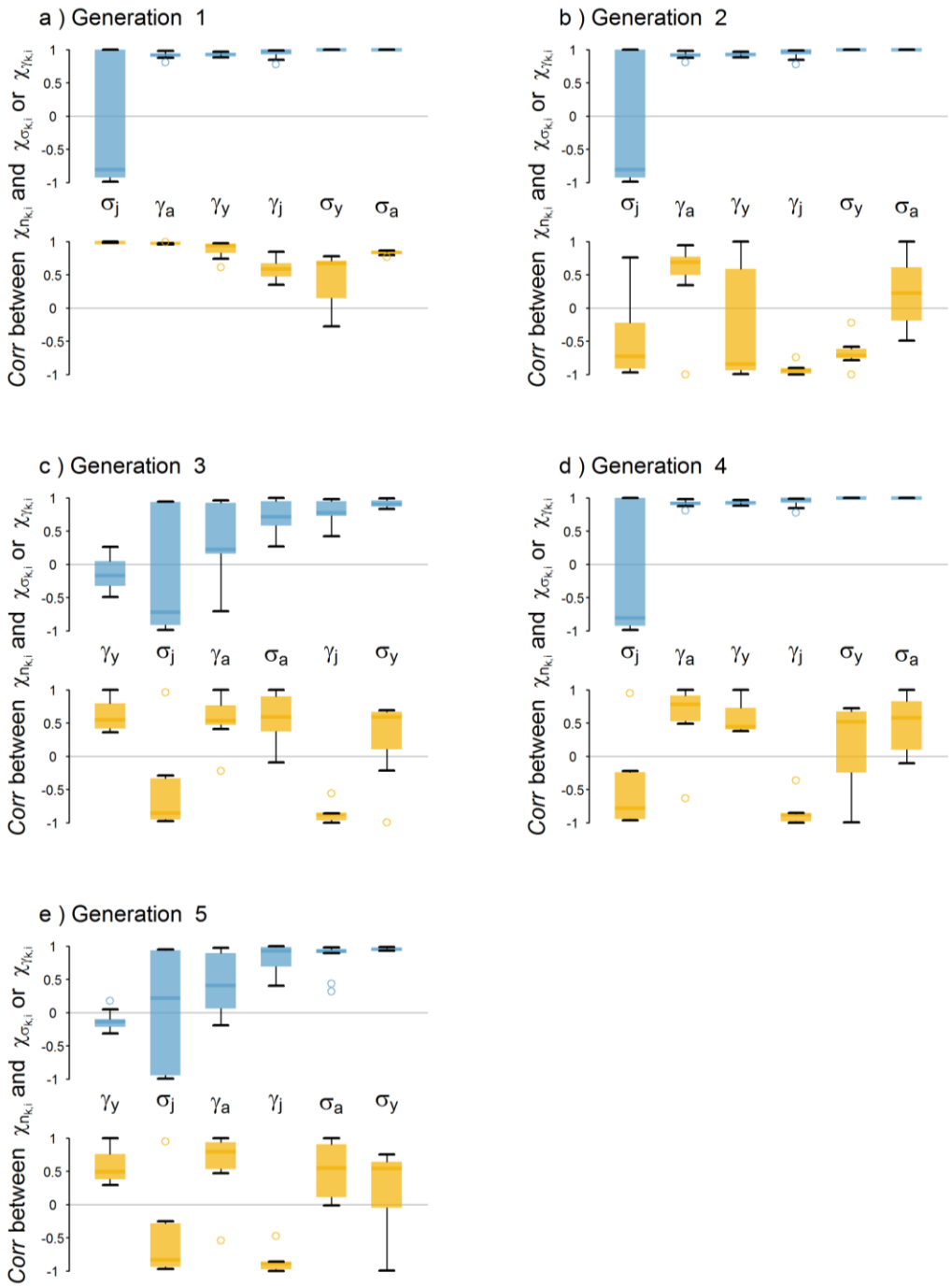


Figure A4.2. The correlation between the contribution of age structure ($\chi_{n_{k,i}}$) and survival ($\chi_{\sigma_{k,i}}$) or fecundity ($\chi_{\gamma_{k,i}}$) to variation in realized population growth rates, $\lambda_{realized,t}$. Boxplots describe the direction of the relationship (i.e. positive or negative) between the contribution of age structure, survival, and fecundity for all populations in our study measured using Spearman's rank correlation. Boxplots are in mean rank order for the direction of relationship for native populations (blue). The relationship is generally positive for native populations while the corresponding vital rates for invasive populations (yellow) have generally larger variation in the direction of relationship and are often negative.

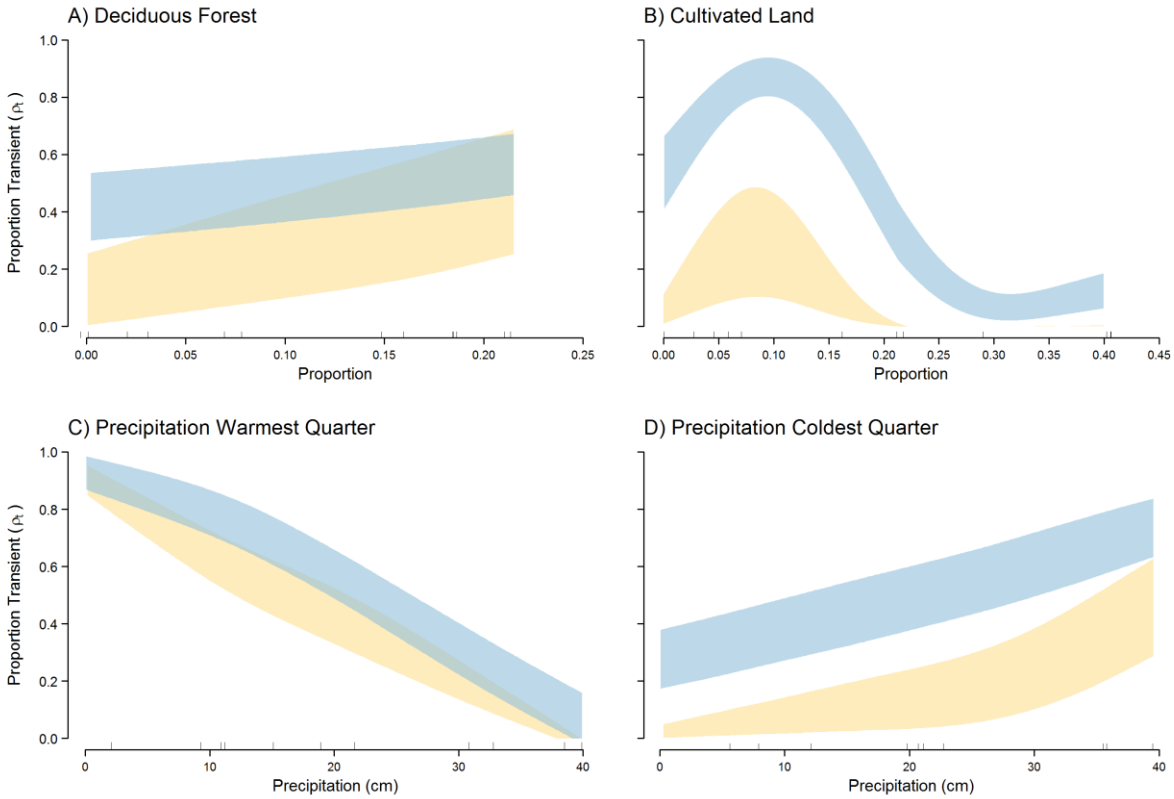


Figure A4.3. Relationship between the ratio of transient dynamics ($\boldsymbol{\rho_t}$) and four environmental conditions. Shaded regions represent the range of expected amount of transient dynamics estimated using restricted maximum likelihood from the first time-step to the fifth time-step.