CONSEQUENCES OF ANIMAL MIGRATION IN AN ERA OF GLOBAL CHANGE

A DISSERTATION
SUBMITTED TO THE FACULTY OF THE UNIVERSITY OF MINNESOTA
BY

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IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

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2025

Acknowledgements

Completing a Ph.D. is an individual endeavor in the same way that cross-country running or skiing are individual sports, which is to say, not really at all. At times, writing this dissertation has felt solitary; reading, writing, and thinking for hours upon hours of effort in pursuit of a distant and uncertain payoff. However, these efforts were made possible by a wonderful community of people who have encouraged me to think in new ways, supported me through challenges, and made the journey towards my Ph.D. more joyful.

First, I would like to thank my advisor, Dr. Allison Shaw. It is difficult to overstate the importance of a Ph.D. advisor, and I feel enormously fortunate to have found Allison. Allison is a wonderful advisor in ways that I did not know to look for when I was applying to graduate school. In addition to supporting the writing and development of these three chapters, she has provided intentional mentorship on everything it means to be a scientist. For example, she has helped me to reflect on my work, plan and execute projects, serve my community, and balance my work as a scientist with the rest of my life. She emphasizes that *people* do science and is a model for humanity and integrity in academia.

I would also like to thank other academic mentors. My committee members, Dr. Jessica Hellmann, Dr. Sarah Hobbie, and Dr. Kate Meyer, have supported the writing of these chapters, provided mentorship on career development, and served as wonderful academic role models. I am also grateful for the generosity of Dr. Mary Heskel, Dr. Jake Grossman, and Dr. Kate Meyer (again!) who have provided mentorship on teaching careers at small liberal arts colleges and helped me access opportunities to teach at Macalester, St. Olaf, and Carleton! I would also like to thank my undergraduate mentors, Dr. Dan Hernandez

and Dr. Eric Egge, for getting me started as a mathematician and ecologist and continuing to believe in me.

The graduate student community in EEB has also helped my intellectual development and made life more fun! Starting graduate school during a global pandemic was odd, to say the least. Sharing the experience of Zoom University with the Co(V)hort (Aarcha, Alyssa, Anya, Ave, Charlotte, Hanan, James, Lili, Maria K, Maria P., Mariana, Nishia) made it more manageable. My office mates (Aarcha, Katie P., Maria K.) have made coming to work fun. The Shaw Lab and Shaw Lab Friends™ (Naven, Dennis, Steph, Ching-Lin, Peter, Amanda, Ruby, Cece, Jess, Aarcha, Alyssa, Ave, Chris, Matt) have helped me learn how to be a theorist and have been great to bounce ideas around with.

My students and mentees have helped me to think more broadly, express myself more clearly, and reconnect with why I started doing this in the first place. So, thank you to my students in Foundations of Biology Part 1 Lab, Ecology: Theory and Concepts, Big Data in Ecology, and Global Change Biology and to my mentees, Cece, Jess, and Ruby.

Last, and certainly not least, I would like to thank my friends and family. I am incredibly lucky to have spent the past years living in the Twin Cities, close to my people. My mom has walked to work with me, given me treats to take home after Sunday dinner, and shown me that it's possible to balance academia with a full life. My dad sparked my love of the outdoors and is always curious about my work and proud of me. My sister, Lisa, has been my companion in life since she came on the scene 27 years ago and reminds me of the importance of fun. Torte, my sibling turned student, is a model of passion for science and nature and unquenchable curiosity. Sam, my partner for the last 3 years, has supported me through the most challenging moments, celebrated my victories with me, and made every day better. The rest of my family in Minnesota continues to show up for each other and remind me of what really matters. I am so lucky to call all of you home.

Dedication

To my family.

Who have given me
the courage to venture out,
the strength and wisdom to enjoy the journey,
and a loving home to return to.

Abstract

Due to the importance of migration as an adaptation to environmental variation for many animals, it is clear that migration mediates the consequences of environmental change for migratory populations. Migration also clearly mediates the consequences of environmental change for broader ecosystems whenever changes to migratory strategies and migratory population sizes affect the ecological role of migrants. However, because both migration and environmental change are complex phenomena, it is less clear how migration mediate the consequences of environmental change. In this thesis I develop ecological theory to elucidate ways in which migration might mediate the consequences of climate change for migratory populations (Theme 1, Chapters 1 and 2) and the consequences of environmental change for disease dynamics (Theme 2, Chapter 3). In Chapter 1, I build a model to understand the role of migratory cues in determining the severity of phenological mismatch experienced by migrants following phenological change. In Chapter 2, I explore how spatial aggregation of populations mediates the effect of spatially restricted disturbances, like extreme climate events. In Chapter 3, I determine how two traits associated with migration, tolerance to infection and pace of life, affect pathogen trait evolution and the impact of disease on populations following spillover events. Together, these projects advance our understanding of how migration mediates the consequences of environmental change. This in turn improves our ability to make predictions and design effective conservation strategies in our current era of rapid environmental change.

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Introduction

We are living in an era of profound global change, defined by the intertwined crises of climate change and biodiversity loss (Pörtner et al. 2023). Climate change has manifested not just in warming temperatures, but also in changing patterns of precipitation, more frequent and severe extreme climate events, and massive wildfires (Crimmins 2023). The velocity of climate change, along with the specific challenges and opportunities for life on earth posed by climate change, vary across the earth (Loarie et al. 2009, Crimmins 2023). As animals respond to climate change, they also respond to other global change factors including land use change, pollution, overexploitation by people, rapid human transport of organisms, and the changes to ecological interactions that result from all of these factors (Pörtner et al. 2023). The consequences of these changes for animal life on earth have been numerous and varied. Some populations are in decline while others have expanded their ranges. The effects of global change for animals often impact humans. For example, disease spillover events, which can result in disease outbreaks in humans and livestock, often have a change in the ecology of an animal host at their root (Daszak et al. 2000).

As a defining feature of the lives of many animals, movement mediates the response of populations, ecological communities, and ecosystems to global change (Nathan et al. 2008). Movement is one way that animals can adapt to environmental change (Parmesan and Yohe 2003, Nathan et al. 2008, Loarie et al. 2009). Animals are not always able to quickly adapt their movement patterns to novel environmental conditions (Sutherland 1998, Faaborq et al. 2010, Sawyer et al. 2019) and failure to adapt movement patterns to global change can lead to loss of fitness and population declines (Møller et al. 2008). Animal movement affects ecological processes by transporting nutrients, energy, pathogens and propagules and modifying trophic interactions (Bauer and Hoye 2014). When animal movement patterns change or populations of moving animals decline, there are ecosystem consequences (Seebacher and Post 2015), including changes in the ability of plants to track climate change (Fricke et al. 2022) and changes to the carbon cycle (Schmitz and Sylvén 2023). While it is abundantly clear *that* animal movement mediates the

consequences of global change, due to the complexity of both animal movement and global change, it remains unclear exactly *how.*

One particularly fascinating form of movement in the context of global change is animal migration. Migration can be defined as a seasonal to-and-fro movement of a population between locations where conditions are alternately favorable and unfavorable (Dingle and Alistair Drake 2007). In many cases, migration can be understood as an adaptation to the fact that the environment varies over time (Stratmann et al. 2023). In order to take advantage of seasonally advantageous conditions, animals move. This has historically been an enormously successful strategy, observed in all major vertebrate lineages as well as in many invertebrates, with flocks and herds and schools defined by their abundance as much as by their movements (Fryxell et al. 1988, Dingle and Alistair Drake 2007, Kauffman et al. 2021, Stratmann et al. 2023). Migration benefits many animals in a world where environmental conditions vary over space and time. However it is unclear what the consequences of migration will be in a world filled with long-term, directional changes in environmental conditions.

In light of the lack of clarity about *how* migration mediates the consequences of global change, theory development is a useful tool. The development of ecological theory using mathematical models clarifies the link between mechanisms and patterns (Servedio et al. 2014, Shaw et al. 2024), provides narratives that structure our understanding of empirical evidence, and generates predictions for further testing (Otto and Rosales 2020). This theoretical work can be used to identify what mechanisms may be at play in different empirical situations, design empirical studies that allow us to evaluate the presence and strength of these mechanism/pattern relationships, make predictions, and design conservation strategies that address the root cause of problems we seek to solve (Grainger et al. 2021).

In this thesis, I explore two themes by developing ecological theory. In Theme 1, covered by Chapters 1 and 2, I explore how migration mediates the consequences of global change for migratory populations. In Theme 2, covered by Chapter 3, I explore how migration mediates the consequences of global change for disease dynamics.

Theme 1: How does being migratory mediate the population consequences of global change?

We have witnessed the decline of many migratory populations in response to habitat destruction, barriers to migration, overexploitation and climate change (Wilcove and Wikelski 2008, Brower et al. 2012, Bairlein 2016). However, establishing a clear link between migration and vulnerability to global change factors is challenging due to mixed empirical evidence and a wide variety of mechanistic possibilities. Empirically, we see cases in which migratory populations have declined and cases where they have not (Both et al. 2006, 2010, Nilsson et al. 2006, Post and Forchhammer 2008, Møller et al. 2008, Mallory et al. 2020). When we focus on climate change, some of the declines have been attributed to climate change while others have been attributed to other global change factors or interactions between climate change and other global change factors (Robinson et al. 2009, Both et al. 2010, Rushing et al. 2020, Kauffman et al. 2021). Several mechanisms have been posed to explain why migratory populations are more vulnerable to climate change while other mechanisms have been posed to explain why migratory populations might be especially resilient to climate change (Wilcove 2008, Robinson et al. 2009, Rubenstein and Hack 2013, Gill 2015, Finch et al. 2017, Kauffman et al. 2021, Gurarie et al. 2021). Again, while it may seem obvious *that* migration mediates the population consequences of global change, it is unclear exactly *how* and what distinguishes the cases where migration is associated with population declines due to climate change from cases where it is not (Robinson et al. 2009, Seebacher and Post 2015, Shaw 2016). This means our understanding of the conditions under which migration makes animals more or less vulnerable to climate change is limited, which limits our ability to allocate conservation resources effectively and design effective conservation strategies.

The first step towards developing theory to understand how migration mediates the population consequences of global change is to consider what mechanisms might be at play. In the case of understanding how migration mediates the consequences of climate change for populations, this looks like asking: 'what aspects of climate change might impact migratory populations?' and 'what about being migratory influences the response of populations to these aspects of climate change?' Due to the complexity of the

migration syndrome and of climate change, there are many answers to these questions. Many aspects of climate change, including warming temperatures, phenological change, or increasing severity and frequency of extreme climate events might influence migratory populations. Although animal migrants are a diverse group, migratory populations have many shared characteristics that potentially affect response to climate change including use of multiple habitats, tendency to aggregate, movement according to precise schedules, ability to exploit ecological opportunities and environmental variation, and capacity for long distance movement (Alerstam et al. 2003, Rubenstein and Hack 2013, Somveille et al. 2018). After determining a mechanism of interest, we can explore what patterns it can generate (Shaw et al. 2024). We can also consider differences among migratory populations as we seek to understand the conditions under which the mechanism generates different patterns (Shaw et al. 2024). Finally, we can explore how the mechanism interacts with other mechanisms that might be at play (Shaw et al. 2024).

In Chapters 1 and 2 of this thesis, I explore two ways in which migration could mediate the response of populations to specific aspects of climate change.

Chapter 1: How do migration cues mediate the population effects of phenological change?

First, we consider that by definition, migrants move between habitats and that migrants often exploit ecological opportunity and variation and use precise schedules to do so (Alerstam et al. 2003, Rubenstein and Hack 2013). These similarities may affect the response of populations to phenological change associated with climate change and the fact that phenological change is happening unevenly around the world (Gordo 2007, Jones and Cresswell 2010, Cohen et al. 2018). If the fitness benefit of migration relies on the timing of migration, and the cues that govern when migration occurs become decoupled from the ecological opportunity migration allows animals to exploit (i.e. phenological mismatch occurs), there could be fitness consequences (Møller et al. 2008, Robinson et al. 2009, Both et al. 2010, Shaw 2016, Connare and Islam 2022).

The extent to which this mechanism can lead to a decline in migratory populations depends in part on what cue is used to decide when to migrate because this

determines the extent to which the cue becomes desynchronized from the ecological opportunity. Migratory animals use a wide variety of cues to trigger migration including photoperiod and habitat quality based cues like green-up or snowmelt (Shaw and Couzin 2013, Kölzsch et al. 2016, Bastille-Rousseau et al. 2019, Gurarie et al. 2019, Kauffman et al. 2021, Cameron et al. 2021). While cue type has been asserted as a reason why some migratory populations are in decline while others are not, cue type is challenging to disentangle from variables like migration distance in empirical case studies (Both et al. 2006, Post and Forchhammer 2008, Laforge et al. 2021). In Chapter 1, using a mathematical model, I isolated the effect of cue type on the severity of phenological mismatch in migratory animals.

Chapter 2: How does population aggregation mediate the population effects of extreme climate events?

Spatial aggregation is a defining feature of migratory populations (Fryxell et al. 1988, Rubenstein and Hack 2013), though there is variation in the degree to which migratory populations are spatially aggregated (Gilroy et al. 2016, Finch et al. 2017). When we think about migration, the images that might come to mind include monarch butterflies blanketing trees in Mexico, enormous flocks of sandhill cranes in Nebraska, and coursing herds of african ungulates. High levels of population spatial aggregation could mean that disturbances in relatively small areas have a large impact on populations (Finch et al. 2017, Lisovski et al. 2021). In the context of climate change, spatial aggregation of a population can mediate the effect of the increase in frequency and severity of extreme climate events.

Empirically, we have seen many cases in which migratory populations are affected by extreme climate events and other climate related disturbances including cold spells (Briedis et al. 2017), winter storms (Cohen et al. 2021, Acker et al. 2021), wildfires (Overton et al. 2022), and climate anomalies (Boano et al. 2020). In addition to spatial aggregation, there are other aspects of space use that can affect population response to climate related disturbance including site fidelity (Kreling et al. 2021) and the ability to systematically avoid locations affected by disturbance and seek out suitable locations (Boucek et al. 2017, Kreling et al. 2021, Acker et al. 2021, Stratmann et al. 2023). In Chapter 2,

using a mathematical model, I isolated the effect of spatial aggregation on population response to spatially and temporally restricted disturbances.

Theme 2: How do migratory animals mediate the consequences of global change for disease dynamics?

Chapter 3: How do host traits associated with migration affect pathogen evolution following spillover?

Global changes including climate change, land use change, and biotic exchange, can alter contact patterns between potential hosts and therefore patterns of disease spillover (Brooks and Hoberg 2007, Altizer et al. 2013, 2018, Plowright et al. 2021, Baker et al. 2022). Novel interactions between migratory animal populations and other species can occur due to changes to migration routes and changes to the ecosystems that these routes pass through. These novel interactions can lead to disease spillover events, including spillover events between migratory and non-migratory populations (Daszak et al. 2000, Fuller et al. 2012, Satterfield et al. 2018, Rayl et al. 2021). The impact of disease spillover events on populations depends on how the transmission rate and virulence of pathogens evolve following spillover. Pathogen evolution is influenced by host characteristics including resistance to infection, tolerance to infection, recovery rate, rate of contact with susceptible hosts and pathogen-independent mortality rate (Anderson and May 1982, Altizer 2001, Power and Mitchell 2004, Roode et al. 2006, Alexander 2007, Alizon et al. 2008, Ashander et al. 2012, Messinger and Ostling 2013, Ugelvik et al. 2017). There are many ways in which migratory animals might differ from migratory animals in traits relevant for pathogen evolution. For example, there is evidence that migratory animals differ in their pace of life (Soriano-Redondo et al. 2020), immunocompetence (Poulin and de Angeli Dutra 2021) and tolerance to infection. Therefore, it seems very likely that host population migration will influence pathogen evolution following spillover. However, again, describing *how* host migration influences pathogen evolution is made difficult by mixed empirical evidence and a wide variety of mechanistic possibilities.

In order to make predictions about how such spillover events will affect pathogen evolution, it is valuable to think about how the multiple mechanisms that may be at play interact with each other (Shaw et al. 2024). In Chapter 3, I explore how two host traits, pace of life and tolerance to infection, interact to determine how pathogen transmission rate and virulence evolve following spillover between a migratory and non-migratory host population and go on to illustrate how this affects host population dynamics.

References

Acker, P., F. Daunt, S. Wanless, S. J. Burthe, M. A. Newell, M. P. Harris, H. Grist, J. Sturgeon, R. L. Swann, C. Gunn, A. Payo-Payo, and J. M. Reid. 2021. Strong survival selection on seasonal migration versus residence induced by extreme climatic events. Journal of Animal Ecology 90:796–808.

Alerstam, T., A. Hedenström, and S. Åkesson. 2003. Long-distance migration: Evolution and determinants.

Alexander, D. J. 2007. An overview of the epidemiology of avian influenza. Vaccine 25:5637–5644.

Alizon, S., A. E. M. J. Keeling, and E. M. C. Whitlock. 2008. Transmission-Recovery Trade-Offs to Study Parasite Evolution. The American Naturalist 172:E113–E121.

Altizer, S. 2001. Migratory behaviour and host-parasite co-evolution in natural populations of monarch butterflies infected with a protozoan parasite. Evol. Ecol. Res. 3.

Altizer, S., D. J. Becker, J. H. Epstein, K. M. Forbes, T. R. Gillespie, R. J. Hall, D. M. Hawley, S. M. Hernandez, L. B. Martin, R. K. Plowright, D. A. Satterfield, and D. G. Streicker. 2018. Food for contagion: Synthesis and future directions for studying host –parasite responses to resource shifts in anthropogenic environments. Philosophical Transactions of the Royal Society B: Biological Sciences 373.

Altizer, S., R. S. Ostfeld, P. T. J. Johnson, S. Kutz, and C. D. Harvell. 2013. Climate change and infectious diseases: From evidence to a predictive framework. Science.

Anderson, R. M., and R. M. May. 1982. Coevolution of hosts and parasites. Parasitology 85:411–426.

Ashander, J., M. Krkošek, and M. A. Lewis. 2012. Aquaculture-induced changes to dynamics of a migratory host and specialist parasite: a case study of pink salmon and sea lice. Theoretical Ecology 5:231–252.

Bairlein, F. 2016. Migratory birds under threat. Science 354:547–548.

Baker, R. E., A. S. Mahmud, I. F. Miller, M. Rajeev, F. Rasambainarivo, B. L. Rice, S. Takahashi, A. J. Tatem, C. E. Wagner, L.-F. Wang, A. Wesolowski, and C. J. E. Metcalf. 2022. Infectious disease in an era of global change. Nature Reviews Microbiology 20:193–205.

Bastille-Rousseau, G., C. B. Yackulic, J. P. Gibbs, J. L. Frair, F. Cabrera, and S. Blake. 2019. Migration triggers in a large herbivore: Galápagos giant tortoises navigating resource gradients on volcanoes. Ecology 100:e02658.

Bauer, S., and B. J. Hoye. 2014. Migratory Animals Couple Biodiversity and Ecosystem Functioning Worldwide. Science 344:1242552.

Boano, G., I. Pellegrino, M. Ferri, M. Cucco, F. Minelli, and S. Åkesson. 2020. Climate anomalies affect annual survival rates of swifts wintering in sub-Saharan Africa. Ecology and Evolution 10:7916–7928.

Both, C., S. Bouwhuis, C. M. Lessells, and M. E. Visser. 2006. Climate change and population declines in a long-distance migratory bird. Nature.

Both, C., C. A. M. Van Turnhout, R. G. Bijlsma, H. Siepel, A. J. Van Strien, and R. P. B. Foppen. 2010. Avian population consequences of climate change are most severe for long-distance migrants in seasonal habitats. Proceedings of the Royal Society B: Biological Sciences 277.

Boucek, R. E., M. R. Heithaus, R. Santos, P. Stevens, and J. S. Rehage. 2017. Can animal habitat use patterns influence their vulnerability to extreme climate events? An estuarine sportfish case study. Global Change Biology 23:4045–4057.

Briedis, M., S. Hahn, and P. Adamík. 2017. Cold spell en route delays spring arrival and decreases apparent survival in a long-distance migratory songbird. BMC Ecology 17:1–8.

Brooks, D. R., and E. P. Hoberg. 2007. How will global climate change affect parasite-host assemblages? Trends in Parasitology 23:571–574.

Brower, L. P., O. R. Taylor, E. H. Williams, D. A. Slayback, R. R. Zubieta, and M. I. Ramírez. 2012. Decline of monarch butterflies overwintering in Mexico: is the migratory phenomenon at risk? Insect Conservation and Diversity 5:95–100.

Cameron, M. D., J. M. Eisaguirre, G. A. Breed, K. Joly, and K. Kielland. 2021. Mechanistic movement models identify continuously updated autumn migration cues in Arctic caribou. Movement Ecology 9:54.

Cohen, J. M., D. Fink, and B. Zuckerberg. 2021. Extreme winter weather disrupts bird occurrence and abundance patterns at geographic scales. Ecography 44:1143–1155.

Cohen, J. M., M. J. Lajeunesse, and J. R. Rohr. 2018. A global synthesis of animal phenological responses to climate change. Nature Climate Change 8:224–228.

Connare, B. M., and K. Islam. 2022. Failure to advance migratory phenology in response to climate change may pose a significant threat to a declining Nearctic-Neotropical songbird. International Journal of Biometeorology 66:803–815.

Crimmins, A. R. 2023. Fifth National Climate Assessment. Page Fifth National Climate Assessment. U.S. Global Change Research Program, Washington, DC.

Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife--threats to biodiversity and human health. Science (New York, N.Y.) 287:443–449.

Dingle, H., and V. Alistair Drake. 2007. What is migration? BioScience 57.

Faaborq, J., R. T. Holmes, A. D. Anders, K. L. Bildstein, K. M. Dugger, S. A. Gauthreaux, P. Heglund, K. A. Hobson, A. E. Jahn, D. H. Johnson, S. C. Latta, D. J. Levey, P. P. Marra, C. L. Merkord, N. O. L. Erica, S. I. Rothstein, T. W. Sherry, T. Scott Sillett, F. R. Thompson, and N. Warnock. 2010. Recent advances in understanding migration systems of New World land birds. Ecological Monographs 80.

Finch, T., S. J. Butler, A. M. A. Franco, and W. Cresswell. 2017. Low migratory connectivity is common in long-distance migrant birds. Journal of Animal Ecology 86:662–673.

Fricke, E. C., A. Ordonez, H. S. Rogers, and J.-C. Svenning. 2022. The effects of defaunation on plants' capacity to track climate change. Science 375:210–214.

Fryxell, J., J. Greever, and A. Sinclair. 1988. Why are Migratory Ungulates So Abundant? The American Naturalist 131:781.

Fuller, T., S. Bensch, I. Müller, J. Novembre, J. Pérez-Tris, R. E. Ricklefs, T. B. Smith, and J. Waldenström. 2012. The Ecology of Emerging Infectious Diseases in Migratory Birds: An Assessment of the Role of Climate Change and Priorities for Future Research. EcoHealth 9.

Gill, J. A. 2015. Encountering extreme weather during migration: individual strategies and their consequences. Journal of Animal Ecology 84:1141–1143.

Gilroy, J. J., J. A. Gill, S. H. M. Butchart, V. R. Jones, and A. M. A. Franco. 2016. Migratory diversity predicts population declines in birds. Ecology Letters 19:308–317.

Gordo, O. 2007. Why are bird migration dates shifting? A review of weather and climate effects on avian migratory phenology. Climate Research 35.

Grainger, T. N., A. Senthilnathan, P.-J. Ke, M. A. Barbour, N. T. Jones, J. P. DeLong, S. P. Otto, M. I. O'Connor, K. E. Coblentz, N. Goel, J. Sakarchi, M. C. Szojka, J. Levine, and R. M. Germain. 2021. An empiricist's guide to using ecological theory. The American Naturalist.

Gurarie, E., M. Hebblewhite, K. Joly, A. P. Kelly, J. Adamczewski, S. C. Davidson, T. Davison, A. Gunn, M. J. Suitor, W. F. Fagan, and N. Boelman. 2019. Tactical departures and strategic arrivals: Divergent effects of climate and weather on caribou spring migrations. Ecosphere.

Gurarie, E., S. Potluri, G. C. Cosner, R. S. Cantrell, and W. F. Fagan. 2021. Memories of Migrations Past: Sociality and Cognition in Dynamic, Seasonal Environments. Frontiers in Ecology and Evolution 9:742920.

Jones, T., and W. Cresswell. 2010. The phenology mismatch hypothesis: are declines of migrant birds linked to uneven global climate change? Journal of Animal Ecology 79:98–108.

Kauffman, M. J., E. O. Aikens, S. Esmaeili, P. Kaczensky, A. Middleton, K. L. Monteith, T. A. Morrison, T. Mueller, H. Sawyer, and J. R. Goheen. 2021. Causes, Consequences, and Conservation of Ungulate Migration. Annual Review of Ecology, Evolution, and Systematics 52.

Kölzsch, A., G. J. D. M. Müskens, H. Kruckenberg, P. Glazov, R. Weinzierl, B. A. Nolet, and M. Wikelski. 2016. Towards a new understanding of migration timing: slower spring than autumn migration in geese reflects different decision rules for stopover use and departure. Oikos.

Kreling, S. E. S., K. M. Gaynor, A. McInturff, K. L. Calhoun, and J. S. Brashares. 2021. Site fidelity and behavioral plasticity regulate an ungulate's response to extreme disturbance. Ecology and Evolution 11:15683–15694.

Laforge, M. P., M. Bonar, and E. Vander Wal. 2021. Tracking snowmelt to jump the green wave: phenological drivers of migration in a northern ungulate. Ecology 102.

Lisovski, S., K. Gosbell, C. Minton, and M. Klaassen. 2021. Migration strategy as an indicator of resilience to change in two shorebird species with contrasting population trajectories. Journal of Animal Ecology 90.

Loarie, S. R., P. B. Duffy, H. Hamilton, G. P. Asner, C. B. Field, and D. D. Ackerly. 2009. The velocity of climate change. Nature 462:1052–1055.

Mallory, C. D., S. N. Williamson, M. W. Campbell, and M. S. Boyce. 2020. Response of barren-ground caribou to advancing spring phenology. Oecologia.

Messinger, S. M., and A. Ostling. 2013. The influence of host demography, pathogen virulence, and relationships with pathogen virulence on the evolution of pathogen transmission in a spatial context. Evolutionary Ecology 27.

Møller, A. P., D. Rubolini, and E. Lehikoinen. 2008. Populations of migratory bird species that did not show a phenological response to climate change are declining. Proceedings of the National Academy of Sciences 105:16195–16200.

Nathan, R., W. M. Getz, E. Revilla, M. Holyoak, R. Kadmon, D. Saltz, and P. E. Smouse. 2008. A movement ecology paradigm for unifying organismal movement research. Proceedings of the National Academy of Sciences 105:19052–19059.

Nilsson, A. L. K., Å. Lindström, N. Jonzén, S. G. Nilsson, and L. Karlsson. 2006. The effect of climate change on partial migration - the blue tit paradox: Climate change and Blue Tit partial migration. Global Change Biology 12:2014–2022.

Otto, S. P., and A. Rosales. 2020. Theory in Service of Narratives in Evolution and Ecology. The American Naturalist 195:290–299.

Overton, C. T., A. A. Lorenz, E. P. James, R. Ahmadov, J. M. Eadie, F. Mcduie, M. J. Petrie, C. A. Nicolai, M. L. Weaver, D. A. Skalos, S. M. Skalos, A. L. Mott, D. A. Mackell, A. Kennedy, E. L. Matchett, and M. L. Casazza. 2022. Megafires and thick smoke portend big problems for migratory birds. Ecology 103.

Parmesan, C., and G. Yohe. 2003. A globally coherent fingerprint of climate change impacts across natural systems. Nature 421:37–42.

Plowright, R. K., J. K. Reaser, H. Locke, S. J. Woodley, J. A. Patz, D. J. Becker, G. Oppler, P. J. Hudson, and G. M. Tabor. 2021. Land use-induced spillover: a call to action to safeguard environmental, animal, and human health. The Lancet Planetary Health 5:e237–e245.

Pörtner, H.-O., R. J. Scholes, A. Arneth, D. K. A. Barnes, M. T. Burrows, S. E. Diamond, C. M. Duarte, W. Kiessling, P. Leadley, S. Managi, P. McElwee, G. Midgley, H. T. Ngo, D. Obura, U. Pascual, M. Sankaran, Y. J. Shin, and A. L. Val. 2023. Overcoming the coupled climate and biodiversity crises and their societal impacts. Science 380:eabl4881.

Post, E., and M. C. Forchhammer. 2008. Climate change reduces reproductive success of an Arctic herbivore through trophic mismatch. Philosophical Transactions of the Royal Society B: Biological Sciences 363.

Poulin, R., and D. de Angeli Dutra. 2021. Animal migrations and parasitism: reciprocal effects within a unified framework. Biological Reviews.

Power, A. G., and C. E. Mitchell. 2004. Pathogen Spillover in Disease Epidemics. The American Naturalist 164:S79–S89.

Rayl, N. D., J. A. Merkle, K. M. Proffitt, E. S. Almberg, J. D. Jones, J. A. Gude, and P. C. Cross. 2021. Elk migration influences the risk of disease spillover in the Greater Yellowstone Ecosystem. Journal of Animal Ecology 90:1264–1275.

Robinson, R. A., H. Q. P. Crick, J. A. Learmonth, I. M. D. Maclean, C. D. Thomas, F. Bairlein, M. C. Forchhammer, C. M. Francis, J. A. Gill, B. J. Godley, J. Harwood, G. C. Hays, B. Huntley, A. M. Hutson, G. J. Pierce, M. M. Rehfisch, D. W. Sims,

M. Begoña Santos, T. H. Sparks, D. A. Stroud, and M. E. Visser. 2009. Travelling through a warming world: Climate change and migratory species. Endangered Species Research.

Roode, J. C. de, L. R. Gold, and S. Altizer. 2006. Virulence determinants in a natural butterfly-parasite system. Parasitology 134:657–668.

Rubenstein, D. I., and M. A. Hack. 2013. Migration. Pages 309–320 Encyclopedia of Biodiversity. Elsevier.

Rushing, C. S., J. A. Royle, D. J. Ziolkowski, and K. L. Pardieck. 2020. Migratory behavior and winter geography drive differential range shifts of eastern birds in response to recent climate change. Proceedings of the National Academy of Sciences 117:12897–12903.

Satterfield, D. A., J. C. Maerz, M. D. Hunter, D. T. T. Flockhart, K. A. Hobson, D. R. Norris, H. Streit, J. C. de Roode, and S. Altizer. 2018. Migratory monarchs that encounter resident monarchs show life-history differences and higher rates of parasite infection. Ecology Letters 21.

Sawyer, H., J. A. Merkle, A. D. Middleton, S. P. H. Dwinnell, and K. L. Monteith. 2019. Migratory plasticity is not ubiquitous among large herbivores. Journal of Animal Ecology 88:450–460.

Schmitz, O. J., and M. Sylvén. 2023. Animating the Carbon Cycle: How Wildlife Conservation Can Be a Key to Mitigate Climate Change. Environment: Science and Policy for Sustainable Development 65:5–17.

Seebacher, F., and E. Post. 2015. Climate change impacts on animal migration. Climate Change Responses.

Servedio, M. R., Y. Brandvain, S. Dhole, C. L. Fitzpatrick, E. E. Goldberg, C. A. Stern, J. V. Cleve, and D. J. Yeh. 2014. Not Just a Theory—The Utility of Mathematical Models in Evolutionary Biology. PLOS Biology 12:e1002017.

Shaw, A. K. 2016. Drivers of animal migration and implications in changing environments. Evolutionary Ecology.

Shaw, A. K., A. T. Bisesi, C. Wojan, D. Kim, M. Torstenson, null Naven Narayanan, P. Lutz, R. Ales, and C. Shao. 2024. Six personas to adopt when framing theoretical

research questions in biology. Proceedings of the Royal Society B: Biological Sciences 291:20240803.

Shaw, A. K., and I. D. Couzin. 2013. Migration or residency? the evolution of movement behavior and information usage in seasonal environments. American Naturalist 181.

Somveille, M., A. S. L. Rodrigues, and A. Manica. 2018. Energy efficiency drives the global seasonal distribution of birds. Nature Ecology & Evolution 2:962–969.

Soriano-Redondo, A., J. S. Gutiérrez, D. Hodgson, and S. Bearhop. 2020. Migrant birds and mammals live faster than residents. Nature Communications 11.

Stratmann, T. S. M., M. Forrest, W. Traylor, N. Dejid, K. A. Olson, T. Mueller, and T. Hickler. 2023. Movement drives population dynamics of one of the most mobile ungulates on Earth: Insights from a mechanistic model. Ecology 104:e4071.

Sutherland, W. J. 1998. Evidence for Flexibility and Constraint in Migration Systems. Journal of Avian Biology 29.

Ugelvik, M. S., A. Skorping, O. Moberg, and A. Mennerat. 2017. Evolution of virulence under intensive farming: salmon lice increase skin lesions and reduce host growth in salmon farms. Journal of Evolutionary Biology 30:1136–1142.

Wilcove, D. S. 2008. Animal migration an endangered phenomenon? 24:71–78.

Wilcove, D. S., and M. Wikelski. 2008. Going, going, gone: Is animal migration disappearing? PLoS Biology 6.

Chapter 1

Strength of seasonality and type of migratory cue determine the fitness consequences of changing phenology for migratory animals

Abstract

Phenological mismatch has been highlighted as a reason why climate change is causing declines of migratory populations. The likelihood of declines due to phenological mismatch might depend on what cues trigger migration. Migrants that use environmental cues (e.g. temperature, resource availability) to trigger migration are often considered to be less vulnerable than migrants that use temporal information (e.g. photoperiod). We developed a proof-of-concept model that demonstrates that which cue type performs better in the context of phenological change can depend on differences in seasonal amplitude between the habitats used by a migrant. Environmental cues perform better than temporal cues when the habitat that undergoes a phenological change has a larger seasonal amplitude. This result aligns with observations that populations of short-distance migrants that use environmental cues are less likely to decline as a consequence of phenological mismatch than long-distance avian migrants that use temporal cues. Temporal cues perform better than environmental cues when the habitat that undergoes a phenological change has a smaller seasonal amplitude. This result may correspond to empirical scenarios where the more seasonal habitat is associated with variation in precipitation patterns or human actions that are not changing in phenology. In addition to these results, we offer distinctions that will help clarify future work on phenological mismatch. First, we highlight the difference between the cue *accuracy* (difference

between the timing of migration using the cue and optimal migration timing) and the cue *efficacy* (the difference between the fitness using the cue and the fitness using optimal migration). Second, we recommend considering both how the benefits available from migrating and the benefits that are captured by migrants change with phenological change.

Introduction

Animals that migrate seasonally are often considered to be especially vulnerable to climate change (Wilcove 2008) in part because they are especially susceptible to phenological mismatch (Both et al. 2006, Post and Forchhammer 2008, Bairlein 2016, Cohen et al. 2018, Connare and Islam 2022). Migration between locations where there is seasonality in the favorability of conditions can allow animals to exploit seasonally available resources while avoiding harsh conditions (Dingle and Drake 2007, Armstrong et al. 2016, Fokkema et al. 2020). To benefit from migration, migrants use information available in part of their range (proximate cues) to move to another part of their range at a time favored by ecological factors that affect fitness (ultimate drivers) (Both et al. 2006, Gordo 2007, Post and Forchhammer 2008, Møller et al. 2008, Connare and Islam 2022). Climate change may affect the phenology of the proximate cues migrants use to trigger migration differently than it affects the phenology of the ultimate drivers of migration, leading to phenological mismatch (Shaw 2016).

The empirical evidence for phenological mismatch induced declines of migratory populations is mixed. Some migratory populations already show evidence of declines due to phenological mismatch (e.g. pied flycatchers *Ficedula hypoleuca,* Both et al., 2006; East Atlantic light-bellied brent geese *Branta bernicla hrota,* Clausen & Clausen, 2013; freshwater fish, Kuczynski et al., 2017; European birds, Møller et al., 2008; caribou *Rangifer tarandus,* Post and Forchhammer, 2008). The sizes of other migratory populations appear to be unaffected by phenological mismatch (e.g. Pacific Arctic beluga whales *Delphinapterus leucas,* Hauser et al., 2017; caribou *Rangifer tarandus,* Mallory et al., 2020; European birds, Møller et al., 2008; Vaux's swifts *Chaetura vauxi,* Prytula et al., 2022; bowhead whales *Balaena mysticetus,* Tsujii et al., 2021). Populations that are declining due to phenological mismatch and those that are not may differ both in how the phenology of their ultimate drivers of migration are changing and how their migratory strategies allow them to respond to those changes (Both et al. 2006, Møller et al. 2008). Phenological change in the drivers of migration is one of many environmental factors that may affect the severity of phenological mismatch that migratory populations experience.

Logically, larger magnitude phenological changes in the drivers of migration can lead to more severe phenological mismatch if the phenology of migration fails to adjust. It is also relevant that the effects of climate change are unevenly distributed around the world such that different regions vary in climate change velocity (Garcia et al. 2014, Wang et al. 2021). One consequence of this uneven distribution of climate change is that the correlation between the timing of events at different habitats used by a migrant may change. For example, advancing spring phenology at a migrant's breeding habitat may not be matched by the same magnitude of phenology shift at a migrant's wintering habitat (Taylor et al. 2016). The degree of seasonality in the habitats used by a migratory population may also affect the consequences of phenological mismatch. The degree of seasonality is based in part on seasonal amplitude (i.e. the magnitude of seasonal change) (Lisovski et al. 2017). Some habitats have larger seasonal amplitude than others and many habitats used by migrants are characterized by large peaks in resource availability (Alerstam et al. 2003, Durant et al. 2007, Both et al. 2010, Bauer and Hoye 2014, Saalfeld and Lanctot 2017). Larger seasonal amplitude can lead to higher consequences of missing out on peaks in habitat quality or failing to avoid periods of poor habitat quality.

One aspect of migration strategy that is important for determining the probability and severity of the population consequences of phenological mismatch is the proximate cue that triggers migration (Wilcove and Wikelski 2008). Migrants use different types of cues to trigger migration, including environmental cues and temporal cues. We take environmental cues to be environmental factors relevant to fitness, like resource availability (e.g. green-up or snow/ice melt: Kauffman et al., 2021; Kölzsch et al., 2016; Laforge et al., 2021) or temperature. These proximate cues may fail to continue to match the ultimate drivers of migration because of climate change happening at different rates in different places (Jones and Cresswell 2010, Visser et al. 2012, Charmantier and Gienapp 2014, Lameris et al. 2017). We take temporal cues to be factors that directly indicate the date of the year, commonly photoperiod or circannual rhythms (Gwinner 1996, Post and Forchhammer 2008, Chmura et al. 2020, Fudickar et al. 2021, Tillotson et al. 2021, Connare and Islam 2022). The phenology of temporal cues do not change, so

they will fail to match the drivers of migration if the phenology of the ultimate drivers of migration shift. Although selection across generations might change which cues are used over time, the initial impact of changes in phenology will be determined by the plasticity associated with the cues that are currently used by individuals in a population (McNamara et al. 2011, Charmantier and Gienapp 2014, Gill et al. 2019).

It has been asserted that migrants using temporal cues are more vulnerable to phenological mismatch than those using environmental cues because temporal cues offer less adaptive plasticity than environmental cues (Visser et al. 2012, Donohue and Piiroinen 2015, Laforge et al. 2021, Connare and Islam 2022). However, this claim often conflates cue type with migration distance since short-distance migrants often use environmental cues and migrants that are identified as using temporal cues often migrate long distances (Gwinner 1996, Chmura et al. 2019, 2020, Connare and Islam 2022). Few empirical studies directly compare the responses of populations with different migration strategies to the same cues (Chmura et al. 2020) and theoretical work may only consider the constraints imposed by temporal cues (Donohue and Piiroinen 2015). The environmental conditions at the departure and destination habitats of longer distance migrants are often less similar than those of shorter distance migrants (Rubolini et al. 2010, Clausen and Clausen 2013, Saalfeld and Lanctot 2017). This means that there may also be larger differences in the amplitude of seasonality and the magnitude of phenological change between the habitats used by a migrant, which may mediate the fitness effects of phenological change. It is not clear that long-distance migrants would perform better in the context of climate-altered conditions if they used environmental cues that are available to them. It would be next to impossible to explore this counterfactual empirically, but by using mathematical models we can isolate the effects of cue type. Our understanding of the conditions under which use of temporal cues might make migrants more vulnerable to phenological mismatch is currently limited and could be improved through mathematical modelling.

Here, we developed a calculus-based proof-of-concept model (Servedio et al. 2014) that provides an understanding of how changing phenology affects the fitness benefits of migration for migrants using different cue types. Specifically, we investigated

how the cues that are used to trigger migration interact with environmental factors (seasonal amplitude, difference in habitat quality peak timing between the habitats used, and magnitude of phenological shift) to determine how climate change alters the benefits of migration. Many mathematical models have been developed to consider phenological mismatch (McNamara et al. 2011, Johansson and Jonzén 2012, Donohue and Piiroinen 2015) and several studies have asserted that migratory cue type plays a role in determining the strength of phenological mismatch experienced by migratory populations (Durant et al. 2005, Charmantier and Gienapp 2014, Donohue and Piiroinen 2015). To our knowledge, however, no models have made direct comparisons between the phenological mismatch that would be experienced using different types of cues when controlling for the system of habitats used by a migratory population as we do here. The central comparison in our model is between the benefits of migration following phenological change for migratory individuals using either environmental or temporal cues. To support this comparison, we illustrate how the cumulative fitness of migrants compares with that of residents in order to demonstrate the benefit of migration in our model. We also compare the benefits of migration before and after phenological change to understand the factors besides cue type that determine the effects of phenological change.

Methods

Overview

In this model, migrants move between two habitats in which the habitat quality (i.e. environmental factors relevant to fitness) varies as a function of time. We determine the optimal time at which migrants using temporal cues would migrate and similarly the optimal level of habitat quality in the departure habitat at which migrants using environmental cues would migrate. We then assess the cumulative habitat quality experienced over the course of the year by an individual migrating at times determined by either environmental or temporal cues. This cumulative habitat quality is taken to be the

fitness of the individual, as defined by a cumulative fitness function (Shaw and Couzin 2013). Then, the function that describes quality of one of the habitats is shifted in time (e.g., earlier spring or later spring) to represent phenological change. We assess the cumulative habitat quality experienced in the new system of habitats by a migrant using either the temporal or environmental cues that were optimal before the temporal shift. Calculations were performed either by hand or using Mathematica. The variables, parameters, and functions used in the model are summarized in Table 1.1.

Habitat System

We begin by defining the two habitats a migrant moves between (i.e. the habitat system) by the habitat quality functions

$$H_1(t) = a * \sin(t) \tag{1.1a}$$

for habitat 1 and

$$H_2(t) = \sin(t - b) \tag{1.1b}$$

for habitat 2. We use sine curves to represent cyclic variation in the environmental factors that are relevant to fitness (e.g. the ultimate drivers of migration) for tractability (Figure 1.1a). Representing cyclic variation in habitat quality as the driver of migration is best suited for species whose migrations are driven by variation in the environment as opposed to species whose migrations are driven by differences in habitat requirements for different life stages (Shaw 2016). In these functions a describes the amplitude of habitat 1 relative to habitat 2 (the habitats differ in amplitude if $a \neq 1$), and b describes the horizontal displacement (i.e. temporal shift) of habitat 2 relative to habitat 1. We take the average habitat quality at both habitats to be zero and do not explore differences in average habitat quality between the two habitats (i.e. vertical displacement) in this study.

Optimal Migration

We make the logistical assumption that migration occurs instantaneously. In this case, optimal migration allows a migrant to be in the habitat with the highest habitat quality at

every point in time (i.e. in habitat 1 whenever $H_1(t) > H_2(t)$ and in habitat 2 when $H_1(t) < H_2(t)$). Thus, migration should occur whenever one habitat's quality function crosses over the other (t such that $H_1(t) = H_2(t)$) (Figure 2.1b). This optimal migration condition is met at some time t^* and some habitat quality h^*. Thus, we could consider the migrant to be perceiving and using information about time (temporal cues) or habitat quality in the habitat they are departing from (environmental cues) to migrate optimally. We consider when optimal migration occurs over the course of one annual cycle, represented by the time interval $[0,2\pi]$. The migrant starts in habitat 1 and then migrates from habitat 1 to habitat 2 at (t_1^*, h_1^*) which is the first point where $H_1(t) = H_2(t)$ and then migrates from habitat 2 to habitat 1 at $(t_2^*\, h_2^*)$, which is the second point where $H_1(t) = H_2(t)$. We solve $H_1(\tau_1^*) = h_1^*$ and $H_2(\tau_2^*) = h_2^*$ for τ_1^*, τ_2^* to identify when optimal migration occurs using environmental cues. Note that the times at which optimal migrations using temporal and environmental cues occur are the same, that is $t_1^* = \tau_1^*$ and $t_2^* = \tau_2^*$.

We define the cumulative habitat quality experienced by a migrant in a year as the integral of the habitat quality function of whichever habitat the migrant is in over the time interval $[0,2\pi]$ (Figure 1.1b). We interpret this as a metric of fitness by assuming that the survival and fecundity of an individual can be represented by a cumulative fitness function (Shaw and Couzin 2013). The habitat quality experienced by a migrant over the course of a year (hereafter 'fitness') is given as a function

$$Q(H_1, H_2, t_1, t_2) = \int_0^{t_1} H_1(t)dt + \int_{t_1}^{t_2} H_2(t)dt + \int_{t_2}^{2\pi} H_1(t)dt \qquad (1.1c)$$

of the two habitat quality functions (H_1, H_2) and the times at which migration occurs (t_1, t_2). In the original habitat system, we define the fitness of migrants using temporal cues as Q_t^* and the fitness of migrants using environmental cues as Q_e^*. We note that because the integral of a sine function with an average habitat quality value of zero over the time interval $[0,2\pi]$ is equal to zero, Q can also be understood as the net fitness benefit of migration when compared with residency at one habitat year round (hereafter 'benefit of migration') or as the fitness cost of migration that could be tolerated before migration becomes net costly.

Environmental change and impact on migration

To represent a change in the timing of habitat quality, we translate $H_2(t)$ horizontally by a value c (Figure 1.1 c-d). The new habitat quality function for habitat 2 is given by

$$H_{2,N}(t) = \sin(t - b - c) \quad (1.1d)$$

The pair of habitat functions, $H_1(t)$ and $H_{2,N}(t)$ is the new habitat system.

We calculate the fitness of migrants in the new habitat system using either temporal cues or environmental cues that were optimal in the original system. Cues that were optimal in the previous habitat regime but may not be optimal in the new habitat regime are denoted as $\hat{t_1}, \hat{t_2}, \hat{h_1}, \hat{h_2}$. Temporal cues occur at the same time before and after the change in the habitat quality function (i.e. $\hat{t_1} = t_1^*$ and $\hat{t_2} = t_2^*$) (Figure 1.1c). The fitness of migrants using temporal cues is $\hat{Q_t} = Q(H_1, H_{2,N}, \hat{t_1}, \hat{t_2})$. In contrast to when the cues were timed optimally, temporal and environmental cues no longer occur at the same time. To calculate the fitness of migrants using environmental cues, we find what times an individual would migrate using old environmental cues by solving for $\hat{\tau_1}$ in $H_1(\hat{\tau_1}) = h_1^*$ and for $\hat{\tau_2}$ in $H_{2,N}(\hat{\tau_2}) = h_2^*$ (Figure 1.1d). The new fitness of migrants using environmental cues is $\hat{Q_e} = Q(H_1, H_{2,N}, \hat{\tau_1}, \hat{\tau_2})$.

We can compare these timing and fitness values with scenario of optimal migration in the new system. Optimal migration in the transformed set of functions occurs at (t_1^{**}, h_1^{**}) and (t_2^{**}, h_2^{**}) such that $H_1(t^{**}) = H_{2,N}(t^{**})$. We represent the times at which optimal migration using environmental cues occurs as τ_1^{**} and τ_2^{**} which are equal to t_1^{**} and t_2^{**}respectively. The fitness that would be experienced using optimal cues is given by $Q_t^{**} = Q(H_1, H_{2,N}, t_1^{**}, t_2^{**})$ for temporal cues and $Q_e^{**} = Q(H_1, H_{2,N}, \tau_1^{**}, \tau_2^{**})$ for environmental cues.

We consider two metrics of cue performance: the *accuracy* of the cue and the *efficacy* of the cue. The *accuracy* of the cue is the difference between the timing of migration using the cue and optimal migration timing (for temporal cues: $|\hat{t_1} - t_1^{**}| + |\hat{t_2} - t_2^{**}|$, for environmental cues: $|\hat{\tau_1} - \tau_1^{**}| + |\hat{\tau_2} - \tau_2^{**}|$). The *efficacy* of the cue is the

difference between the fitness using the cue and the fitness using optimal migration (for temporal cues: $\left|Q_t^{\wedge} - Q_t^{**}\right|$) , for environmental cues: $\left|Q_e^{\wedge} - Q_e^{**}\right|$).

Results

The central question of our model is: under what conditions is migration using temporal cues more effective than migration using environmental cues following environmental change? To build towards the results of the comparison between migrants using different cue types we begin by presenting results comparing migration with residency in order to illustrate how the benefits of migration work in our model. We also compare the benefits of migration before and after phenological change in order to illustrate how phenological mismatch works in our model. For these two preliminary comparisons, we present limited results in the main text and a more full exploration in the supplement.

1) Benefits of migration: Under what conditions is migration better than residency?

In general, the benefits of migration depend on two factors. The first of these is the habitat system, which determines how much benefit is *available* (the benefit that is captured by optimal migration). The second is the timing of migration, which determines how much of the available benefit is *captured* (that is, how effective the cues are).

The available benefit of migration in a habitat system when the two habitat quality functions have equal amplitudes ($a = 1$) is given by

$$Q_t^* = Q_e^* = 4\sin\left(\frac{b}{2}\right) \qquad (1.2a).$$

This takes a maximum value of 4 when $b = \pi \ mod \ 2\pi$, i.e. when the highest habitat quality at one habitat occurs at the same time as the lowest habitat quality at the other habitat. The available benefit has a minimum value of zero when $b = 0 \ mod \ 2\pi$, i.e. when the highest habitat quality values occur at the same time in both habitats. When the two habitat quality functions have different amplitudes, fitness is largest when $b = \pi \ mod \ 2\pi$ and fitness increases as a increases (Appendix A, Figure A1). Another

qualitative difference in the different amplitudes case is that when the highest habitat quality values occur at the same time ($b = 0\ mod\ 2\pi$), migration is beneficial (Appendix A, Figure A1) because the higher amplitude habitat has better habitat quality when the habitat quality value is positive and worse habitat quality when the habitat quality value is negative.

Following phenological change (i.e. a horizontal shift in habitat 2's quality), migration using previously optimal cues may take an individual into a habitat with worse habitat quality than if it had stayed put. When $a = 1$, the benefit of migration following phenological change is

$$Q_t^{\wedge} = Q_e^{\wedge} = 2\sin\left(\frac{b}{2}\right) + \sin\left(\frac{b}{2} + c\right) \quad (1.2b).$$

(Figure 1.2). When the habitats have different amplitudes ($a \neq 1$), one qualitative difference is that $Q_t^{\wedge}$ is not equal to $Q_e^{\wedge}$ (Supporting Information). We can understand the benefit of migration following phenological change ($Q^{\wedge}$) by understanding how the benefits available from migration and the benefits of migration that are captured vary as b and c vary.

The available benefits of migration vary following Eq. 1.2a. Following phenological change, the benefits of migration that are available are maximized when $b + c = \pi\ mod\ 2\pi$, ($c = \pi - b$ and $c = 3\pi - b$ in Figure 1.2) and minimized at $Q^{\wedge} = 0$ when $b + c = 0\ mod\ 2\pi$, ($c = -b$ and $c = 4\pi - b$ in Figure 1.2). When the benefits available from migration are small, it also means that it does not matter if migration is mistimed (i.e. the accuracy of the cues has little impact on the efficacy of the cues).

Understanding the efficacy of the cues requires considering the balance between time intervals when migrating is worse than being a resident in habitat 1 with time intervals when migrating is better than being a resident in habitat 1. When cues are optimally timed ($c = 0\ mod\ 2\pi$), all of the available benefits are captured and there are no costs from being in the worse habitat. The benefits and costs are equal to each other when $c = \pi\ mod\ 2\pi$ ($Q^{\wedge}$ is zero on the line $c = \pi$ in Figure 1.2). When $Q^{\wedge}$ is negative, it means that the costs of being in habitat 2 when the habitat quality in habitat 1 is better outweigh the benefits of being in habitat 2 when the habitat quality in habitat 1 is worse.

2) Phenological mismatch: Under what conditions do migrants perform worse following phenological change than before phenological change?

The change in the benefits captured by a migrant following phenological change depends on how the benefits that are available change and how the efficacy of the migratory cues changes. When both habitats have the same amplitude ($a = 1$), the difference between fitness before ($Q^* = Q_t^* = Q_e^*$) and after ($Q^{\wedge} = Q_t^{\wedge} = Q_e^{\wedge}$) phenological change is given by

$$Q^{\wedge} - Q^* = 4\cos\left(\frac{b+c}{2}\right)\sin\left(\frac{c}{2}\right) \quad (1.3a)$$

(Figure 1.3). When $a \neq 1$, this impact of phenological change depends on cue type (i.e. $Q_t^{\wedge} - Q_t^*$ is not the same as $Q_e^{\wedge} - Q_e^*$) (Supporting Information). In either case, $Q^{\wedge} - Q^*$ can take a positive or negative value, i.e., the experienced benefit of migration following a change in phenology can be higher or lower than it was originally.

Changing phenology can increase or decrease the available benefit of migration. As noted above, Equation 1.2a is maximized when $b = \pi$. If $b + c$ is closer to π than b is, the available migration benefit increases after the shift. If $b + c$ is further from π than b is, the available migration benefit decreases. Sub-optimal timing of migration following the horizontal shift means that the migrant captures a smaller proportion of the available benefit than when migration was timed optimally. An increase in the benefit of migration following phenological change ($Q^{\wedge} - Q^* > 0$) means that the fraction of the new available benefit of migration that is captured after the phenological shift is greater than the total available benefit of migration before the phenological shift.

3) Cue type: Under what conditions do migrants using environmental cues perform better than migrants using temporal cues?

When the amplitudes of the two habitat functions are the same ($a = 1$, as captured in equation 1.2b), both cue types are equally effective following the horizontal shift to habitat 2's habitat quality function but the timing of migration is different for temporal and environmental cues. Recall that before horizontal translation, the values of the temporal cues are given by equations 2c and 2d. Following horizontal translation of $H_2(t)$, the time at which migration using old temporal cues occurs remains the same ($t_1^* = \hat{t_1}$ and $t_2^* = \hat{t_2}$) as does the migration from habitat 1 to habitat 2 using old environmental cues ($\tau_1^* = \hat{\tau_1}$). The migration from habitat 2 to habitat 1 using environmental cues now occurs at time

$$\hat{\tau_2} = \frac{1}{2}(b + 2c + 3\pi) \tag{1.4a}$$

These times contrast with migration times that are optimal in the new habitat system,

$$t_1^{**} = \tau_1^{**} = \frac{1}{2}(b + c + \pi) \tag{1.4b}$$

and

$$t_2^{**} = \tau_2^{**} = \frac{1}{2}(b + c + 3\pi) \tag{1.4c}$$

That means that all of the migrations are now occurring c units of time different than would be optimal (the cues are equally accurate), but the direction in which the temporal and environmental cues are wrong is different when migrating away from the habitat (habitat 2) that underwent a phenological shift. The consequences of migrating c units of time too early and c units of time too late are identical because of the symmetries associated with sine curves (e.g. increase towards a peak occurs at the same rate as decrease away from a peak), which means the cues are equally effective (see Figure 1.4 for a graphical example).

When the amplitudes of the habitat quality functions are different ($a \neq 1$), either environmental or temporal cues for the migration away from habitat 2 may be more

effective following a horizontal shift in habitat 2's quality. The difference between the fitness of migrants using temporal cues and migrating using environmental cues is

$$Q_t^{\wedge} - Q_e^{\wedge} = \left(\frac{a^2}{\sqrt{1+a^2-2\,a\cos(b)}} - \frac{a\cos(b)}{\sqrt{1+a^2-2\,a\cos(b)}} - 2\cos\left(\mathrm{b} + \mathrm{c} + \mathrm{ArcCos}\left(\frac{\mathrm{a}-\cos(b)}{\sqrt{1+a^2-2\,a\cos(b)}}\right)\right) - 2\cos\left(\frac{c}{2}\right)\cos\left(b + \frac{c}{2} - \mathrm{ArcCos}\left(\frac{-\mathrm{a}+\cos(b)}{\sqrt{1+a^2-2\,a\cos(b)}}\right)\right) + a\cos\left(c + \mathrm{ArcCos}\left(\frac{-\mathrm{a}+\cos(b)}{\sqrt{1+a^2-2\,a\cos(b)}}\right)\right)\right) \quad (1.4d)$$

(Figure 1.5).

For the migration away from the shifting habitat, temporal cues are more effective when the habitat that is not shifting has the larger amplitude ($a > 1$) and environmental cues are more effective when the habitat that is not shifting has the smaller amplitude ($a < 1$), regardless of the direction of the horizontal shift (Figure 1.5). The difference in efficacy between the cue types is larger the more different the amplitudes (the farther a is from 1) and the larger the phenological shift (the farther c is from 0) (Figure 1.5). We understand why this is the case by observing differences in the accuracy of the cues and how they relate to efficacy (see Figure 1.6 for graphical examples).

If the timing of habitat 2 advances (moves left; Figure 1.6a-d), migration from habitat 1 to habitat 2 occurs too late for both cue types. Migration from habitat 2 to habitat 1 occurs too late for temporal cue-triggered migration (Figure 1.6a,c) and too early for environmental cue-triggered migrations (Figure 1.6b,d). If habitat 2 also has a larger amplitude, the negative effects on efficacy from migrating too late from habitat 2 to habitat 1 are larger than the negative effects on efficacy from migrating too early. Thus, environmental cues lead to a larger net benefit of migration (Figure 1.6a vs. Figure 1.6b). On the other hand, if habitat 1 has a larger amplitude the negative effects on efficacy from migrating too early from habitat 2 to habitat 1 are larger than those from migrating too late. Thus, temporal cues lead to a larger net benefit of migration (Figure 1.6c vs. Figure 1.6d).

If habitat 2 falls back in time (moves right; Figure 1.6e-h), migration from habitat 1 to habitat 2 occurs too early for both cue types. Migration from habitat 2 to habitat 1 occurs too early for temporal cue-triggered migration (Figure 1.6e,g) and too late for

environmental cue-triggered migration (Figure 1.6f,h). If habitat 2 has a larger amplitude, the negative effects on efficacy from migrating too early from habitat 2 to habitat 1 are larger than those from migrating too late. Thus, environmental cues lead to a larger net benefit of migration (Figure 1.6e vs. Figure 1.6f). On the other hand, if habitat 1 has a larger amplitude the negative effects on efficacy from migrating too late from habitat 2 to habitat 1 are larger than those from migrating too early. Thus, temporal cues lead to a larger net benefit of migration (Figure 1.6g vs. Figure 1.6h).

Given the habitat quality functions we use here, the differences in accuracy between the cue types are entirely due to differences in efficacy. Migration that occurs d units of time earlier than optimal yields the same fitness as migration that occurs d units of time later than optimal, i.e.

$$Q\left(H_1, H_{2,N}, t_1^{**} - d, t_2^{**}\right) - Q\left(H_1, H_{2,N}, t_1^{**} + d, t_2^{**}\right) = 0 \quad (1.4e)$$

Discussion

Phenological mismatch may negatively affect migratory populations. However, our understanding of the conditions under which migratory populations are especially vulnerable to phenological mismatch is limited. The type of cues that are used to trigger migration has been proposed as a factor in whether the proximate cues will continue to be aligned with the ultimate drivers of migration under climate change (Wilcove and Wikelski 2008, Visser et al. 2012, Laforge et al. 2021, Connare and Islam 2022). However, we lack theoretical predictions about when specific cue types will be advantageous under which conditions. In this paper, we have developed a model to address the question: How does the change in the benefit of migration following phenological change depend on which cue type is used?

Our model answers our central question by revealing that differences in strength of seasonality between the habitats used by a migrant can lead to either environmental or temporal cues being more effective following phenological change. Many migrants, especially those that seek refuge from seasonally poor habitat, move between habitats that differ in their seasonal amplitude (e.g. birds that migrate between circumpolar regions with large seasonal variation in habitat quality and tropical regions with more

limited seasonal variation in habitat quality) (Shaw 2016). In our model, we find that if the habitat that is undergoing a phenological shift has a stronger seasonality, environmental cues are more effective, regardless whether the shift moves habitat quality earlier or later in time. If the habitat that is undergoing a phenological shift has a weaker seasonality, temporal cues are more effective, regardless of the direction of the shift. Although each cue type is more effective for an equal amount of the parameter space explored in our model, it is more straightforward to identify empirical scenarios in which our model would predict that environmental cues are more effective.

Our model predicts that environmental cues will be more effective for latitudinal migrants for whom habitat quality is roughly associated with temperature. We observe that spring timing is advancing faster in circumpolar regions, where seasonal amplitude in habitat quality is larger, than it is near the equator, where seasonal amplitude is smaller (Lameris et al. 2017, Lisovski et al. 2017). Our model predicts that environmental cues would be more effective in a case like this. This aligns with observations that long-distance migratory birds that use temporal cues face the most negative consequences of phenological mismatch (Visser et al. 2012, Connare and Islam 2022) and offers an explanation for this phenomenon. Relaxing our assumption that only one habitat undergoes phenological change expands the set of conditions under which environmental cues are more effective, as long as the phenological changes move in the same direction. As an extreme case, when both habitats undergo phenological changes in the same direction with identical magnitudes, environmental cues will continue to be perfectly accurate and maximally effective while temporal cues will be inaccurate in both directions and be less effective. Thus, in many cases, our modelling framework supports the assertion that migration using environmental cues is more effective in the context of phenological change.

Identifying cases in which our model predicts that temporal cues will be more effective is less straightforward. For temporal cues to be more effective than environmental cues following phenological change in our model, the less seasonal habitat needs to undergo larger phenological changes. This could occur when anthropogenic factors like hunting seasons play a role in the seasonality of habitat quality (e.g. in red

deer, Debeffe et al. 2019). Habitats in which seasonal hunting occurs may have larger seasonal amplitudes in habitat quality and the phenology of a hunting season may not change if it is determined by government regulations. Temporal cues may also be more effective than environmental cues for some altitudinal migrants and migrations driven by patterns of rainfall or extreme weather events. For example, White-ruffed Manakins (*Corapipo altera*) migrate downhill to avoid seasonal storm risk at higher altitudes (Boyle et al. 2010). In this case, habitat quality may have a larger seasonal amplitude at high altitudes. There is also some indication that climate change velocity is slower in montane environments (Loarie et al. 2009). This scenario roughly aligns with the conditions our model predicts would lead to environmental cues being more effective, but more information is needed to use our model to predict which cue type will be most effective in migrations driven by rainfall. Although many areas near the equator clearly show high degrees of seasonality in precipitation (Lisovski et al. 2017) that are relevant for many migratory animals, and patterns of climate velocity for rainfall are similar to those for temperature (Loarie et al. 2009), changes in rainfall are less predictable than changes in temperature (Loarie et al. 2009). The diverse nature of changes in precipitation around the world means that rainfall-driven migrations may be useful for empirical tests of our model's prediction that differences in the efficacy of temporal and environmental cues occur because of differences in the strength of seasonality and degree of phenological change between habitats.

We have also developed a framework that future studies of phenological mismatch could adopt, distinguishing between the accuracy of migration cues and their efficacy. One way to discuss the magnitude of phenological mismatch is as the difference between the time at which an event (e.g. migration) actually occurs and the time that would be optimal, that is, the accuracy of the cue for that event. Empirical studies often take this approach, by measuring only changes in timing of migration (Mallory et al. 2020, Prytula et al. 2022) or using the difference between the number of days by which arrival timing has advanced and the number of days by which some environmental metric has advanced as a metric of phenological mismatch (Connare and Islam 2022). These approaches can yield valuable insights, however, from a population ecology perspective,

what is important about phenological mismatch are the effects of mis-timed events on fitness, that is, the efficacy of cues. Some empirical work explicitly considers the fitness consequences of migration timing (Møller et al. 2008, Both et al. 2010, Shipley et al. 2020) and theoretical approaches often take fitness consequences as their metrics (McNamara et al. 2011, Johansson and Jonzén 2012, Donohue and Piiroinen 2015).

In our model, cues that are equally inaccurate but in different directions are equally effective due to the sinusoidal nature of the habitat quality functions we used. In other words, differences in efficacy between cue types were purely results of differences in accuracy. This means that using cue accuracy as a proxy for cue efficacy, as has been done in some theoretical work (McNamara et al. 2011), is acceptable if habitat quality functions are sinusoidal. This includes habitat quality functions that are constant over time since this can be achieved by setting the amplitude of a sine curve to zero, although such cases might challenge our assumption that migrants are able to perceive environmental cues. Other functional forms for the habitat quality functions may lead to different results. Migrants often exploit large peaks in resource availability, which may not be well captured by a sine curve. It may also be the case that the increase in habitat quality towards a peak occurs more rapidly than the decrease in habitat quality following a peak. More peaked or asymmetric forms would likely lead to difference in efficacy between cues that are equally inaccurate but in different directions. There may also be differences in efficacy of equally inaccurate cues if habitat quality functions were not smooth, which may be the case in data-derived habitat quality functions. Empiricists could 'use the math' in our paper (Grainger et al. 2021) by calculating the efficacy and accuracy of different cue types using data-derived habitat quality functions in systems where fitness can be understood as a cumulative function of the habitats experienced over time (Shaw and Couzin 2013). It may also be useful to explore qualitative differences in the fitness consequences of early and late migration that appear in empirical systems. For example, in birds, migrating too early might lead to death and starvation (Newton 2007) while migrating too late may lead to failure to find a nest site (Lepage et al. 2000). Future theoretical work separating fitness into mortality and fecundity would improve the

applicability of our modelling framework to migrations driven by different habitat requirements for foraging and breeding (Shaw 2016).

Our model also demonstrates that phenological change can affect both the available benefit of migration and how effective migrants are at capturing this benefit. This implies that we must understand how both of these factors are changing in order to predict the effects of phenological change on migrants. The observation that benefits available are maximized when one habitat reaches its maximum habitat quality at the same time that the other habitat reaches its minimum helps us to understand why some migrants undertake extremely long migrations across the equator (e.g. arctic tern *Sterna paradisaea).* The more synchronized the timing of the peaks were in our model, the lower the benefits available from migration. This aligns with prediction that loss of phenological diversity of resources can have negative fitness impacts on mobile consumers (Armstrong et al. 2016). There are other ways in which climate change is altering the benefits available from migration. For example, warming oceans may make fish migration more beneficial by increasing the quality of circumpolar habitats during the summer while they remain unsuitable during the winter due to polar night (Ljungström et al. 2021). Although our model does not include changes to the size of peaks in habitat quality, predicting the outcome of climate change for fitness requires a consideration of both how the benefits available from migration are changing and the ability of animals to capture those benefits. We recommend that future empirical work on phenological mismatch 'adopt our framework' (Grainger et al. 2021) by studying how the benefits available from migration are changing in addition to changes in how effective migrants are at capturing those benefits.

Future work could explore under what conditions explicitly accounting for the migration trip itself changes our understanding of migrant cues and phenological change. Although we can interpret our results in light of migration distance (given that longer-distance migrants are more likely to use temporal cues, and that habitats used by longer-distance migrants are likely to be more different from one another), our model ignores details of the migration trip. Exploring the assumption that short and long distance migrants have different fitness costs of migration could be done with a simple extension

of our model, since the net-benefit of migration could be calculated as the benefit of migration minus a fitness cost. However, exploring any other details of the migration trip (time spend migrating, path taken, conditions encountered) would require creating a new model. Developing such a model would first require a careful consideration of what happens during the migration period (e.g., how migrants experience habitat quality while migrating (Shaw and Couzin 2013), whether migrants can change speed during migration based on new information (Ahola et al. 2004)). Once these choices were made, one could use similar calculations to those used in this model to identify optimal temporal and environmental cues, and how accurate and effective these cues are following phenological change. We expect that the longer migration takes, the smaller the range of habitat systems for which optimal migration leads to better fitness than residency would be if the habitat quality experienced during migration is zero or negative. We do not expect that the amount of time migration takes would have much effect on the relationship between seasonal amplitude and which cue type is more effective in the context of phenological change.

Future theoretical work could relax some of the assumptions in our model to explore how other aspects of biology including adaptation, plasticity, competition, and life history event timing might affect predictions. These ideas have been explored in other models of migration (Kingsolver et al. 2002, Kokko 2011, Johansson and Jonzén 2012, Fokkema et al. 2020, Kikuchi and Reinhold 2021, Ejsmond et al. 2021, Gurarie et al. 2021) and integrating them into our modelling framework could help to understand how cue type mediates the effects of these factors and vice versa. Future models could apply our framework to a population in which there is variation in cue values (McNamara et al. 2011). This would allow us to identify how cue type affects population-level vulnerability to phenological mismatch and adaptive capacity (Chadwick et al. 2006, Rubolini et al. 2010, Gilroy et al. 2016, Tillotson et al. 2021). Further work could also use our modelling framework to consider the consequences of combining and weighting different cue types to trigger migration (McNamara et al. 2011, Chmura et al. 2019, 2020). In our model, the two cue types are always inaccurate in opposite directions. This

makes it likely that a weighted average of the two cue types would be more accurate and effective than using either cue type on its own.

Future models could also make different assumptions when representing climate change. Representing climate change as a change in season length instead of a horizontal shift in the habitat quality function could be more reflective of processes in the real world and lead to different results (Armstrong et al. 2016, Wang et al. 2021). When relaxing our assumption that the change in phenology is uniform across the entire year, it is important to note that the differences in the migration timing associated with different cues only occur during migration away from a habitat that has undergone a phenological shift. Thus, if phenological shifts are not uniform across the year, we would only expect differences in the efficacy of cues if phenological change occurs in the habitat a migrant is departing from when the migrant is departing. If this condition for differences in the efficacy of cues is met, we would expect the conditions under which each cue type performs best to be similar to what we see in our model. It may also be interesting to incorporate predictability of seasonal variation (Lisovski et al. 2017) and variance of optimal timing across years (McNamara et al. 2011) in order to consider year-to-year variation in the strength of a phenological shift. This could influence the interplay between cues and climate change if bet-hedging favors one cue type over the other. Finally, by changing habitat quality variation at different temporal scales, we could consider phenomena like 'false springs', which are becoming more frequent under climate change (Wang et al. 2021).

Phenological mismatch has been recognized as a mechanism by which climate change might threaten migratory animals (Wilcove and Wikelski 2008). Although the type of cue that is used to trigger migration has been recognized as a factor that might be important in predicting vulnerability to phenological mismatch (Visser et al. 2012, Laforge et al. 2021, Connare and Islam 2022), it has been unclear under what conditions different temporal or environmental cues are more effective because claims regarding the effects of cue type often conflate cue with migration distance (James and Abbott 2014, Chmura et al. 2019, Laforge et al. 2021, Connare and Islam 2022). Migration distance is also often correlated with the accuracy of the environmental cues that are available and

the differences in the strength of seasonality and magnitude of phenological change between a migrant's habitats. We have shown that these factors are also important determinants of which cue type is most effective. Our model reveals that which of the habitats used by a migrant has stronger seasonality and which habitats are experiencing changes in phenology are important to determining which cue type performs better. In the case of latitudinal migrants for whom temperature is a key determinant of habitat quality, our model predicts that environmental cues are more effective at capturing the benefits of migration, aligning with observations of long-distance avian migrants (Visser et al. 2012, Connare and Islam 2022). Our model also offers a theoretical framework that can be used to assess the importance of migratory cue type in the context of changing seasonal patterns of habitat quality.

References

Ahola, M., Laaksonen, T., Sippola, K., Eeva, T., Rainio, K. and Lehikoinen, E. 2004. Variation in climate warming along the migration route uncouples arrival and breeding dates. - Global Change Biology in press.

Alerstam, T., Hedenström, A. and Åkesson, S. 2003. Long-distance migration: Evolution and determinants.

Armstrong, J. B., Takimoto, G., Schindler, D. E., Hayes, M. M. and Kauffman, M. J. 2016. Resource waves: phenological diversity enhances foraging opportunities for mobile consumers. - Ecology 97: 1099–1112.

Bairlein, F. 2016. Migratory birds under threat. - Science 354: 547–548.

Bauer, S. and Hoye, B. J. 2014. Migratory Animals Couple Biodiversity and Ecosystem Functioning Worldwide. - Science 344: 1242552.

Both, C., Bouwhuis, S., Lessells, C. M. and Visser, M. E. 2006. Climate change and population declines in a long-distance migratory bird. - Nature in press.

Both, C., Van Turnhout, C. A. M., Bijlsma, R. G., Siepel, H., Van Strien, A. J. and Foppen, R. P. B. 2010. Avian population consequences of climate change are most severe for long-distance migrants in seasonal habitats. - Proceedings of the Royal Society B: Biological Sciences in press.

Boyle, W. A., Norris, D. R. and Guglielmo, C. G. 2010. Storms drive altitudinal migration in a tropical bird. - Proceedings of the Royal Society B: Biological Sciences 277: 2511–2519.

Chadwick, E. A., Slater, F. M. and Ormerod, S. J. 2006. Inter- and intraspecific differences in climatically mediated phenological change in coexisting Triturus species. - Global Change Biology 12: 1069–1078.

Charmantier, A. and Gienapp, P. 2014. Climate change and timing of avian breeding and migration: evolutionary versus plastic changes. - Evol Appl 7: 15–28.

Chmura, H. E., Kharouba, H. M., Ashander, J., Ehlman, S. M., Rivest, E. B. and Yang, L. H. 2019. The mechanisms of phenology: the patterns and processes of phenological shifts. - Ecol Monogr 89: e01337.

Chmura, H. E., Wingfield, J. C. and Hahn, T. P. 2020. Non-photic environmental cues and avian reproduction in an era of global change. - Journal of Avian Biology in press.

Clausen, K. K. and Clausen, P. 2013. Earlier Arctic springs cause phenological mismatch in long-distance migrants. - Oecologia 173: 1101–1112.

Cohen, J. M., Lajeunesse, M. J. and Rohr, J. R. 2018. A global synthesis of animal phenological responses to climate change. - Nature Clim Change 8: 224–228.

Connare, B. M. and Islam, K. 2022. Failure to advance migratory phenology in response to climate change may pose a significant threat to a declining Nearctic-Neotropical songbird. - Int J Biometeorol 66: 803–815.

Debeffe, L., Rivrud, I. M., Meisingset, E. L. and Mysterud, A. 2019. Sex-specific differences in spring and autumn migration in a northern large herbivore. - Sci Rep 9: 6137.

Dingle, H. and Drake, V. A. 2007. What Is Migration? - BioScience 57: 113–121.

Donohue, J. G. and Piiroinen, P. T. 2015. Mathematical modelling of seasonal migration with applications to climate change. - Ecological Modelling 299: 79–94.

Durant, J. M., Hjermann, D. Ø., Anker-Nilssen, T., Beaugrand, G., Mysterud, A., Pettorelli, N. and Stenseth, N. C. 2005. Timing and abundance as key mechanisms affecting trophic interactions in variable environments. - Ecol Lett 8: 952–958.

Durant, J. M., Hjermann, D. Ø., Ottersen, G. and Stenseth, N. C. 2007. Climate and the match or mismatch between predator requirements and resource availability. - Climate Research 33: 271–283.

Ejsmond, A., Forchhammer, M., Varpe, Ø., Jónsson, J. E. and Jørgensen, C. 2021. Nesting Synchrony and Clutch Size in Migratory Birds: Capital versus Income Breeding Determines Responses to Variable Spring Onset. - The American Naturalist 198: E122–E135.

Fokkema, W., van der Jeugd, H. P., Lameris, T. K., Dokter, A. M., Ebbinge, B. S., de Roos, A. M., Nolet, B. A., Piersma, T. and Olff, H. 2020. Ontogenetic niche shifts as a driver of seasonal migration. - Oecologia 193: 285–297.

Fudickar, A. M., Jahn, A. E. and Ketterson, E. D. 2021. Animal Migration: An Overview of One of Nature's Great Spectacles. - Annual Review of Ecology, Evolution, and Systematics in press.

Garcia, R. A., Cabeza, M., Rahbek, C. and Araújo, M. B. 2014. Multiple Dimensions of Climate Change and Their Implications for Biodiversity. - Science 344: 1247579.

Gill, J. A., Alves, J. A. and Gunnarsson, T. G. 2019. Mechanisms driving phenological and range change in migratory species. - Philosophical Transactions of the Royal Society B: Biological Sciences 374: 20180047.

Gilroy, J. J., Gill, J. A., Butchart, S. H. M., Jones, V. R. and Franco, A. M. A. 2016. Migratory diversity predicts population declines in birds. - Ecology Letters 19: 308–317.

Gordo, O. 2007. Why are bird migration dates shifting? A review of weather and climate effects on avian migratory phenology. - Climate Research in press.

Grainger, T. N., Senthilnathan, A., Ke, P.-J., Barbour, M. A., Jones, N. T., DeLong, J. P., Otto, S. P., O'Connor, M. I., Coblentz, K. E., Goel, N., Sakarchi, J., Szojka, M. C., Levine, J. and Germain, R. M. 2021. ---An empiricist's guide to using ecological theory. - The American Naturalist in press.

Gurarie, E., Potluri, S., Cosner, G. C., Cantrell, R. S. and Fagan, W. F. 2021. Memories of Migrations Past: Sociality and Cognition in Dynamic, Seasonal Environments. - Front. Ecol. Evol. 9: 742920.

Gwinner, E. 1996. Circadian and Circannual Programmes in Avian Migration. - Journal of Experimental Biology 199: 39–48.

Hauser, D. D. W., Laidre, K. L., Stafford, K. M., Stern, H. L., Suydam, R. S. and Richard, P. R. 2017. Decadal shifts in autumn migration timing by Pacific Arctic beluga whales are related to delayed annual sea ice formation. - Global Change Biology 23: 2206–2217.

James, A. R. M. and Abbott, K. C. 2014. Phenological and geographical shifts have interactive effects on migratory bird populations. - Am Nat 183: 40–53.

Johansson, J. and Jonzén, N. 2012. Effects of territory competition and climate change on timing of arrival to breeding grounds: A game-theory approach. - American Naturalist in press.

Jones, T. and Cresswell, W. 2010. The phenology mismatch hypothesis: are declines of migrant birds linked to uneven global climate change? - Journal of Animal Ecology 79: 98–108.

Kauffman, M. J., Aikens, E. O., Esmaeili, S., Kaczensky, P., Middleton, A., Monteith, K. L., Morrison, T. A., Mueller, T., Sawyer, H. and Goheen, J. R. 2021. Causes, Consequences, and Conservation of Ungulate Migration. - Annual Review of Ecology, Evolution, and Systematics in press.

Kikuchi, D. W. and Reinhold, K. 2021. Modelling migration in birds: Competition's role in maintaining individual variation. - Proceedings of the Royal Society B: Biological Sciences in press.

Kingsolver, J. G., Pfennig, D. W. and Servedio, M. R. 2002. Migration, local adaptation and the evolution of plasticity. - Trends in Ecology and Evolution in press.

Kokko, H. 2011. Directions in modelling partial migration: How adaptation can cause a population decline and why the rules of territory acquisition matter. - Oikos in press.

Kölzsch, A., Müskens, G. J. D. M., Kruckenberg, H., Glazov, P., Weinzierl, R., Nolet, B. A. and Wikelski, M. 2016. Towards a new understanding of migration timing: slower spring than autumn migration in geese reflects different decision rules for stopover use and departure. - Oikos in press.

Kuczynski, L., Chevalier, M., Laffaille, P., Legrand, M. and Grenouillet, G. 2017. Indirect effect of temperature on fish population abundances through phenological changes. - PLOS ONE 12: e0175735.

Laforge, M. P., Bonar, M. and Vander Wal, E. 2021. Tracking snowmelt to jump the green wave: phenological drivers of migration in a northern ungulate. - Ecology in press.

Lameris, T. K., Scholten, I., Bauer, S., Cobben, M. M. P., Ens, B. J. and Nolet, B. A. 2017. Potential for an Arctic-breeding migratory bird to adjust spring migration phenology to Arctic amplification. - Global Change Biology 23: 4058–4067.

Lepage, D., Gauthier, G. and Menu, S. 2000. Reproductive consequences of egg-laying decisions in snow geese. - Journal of Animal Ecology 69: 414–427.

Lisovski, S., Ramenofsky, M. and Wingfield, J. C. 2017. Defining the Degree of Seasonality and its Significance for Future Research. - Integrative and Comparative Biology 57: 934–942.

Ljungström, G., Langbehn, T. J. and Jørgensen, C. 2021. Light and energetics at seasonal extremes limit poleward range shifts. - Nat. Clim. Chang. 11: 530–536.

Loarie, S. R., Duffy, P. B., Hamilton, H., Asner, G. P., Field, C. B. and Ackerly, D. D. 2009. The velocity of climate change. - Nature 462: 1052–1055.

Mallory, C. D., Williamson, S. N., Campbell, M. W. and Boyce, M. S. 2020. Response of barren-ground caribou to advancing spring phenology. - Oecologia in press.

McNamara, J. M., Barta, Z., Klaassen, M. and Bauer, S. 2011. Cues and the optimal timing of activities under environmental changes. - Ecology Letters 14: 1183–1190.

Møller, A. P., Rubolini, D. and Lehikoinen, E. 2008. Populations of migratory bird species that did not show a phenological response to climate change are declining. - Proc. Natl. Acad. Sci. U.S.A. 105: 16195–16200.

Newton, I. 2007. Weather-related mass-mortality events in migrants. - Ibis 149: 453–467.

Post, E. and Forchhammer, M. C. 2008. Climate change reduces reproductive success of an Arctic herbivore through trophic mismatch. - Philosophical Transactions of the Royal Society B: Biological Sciences in press.

Prytula, E. D., McKellar, A. E., Schwitters, L. and Reudink, M. W. 2022. Rapid advancement of spring migration and en route adjustment of migration timing in response to weather during fall migration in Vaux's Swifts (Chaetura vauxi). - Canadian Journal of Zoology in press.

Rubolini, D., Saino, N. and Møller, A. 2010. Migratory behaviour constrains the phenological response of birds to climate change. - Clim. Res. 42: 45–55.

Saalfeld, S. T. and Lanctot, R. B. 2017. Multispecies comparisons of adaptability to climate change: A role for life-history characteristics? - Ecology and Evolution 7: 10492–10502.

Servedio, M. R., Brandvain, Y., Dhole, S., Fitzpatrick, C. L., Goldberg, E. E., Stern, C. A., Cleve, J. V. and Yeh, D. J. 2014. Not Just a Theory—The Utility of Mathematical Models in Evolutionary Biology. - PLOS Biology 12: e1002017.

Shaw, A. K. 2016. Drivers of animal migration and implications in changing environments. - Evolutionary Ecology in press.

Shaw, A. K. and Couzin, I. D. 2013. Migration or residency? the evolution of movement behavior and information usage in seasonal environments. - American Naturalist in press.

Shipley, J. R., Twining, C. W., Taff, C. C., Vitousek, M. N., Flack, A. and Winkler, D. W. 2020. Birds advancing lay dates with warming springs face greater risk of chick mortality. - Proceedings of the National Academy of Sciences of the United States of America in press.

Taylor, C. M., Laughlin, A. J. and Hall, R. J. 2016. The response of migratory populations to phenological change: a Migratory Flow Network modelling approach. - Journal of Animal Ecology 85: 648–659.

Tillotson, M. D., Arostegui, M. C., Austin, C. S., Lincoln, A. E., Matsubu, W., McElroy, K. N. and Quinn, T. P. 2021. Challenges in the Identification and Interpretation of Phenological Shifts: Anthropogenic Influences on Adult Migration Timing in Salmonids. - Reviews in Fisheries Science & Aquaculture 29: 769–790.

Tsujii, K., Otsuki, M., Akamatsu, T., Amakasu, K., Kitamura, M., Kikuchi, T., Fujiwara, A., Shirakawa, H., Miyashita, K. and Mitani, Y. 2021. Annual variation of oceanographic conditions changed migration timing of bowhead whales Balaena mysticetus in the southern Chukchi Sea. - Polar Biol 44: 2289–2298.

Visser, M. E., Te Marvelde, L. and Lof, M. E. 2012. Adaptive phenological mismatches of birds and their food in a warming world. - J Ornithol 153: 75–84.

Wang, J., Guan, Y., Wu, L., Guan, X., Cai, W., Huang, J., Dong, W. and Zhang, B. 2021. Changing Lengths of the Four Seasons by Global Warming. - Geophysical Research Letters 48: e2020GL091753.

Wilcove, D. S. 2008. Animal migration an endangered phenomenon? 24: 71–78.

Wilcove, D. S. and Wikelski, M. 2008. Going, going, gone: Is animal migration disappearing? - PLoS Biology in press.

Tables

Table 1.1: Variables, functions and parameters

Habitat 1 function	$H_1(t)$
Original Habitat 2 function	$H_2(t)$
Transformed Habitat 2 function	$H_{2,N}(t)$
Amplitude of Habitat 1 function	a
Horizontal translation between Habitat 1 and Habitat 2 functions	b
Horizontal translation between original Habitat 2 and transformed Habitat 2 functions	c

What time migration occurs using cues that are…	Temporal	Environmental
Optimal based on the original habitat system, in the original habitat system	t_1^*, t_2^*	τ_1^*, τ_2^*
Optimal based on the original habitat system, in the new habitat system	$t_1^{\hat{}}, t_2^{\hat{}}$	$\tau_1^{\hat{}}, \tau_2^{\hat{}}$
Optimal based on the new habitat system, in the new habitat system	t_1^{**}, t_2^{**}	τ_1^{**}, τ_2^{**}

What is the fitness of a migrant using cues that are…	Temporal	Environmental
Optimal based on the original habitat system, in the original habitat system	Q_t^*	Q_e^*
Optimal based on the original habitat system, in the new habitat system	$Q_t^{\hat{}}$	$Q_e^{\hat{}}$
Optimal based on the new habitat system, in the new habitat system	Q_t^{**}	Q_e^{**}
How well do old cues perform in the new habitat system?	Temporal	Environmental
In terms of accuracy	$\left\|t_1^{\hat{}} - t_1^{**}\right\| + \left\|t_2^{\hat{}} - t_2^{**}\right\|$	$\left\|\tau_1^{\hat{}} - \tau_1^{**}\right\| + \left\|\tau_2^{\hat{}} - \tau_2^{**}\right\|$
In terms of efficacy	$Q_t^{\hat{}} - Q_t^{**}$	$Q_e^{\hat{}} - Q_e^{**}$

Figures

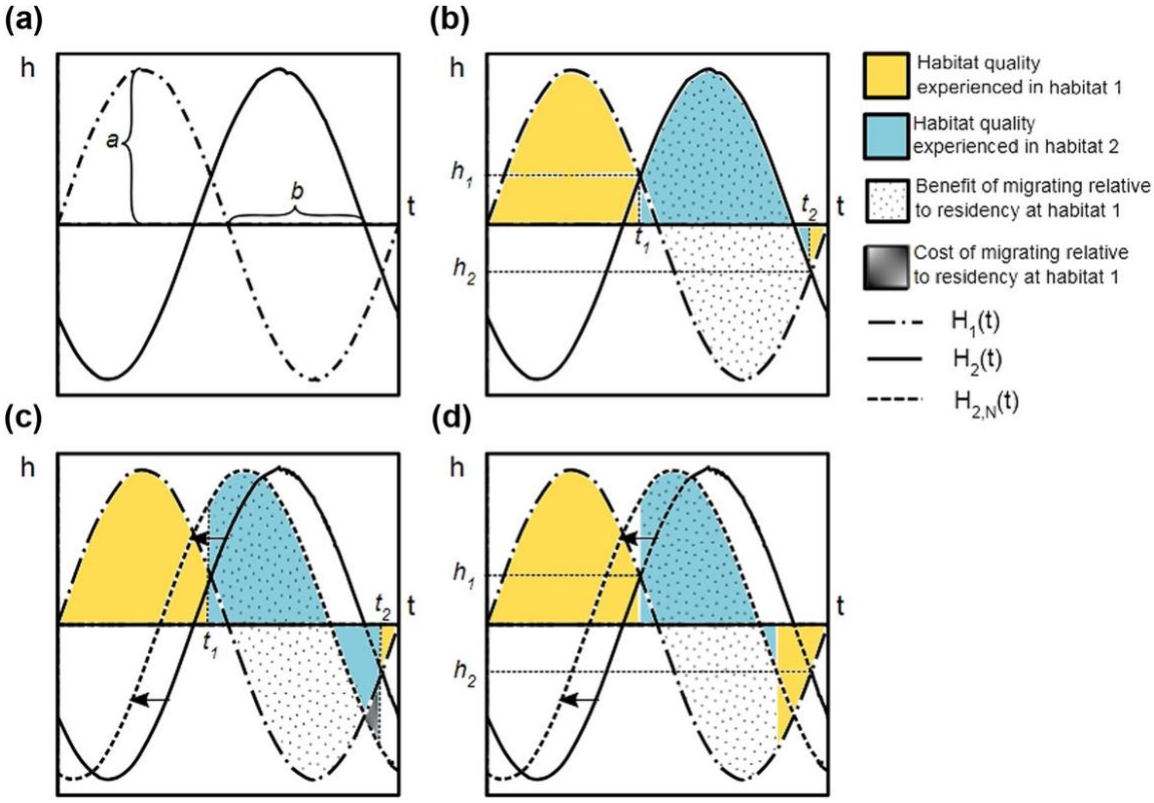

Figure 1.1. Model description. a) We define two habitat functions ($H_1(t)$ and $H_2(t)$) in which habitat quality (h) varies as a function of time (t). b) The migration schedule that maximizes the benefits of migration has migration occurring at the points (t_1^*, h_1^*) and (t_2^*, h_2^*) where these habitat functions cross. After a change to habitat 2, in which the habitat function for habitat 2 is now given by $H_{2,N}(t)$ we consider c) migration using temporal cues and d) migration using environmental cues.

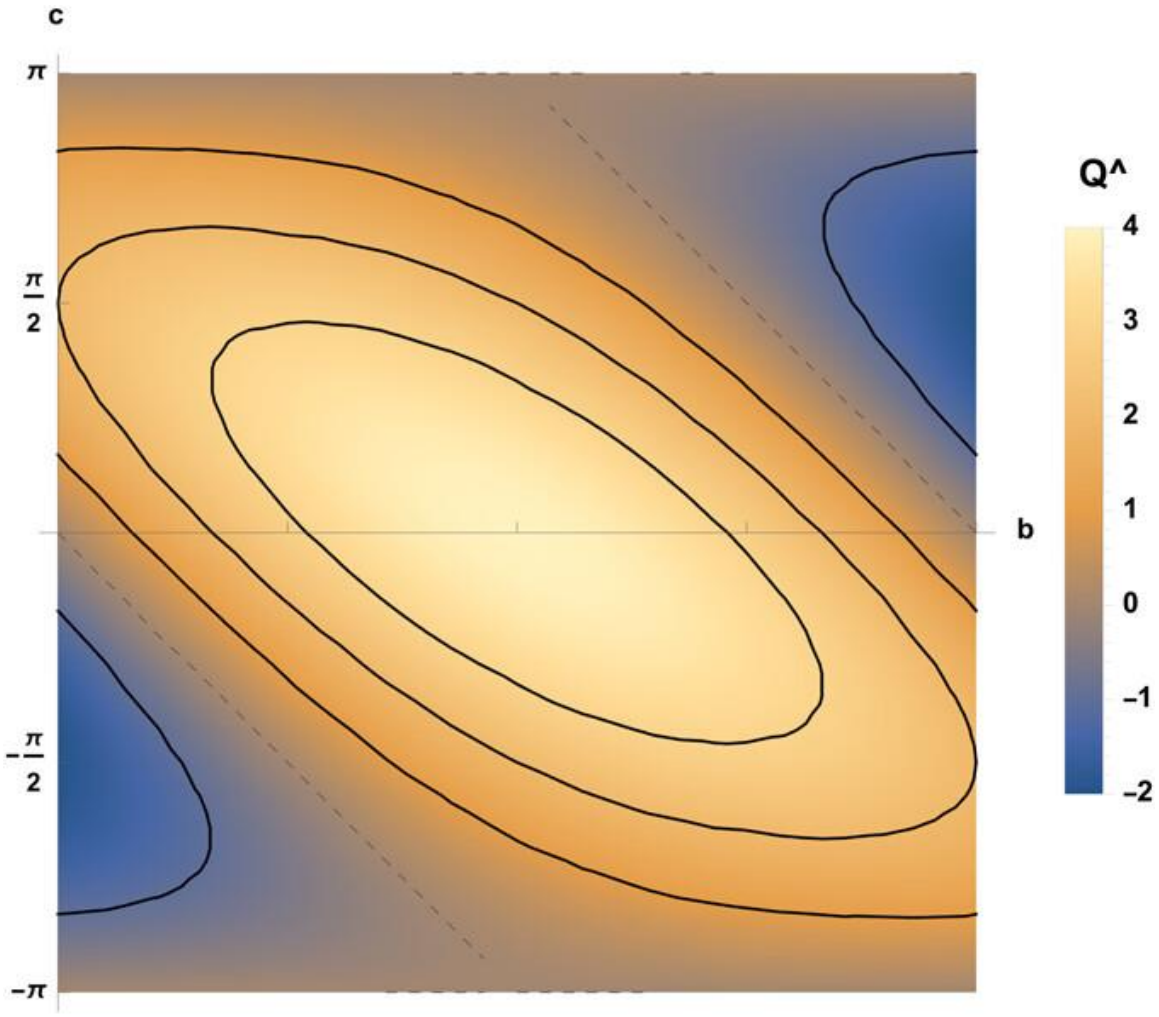

Figure 1.2: Benefit of migration following phenological change when the two habitats have equal amplitudes. The experienced benefit from migration following horizontal shift when both habitat quality functions have amplitude 1 is given by $Q_t^{\wedge} = Q_e^{\wedge} = 2\sin\left(\frac{b}{2}\right) + \sin\left(\frac{b}{2} + c\right)$ where b is the original horizontal shift between habitat 1 and habitat 2 (x-axis) and c is the horizontal shift representing a phenological change to habitat 2 (y-axis). The isoclines where $Q^{\wedge} = 0$ are shown using dashed lines. (migrants do better than residents– yellow areas, migrants do worse than residents – blue areas)

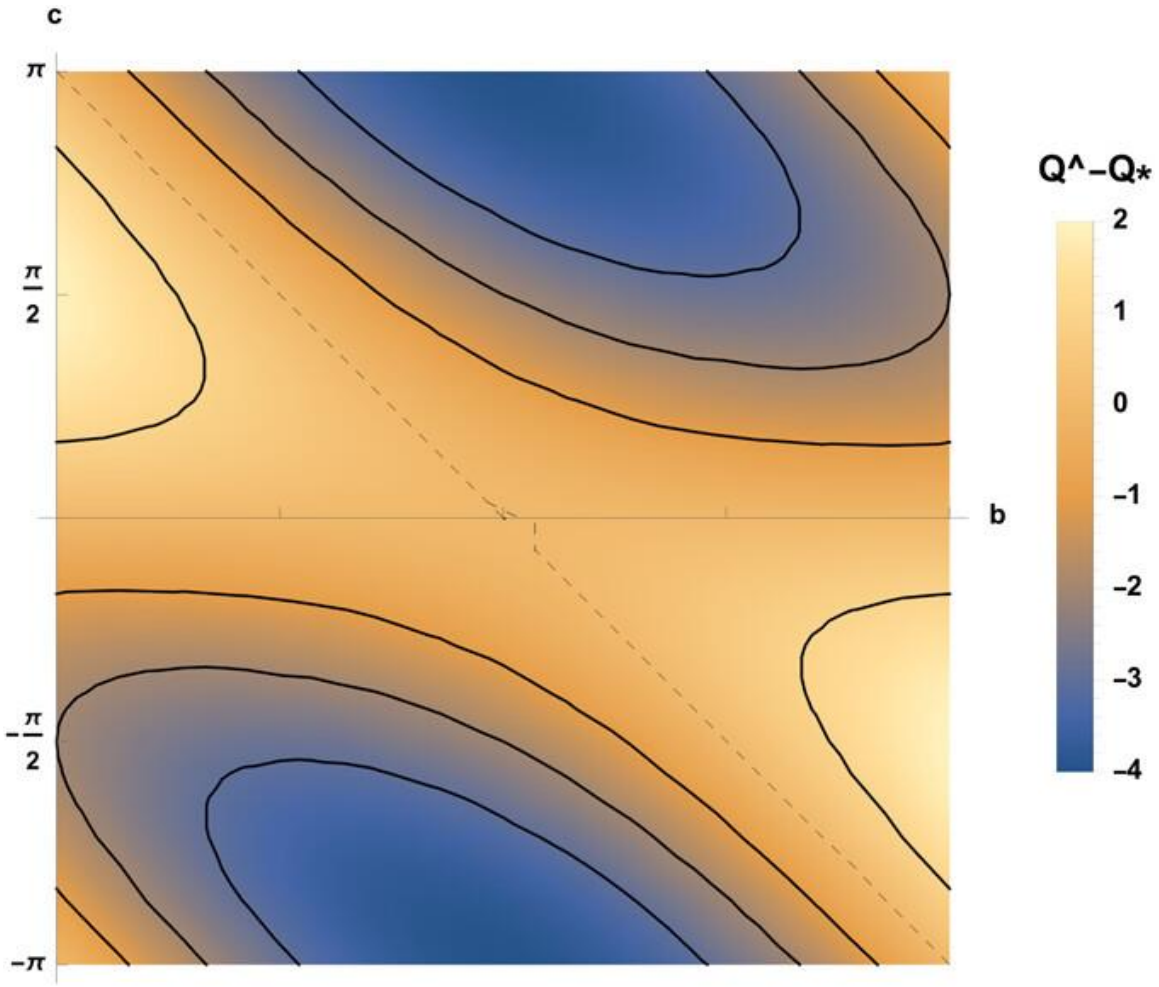

Figure 1.3: Change in benefit of migration when the two habitats have equal amplitudes. Regardless of cue type, the change in the benefit of migration is given by $Q^{\wedge} - Q^{*} = 4\cos\left(\frac{b+c}{2}\right)\sin\left(\frac{c}{2}\right)$. The isocline on which $Q^{\wedge} - Q^{*} = 0$ is shown with a dashed line. (migrants do better after phenological change than before– yellow areas, migrants do worse after phenological change than before – blue areas)

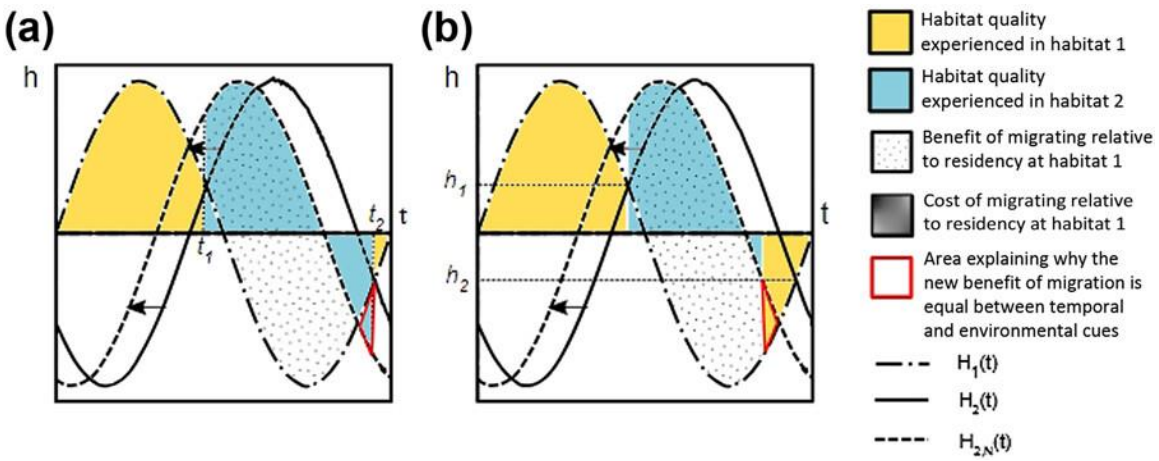

Figure 1.4. Graphical explanation of equal efficacy of temporal and environmental cues. Following a horizontal translation of $H_2(t)$ a) the cost of migrating too late from habitat 2 to habitat 1 using temporal cues is equal to b) the missed benefit due to migrating too early from habitat 2 to habitat 1 using environmental cues.

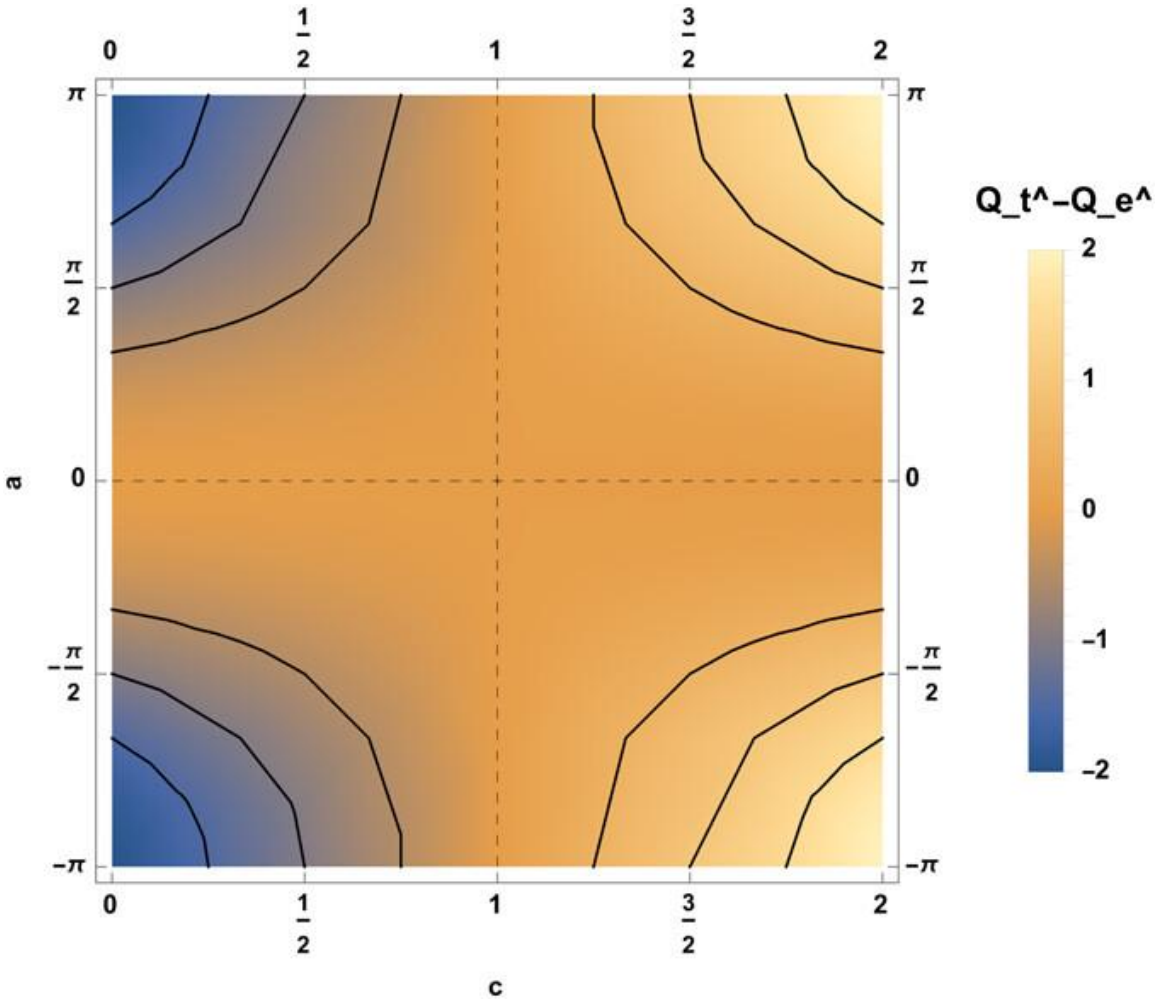

Figure 1.5. Differences between the efficacy of temporal and environmental cues. The difference between the benefit of migration received by a migrant using temporal cues and environmental cues following a horizontal shift of size c to habitat 2 ($Q_t^{\wedge} - Q_e^{\wedge}$) when $H_1(t) = a\sin(t)$ and $H_2(t) = \sin(t-b)$ ($H_{2,N}(t) = \sin(t-b-c)$) as a function of a and c when $b = \pi$. Environmental cues are more effective when habitat 2 has a larger amplitude and habitat 2 undergoes a horizontal shift (blue areas). Temporal cues are more effective when habitat 1 has a larger amplitude and habitat 2 undergoes a horizontal shift (orange areas). The isoclines on which $Q_t^{\wedge} - Q_e^{\wedge} = 0$ are shown using dashed lines.

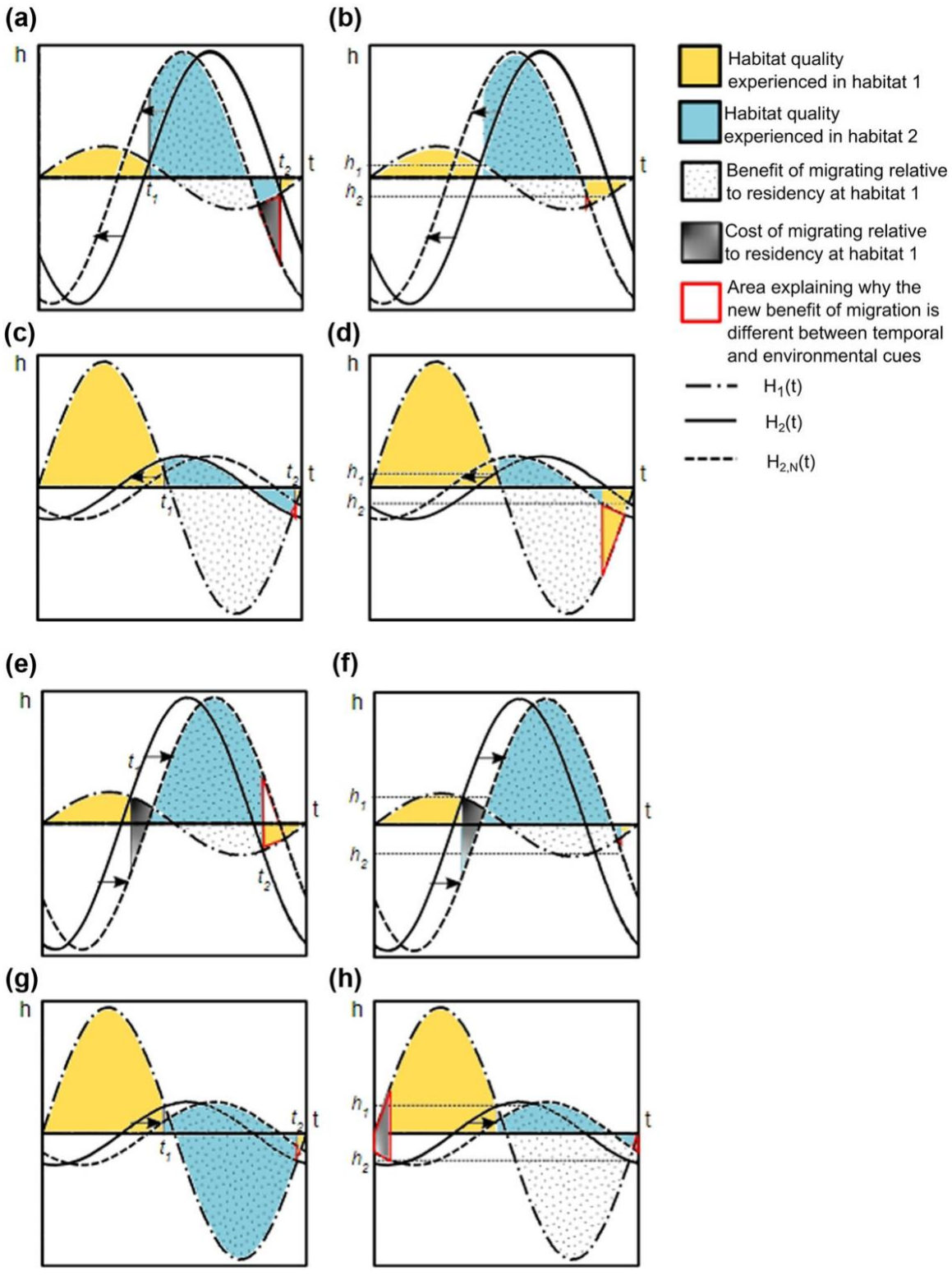

Figure 1.6: Case studies for differences between cues in different amplitudes case. In panels a-d, $H_{2,N}(t)$ is shifted to the left from $H_2(t)$. In these cases, migration from habitat 2 back to habitat 1 occurs later than

optimal using temporal cues (a and c) and earlier than optimal using environmental cues (b and d). When the amplitude of $H_1(t)$ is smaller than the amplitude of $H_2(t)$ (a and b), migrating too late (a) is worse than migrating too early (b). When the amplitude of $H_1(t)$ is larger than the amplitude of $H_2(t)$ (c and d), migrating too late (c) is better than migrating too early (d). In panels e-h, $H_{2,N}(t)$ is shifted to the right from $H_2(t)$. In these cases, migration from habitat 2 back to habitat 1 occurs earlier than optimal using temporal cues (e and g) and later than optimal using environmental cues (f and h). When the amplitude of $H_1(t)$ is smaller than the amplitude of $H_2(t)$ (e and f), migrating too early (e) is worse than migrating too late (f). When the amplitude of $H_1(t)$ is larger than the amplitude of $H_2(t)$ (g and h), migrating too early (g) is better than migrating too late (h).

Chapter 2

Population response to extreme climate events depends on population spatial distribution

Abstract

Extreme climate events, which are increasing in frequency and severity with climate change, can cause mass mortality events in animal populations. Meanwhile, populations of migratory animals around the world are in decline. We illustrate how the spatial aggregation typical in many migratory populations can increase the likelihood of population declines in response to extreme climate events. First, we demonstrate that high levels of spatial aggregation make it possible for higher levels of population mortality to result from spatially limited disturbances. This aligns with observations of mass mortality events due to extreme climate events in migratory animal populations. We go on to use a flow-kick model to demonstrate that because higher levels of spatial aggregation result in less frequent, but more severe impacts, population crashes in response to extreme events are more likely in highly aggregated populations. This provides a mechanism by which migratory populations may be especially vulnerable to climate change. We quantify what regimes of disturbance (with respect to frequency and severity) lead to population collapse versus resilience, and we show how our results depend on the form of disturbance (proportional vs density-dependent). Finally, we compare results from an analytic approximation with those from a simulation and discuss differences. The results of our model can also be used to understand the interacting effects of shifting extreme climate event regimes and land use change. We predict that land use changes that increase the spatial aggregation of populations, such as habitat destruction or degradation of habitat corridors, will increase the likelihood of population declines due to extreme

climate events. Conservation plans that increase the dispersion of populations across the landscape may increase population resilience to changing extreme climate event regimes.

Introduction

Many migratory populations around the world experience mass mortality events (MMEs) linked to extreme climate events (ECEs) (Barton et al., 2023; Newton, 2007). Examples include rain on snow events for caribou and other ungulates (Kauffman et al., 2021), marine heatwaves for seabirds and fish (Jones et al., 2018; Piatt et al., 2020; Roberts et al., 2019), extreme wind events and storms for birds (Louzao et al., 2019; Newton, 2007), and wildfires for many animals (Jolly et al., 2022; Yang et al., 2021). Many of these events are spatially limited but have large effects on populations. Understanding the impact of ECEs on populations is urgent because ECEs are increasing in frequency and severity, which has led to an increase in the frequency and severity of MMEs (Fey et al., 2015, 2019; Lamberti et al., 2020). Mortality events induced by ECEs may contribute to population declines, including the declines of many migratory populations (Wilcove & Wikelski, 2008). Further, mass mortality events are relevant to community and ecosystem dynamics (Fey et al., 2019).

The spatial aggregation typical of migratory populations (Rubenstein & Hack, 2013) may mediate population-level responses to ECEs or other spatially restricted disturbances. Logically, the space a population occupies determines where it will be affected by disturbances. The more spread out a population is, the more locations at which the population can be affected by disturbances. The more aggregated a population is, the larger the proportion of a population that can be affected by a disturbance affecting any one location. Thus, any factor that affects the distribution of a population across a landscape, including factors affecting habitat selection and habitat availability (Picardi et al., 2024), will affect population response to disturbance.

Migratory populations may exhibit spatial aggregation for many reasons. There may be benefits of aggregation, like predator avoidance, navigation and mate finding (Parrish & Edelstein-Keshet, 1999; Rubenstein & Hack, 2013). Migratory populations may also exhibit spatial aggregation because their movement allows them to exploit peaks in resource availability that can support large numbers of individuals for a short period of time (Fryxell et al., 1988), thus reducing the effect of spatial proximity on competition intensity (Law et al., 2003). Although there are also costs associated with

aggregation (e.g. competition, disease, potential for an increase in vulnerability to predation (Parrish & Edelstein-Keshet, 1999)), these costs have historically not prevented aggregation from persisting through evolutionary time in many migratory populations. An increase in the costs of aggregation associated with extreme climate events may shift this balance. It is unclear when we should expect that population spatial aggregation will increase vulnerability to ECEs.

The spatial distribution of populations is also influenced by patterns of habitat availability. That is, if we assume that the habitat used by a population is a subset of the habitat that is available (Johnson, 1980; Picardi et al., 2024), it is likely that we will observe more spatially aggregated populations if the habitat that is available is spatially aggregated. Therefore, patterns of spatial distribution due to habitat availability also have the potential to mediate response to extreme climate events. Habitat availability may be subject to management action, so it is useful to understand how the distribution of populations mediates the effects of extreme climate events when designing management plans in the face of climate change.

Flow-kick models, which combine continuous changes ('flows') and discrete changes ('kicks'), have been developed to analyze the effects of discrete disturbance events. This type of model has been used to consider the effects of different harvesting regimes on population dynamics (Meyer et al., 2018). In this paper, the size of the harvest was independent of the size of the population. We do not expect that disturbances due to ECEs would take the same functional form. Rather, we expect that ECEs that cause direct mortality (e.g. wildfires, extreme wind events) would be more likely to lead to the mortality of a fixed proportion of the population. It is also possible that ECEs that generally act by restricting access to food or other resources may lead to mortality for a larger proportion of the population when the population size is higher (e.g. rain on snow events (Hansen et al., 2019)).

We developed a mathematical model to determine how the spatial distribution of a population affects response to spatially restricted disturbances. We determined how spatial aggregation affects the severity and frequency of disturbance that is experienced by a population and used a flow-kick modelling framework (Meyer et al., 2018) to

consider the effects of different disturbance regimes on population dynamics. This understanding of how spatial population distribution interacts with disturbance regimes provides a mechanism by which migration as a movement strategy may mediate population response to climate change. This model also allows us to make predictions about how changing disturbance regimes and patterns of habitat availability can interact to determine population dynamics.

Methods

Part 1: Functional forms of kicks

In a flow-kick population model, the population grows according to a flow function between instantaneous kicks to population size. In our model, the population size changes according to a logistic growth model with a strong Allee effect,

$$\frac{dN}{dt} = N(1 - \frac{N}{K})(\frac{N}{A} - 1) \quad (2.1)$$

where N is the population size, K is the carrying capacity and A is the Allee threshold (i.e., the threshold below which a population cannot grow). This population growth (the "flow") occurs for some time interval τ. Then, we implement a discrete "kick" that eliminates some proportion κ of the population. We consider two possible forms for the kick; kicks that are proportional to population size and kicks that are density-dependent such that the proportion of the population eliminated by the kick is smaller at smaller population sizes. For a proportional kick, the population size after the kick is

$$N(1 - \kappa). \quad (2.2a)$$

Alternately, for a density dependent kick, the population size after the kick is

$$N(1 - \kappa(N/100)). \quad (2.2b)$$

We use the methods in Meyer et al. 2018 to find the resilience threshold. The resilience threshold divides the κ and τ parameter space into a region where disturbances are small/rare enough that the population can recover from them, and a region where disturbances are large/frequent enough that the population is driven extinct by them (Figure 2.1c).

Part 2: Effects of spatial extent of disturbance and population spatial distribution

To incorporate the spatial extent of disturbance and spatial distribution of a population into a flow-kick model, we divide the landscape into T total sites and say that a disturbance impacts D of the T sites and the population is divided evenly across H of the T sites (where there is potentially some overlap between the H occupied and D disturbed sites). We also say that disturbances occur on the landscape every ν time steps and that when a disturbance impacts a site occupied by the population, in the case of proportional kicks, a proportion μ of the individuals occupying that site will die. In the case of density-dependent kicks, this proportion is modified by the population size. Note that the time interval at which disturbances impact the population (τ) is determined by the frequency at which disturbances occur on the landscape (ν) and the probability that any given disturbance affects sites occupied by the population.

Analytical Methods

When the population either occupies exactly one site ($H = 1$) or is spread out across all the sites ($H = T$), we can express the flow-kick model parameters κ (kick size) and τ (kick interval) as functions of T, D, H, ν and μ. This allows us to explore the resilience thresholds analytically using the methods in Meyer et al. 2018. When $H = 1$, $\tau = \nu(T/D)$, i.e., disturbances will affect the population every $\nu(T/D)$ time steps because the probability that any given disturbance will affect the cell occupied by the population is D/T and disturbances happen every ν time steps. When a disturbance impacts the population, the entire population will be in an affected cell, so $\kappa = \mu$. In this case, the average disturbance severity (κ/τ) is $\mu D/\nu T$. When $H = T$, $\tau = \nu$, i.e., disturbances will affect the population every ν time steps because every disturbance that occurs will affect the population. When a disturbance impacts the population, D/T of the population will be in an affected cell, so $\kappa = \mu(D/T)$. In this case, the average disturbance severity (κ/τ) is $\mu D/\nu T$. Note that the average disturbance severity (κ/τ) is the same when $H = 1$ and $H = T$. However, as long as not all sites are disturbed ($D < T$), both the kick size (κ) and

the kick interval (τ) are smaller when all sites are occupied ($H = T$) than when only one site is ($H = 1$).

When population spatial aggregation is intermediate (i.e. $1 < H < T$), a disturbance with the same values of D and μ can have one of many possible impacts on a population (i.e. many possible values of κ, τ). Instead of considering the impact of a single combination of κ and τ (as above), we must consider the distribution of possible kick sizes and their associated frequencies. To do this analytically, we use the hypergeometric distribution to determine the frequency with which disturbances will affect different proportions of the population. Specifically, we know that when a disturbance affecting D of T sites occurs, the probability that exactly S of the H occupied sites (and thus a proportion exactly S/H of the population) will be affected by the disturbance is given by the probability mass function of the hypergeometric distribution

$$\Pr(S = s) = \frac{\binom{D}{s}\binom{T-D}{H-s}}{\binom{T}{H}}. \tag{2.3}$$

The same probability mass function applies if both the inhabited sites and the disturbed sites are chosen randomly or if one of these is fixed and the other is chosen randomly.

We define τ_S to be the time interval between kicks that affect S occupied sites. Although τ_S is stochastic, we can make an approximation that it is is equal to the time interval at which extreme events occur on the landscape (ν) divided by the probability that exactly S sites will be affected by the disturbance ($Pr(S = s)$), thus

$$\tau_S = \frac{\nu\binom{T}{H}}{\binom{D}{s}\binom{T-D}{H-s}}. \tag{2.4a}$$

We define κ_S to be the proportion of the population killed by disturbances that affect S occupied sites, which is equal to the proportion of individuals in affected sites that are killed (μ) multiplied by the proportion of the population that is in an affected site (S/H). Thus,

$$\kappa_S = \frac{S}{H}\mu. \tag{2.4b}$$

In general, S (the number of occupied sites that are disturbed) cannot be larger than either D (the number of disturbed sites) or H (the number of occupied sites). At the

other extreme, if the number of occupied sites added to the number of disturbed sites is greater than the total number of sites ($H + D > T$) then some sites must be both occupied and disturbed. Specifically, at least $H + D - T$ sites must be both occupied and affected by disturbance. Taken together, this lets us put bounds on possible values of *S*:$H + D - T \leq S \leq H, D$. When this leaves only one possible value of *S*, then we can use the methods for the extreme values of spatial distribution (above) to approximate population dynamics since we have one value of τ and one value of κ.

When there are several possible values of *S*, determining whether the population is resilient to the kick regime analytically is more challenging. If there exists a possible value of *S* for which the population would crash in response to kicks that kill a proportion κ_S occurring at time interval τ_S, the population will crash in response to the full regime of kicks. However, picking out one value of *S* means that we are taking only a subset of the kicks that occur into account. It is possible that even if for every possible value of *S*, the population is resilient to kicks with severity κ_S occurring every τ_S time steps, the population will still not be resilient to the full suite of kicks. We can go further and note that if there is some possible *S* value S_i for which the frequency of kicks affecting at least S_i sites is higher than the maximum frequency that can yield resilience to kicks that affect S_i/H of the population, the population will not persist. To determine whether a population is likely to be resilient to a regime of kicks, for each possible value of S_i, we find κ_{S_i} and the frequency with which kicks affect at least S_i sites

$$\tau_{S_i \leq S} = \sum_{S=S_i}^{H,D} \tau_S \, . \tag{2.4c}$$

Then, for all possible S_i values, we determine if $(\kappa_{S_i}, \tau_{S_i \leq S})$ is in the resilient region of (κ, τ) parameter space or not. If for any value of S_i , $(\kappa_{S_i}, \tau_{S_i \leq S})$ is in the non-resilient region, we say that the population is not resilient to the kick regime.

We use these tests to assess whether a population crash is likely for the same parameter values that we explore in our simulation (see Table 2.1).

Simulation Methods

The analytical tests described may not capture all of the kick regimes that are likely to lead a population to crash because they look only at a subset of the kicks that are occur

and do not consider the full severity of some kicks. Specifically, for every S_i, we do not consider the effects of kicks that affect $S < S_i$ sites occupied by the population and for all $S > S_i$ we do not consider that κ_S is larger than κ_{S_i}. In order to capture the full effect of all kicks in a given kick regime and to incorporate the stochasticity we expect in real world scenarios, we built a simulation.

In the simulation, first, the population grows according to a logistic growth model with a strong Allee effect (Eq. 2.1) for a time interval ν *(*the time interval at which disturbance occurs on the landscape). Next, the number of sites that are both occupied and affected by the disturbance (S) is drawn from Hypergeometric(*T,D,H).* The magnitude of disturbance (κ_S) is given by Eq. 2.4b. Finally, the kick is implemented and the population size is equal to either Eq. 2.2a for a proportional kick or Eq. 2.2b for a density-dependent kick. These steps are repeated 100 times.

To determine how population spatial aggregation affects the probability of population persistence in the face of a given disturbance regime, we count the number of simulations out of 100 for which the population has size 0 at the end of 100 flow-kick iterations. In our simulations, the total number of sites (T) is 100, the time interval at which disturbances occur on the landscape (ν) is 1 and the proportion of the population in affected cells that is killed (μ) is 1. We run a simulation for all combinations of a number of sites affected by a disturbance (D) between 1 and 100 and a number of sites occupied by a population (H) between 1 and 100.

Results

Part 1: Functional forms of kicks

For both proportional and density-dependent kicks, there are two ways in which whether or not a population crashes in response to a kick regime depends on the severity (κ) and frequency (τ) of the kicks. First, kicks that are sufficiently severe will lead to a crash at any frequency (i.e. there is a maximum value of κ to which a population can be resilient). Any κ larger than $1 - A/K$ will lead to a population crash for both kick types because it

will reduce the population size below the Allee threshold (Figure 2.1c, the region to the right of κ=0.8). Second, the population can crash if κ is smaller than $1 - A/K$ but kicks are sufficiently frequent (τ is sufficiently small). Kick regimes in which the kicks are more severe (κ is larger) and kick regimes in which kicks are more frequent (τ is smaller) are more likely to lead to population crashes (Figure 2.1c). Further, when the average kick severity (κ/τ) is held constant, there are cases in which lower severity kicks occurring at high frequencies (κ is small and τ is small) do not lead a population to crash while higher severity kicks occurring at low frequencies (κ is large and τ is large) do (Figure 2.1).

There are differences between the functional forms of kicks. The resilience boundary (i.e. the line that divides kick regimes that lead to population crashes from those that do not) is further towards high severity/ low frequency for density-dependent kicks than for proportional kicks (Figure 2.1c). If we investigate a particular average kick severity (κ/τ), the minimum value of κ required to induce a population crash is lower for proportional kicks than for density-dependent kicks (Figure 2.1). Similarly, for any given value of κ, the value of τ necessary for resilience is smaller for density-dependent kicks than for proportional kicks (Figure 2.1c).

Part 2: Effects of spatial extent of disturbance and population spatial distribution

Analytical Approach

Recall that the average disturbance severity (κ/τ) is equal when one site is occupied ($H = 1$) and when all sites are occupied ($H = T$), but disturbance frequency is higher when $H = T$ and disturbance severity is higher when $H = 1$. Using the results in Part 1, this indicates that less spatially aggregated populations may be more resilient to disturbance. We observe that larger spatial extent of disturbance (D) can increase the likelihood that a population will crash in both the $H = 1$ and the $H = T$ case but how this occurs is different. When H=T this is because the larger the spatial extent of disturbance, the larger the kick size (κ) will be (i.e. higher values of D can move the kick regime to

the right across the resilience boundary in Figure 2.1c). When *H=1* this is because the larger the spatial extent of disturbance, the higher the frequency of kicks (τ) (i.e. higher values of *D* can move the kick regime to down across the resilience boundary in Figure 2.1c). This difference means that the proportion of an affected population that will be killed by a disturbance (μ) and the spatial extent of disturbance (*D*) interact differently in the $H = 1$ and $H = T$ cases. It is generally true that in the $H = T$ case, for all combinations of μ, υ, there is a sufficiently small D value such that the population will not crash while some values of μ will always cause a population to crash in the $H = 1$ case. Namely, when $\mu > 1 - A/K$, a sufficiently small value of *D* can lead to resilience in the *H=T* case but not the *H=1* case.

We use properties of the hypergeometric distribution to make more general statements about the relationship between population spatial aggregation and experienced frequency and severity of disturbance. The mean number of affected sites (*S)* in the hypergeometric distribution is *H(D/T)*. If we divide this by *H* to get the proportion of the population affected by disturbance, we get *D/T* which is constant as we vary *H*. Thus, the number of sites occupied by the population *(H)* does not affect the mean proportion of the population affected by a disturbance. However, *H* does affect the probability that different proportions of the population are affected. As we saw above, if *H=1*, then 100% of the population will be affected by a disturbance *D/T* of the times a disturbance occurs. At the other extreme, if *H=T,* then *D/T* of the population will be affected by a disturbance every time a disturbance occurs. If *H>T-D* (the population is spread out across a minimum number of sites) then at least *H-T+D* sites occupied by the population will be affected by any disturbance. Therefore, if the population is sufficiently spread out, at least some of the population will be affected by every disturbance. Similarly, if *H>D* then there is a maximum proportion less than 100% (*D/H)* of the population that can be affected by any disturbance. Therefore, if a population is sufficiently spread out relative to the spatial scale of the disturbance, it will never experience a disturbance that impacts the entire population. We continue to see that larger values of *H* increase the frequency and decrease the severity of extreme event impacts, strengthening the argument that

lower levels of spatial aggregation decrease the likelihood of crashes in response to extreme events.

We use two criteria to determine whether a kick regime is likely to induce a population crash. First, any kick regime that includes individual kicks that bring a population at carrying capacity below its Allee threshold is likely to induce a population crash (criteria 1). For both density-dependent and proportional kicks, this condition is met when a single kick that can affect at least $(1 - A/K)/\mu$ of the population. Given a sufficiently high value of μ, this is equivalent to the proportion of sites affected by a disturbance being sufficiently large, i.e. $D/H > (1 - A/K)/\mu$. With the parameter values used in our simulation (A=100, K=20, μ=1), any case in which $D > 0.8H$ is likely to lead to a population crash due to a single kick (Figure 2.2, yellow regions). Second, we consider values of D, H for which we predict that the population will crash because there exists a possible S_i for which (κ_{S_i},$\tau_{S_i \leq S}$) is in the 'not resilient' region of (κ, τ) parameter space (criteria 2). We find that there are more D, H values for which this is true for proportional than for density-dependent kicks. These values are for intermediate levels of D and high levels of H (Figure 2.2, green regions).

Simulation Approach

Decreasing the spatial aggregation of a population decreases the likelihood of a crash and increasing the spatial extent of disturbance increases the likelihood of a crash, matching our analytic results. When the number of disturbed sites (D) is sufficiently large, populations always crash regardless of population spatial distribution. When D is sufficiently small, populations never crash regardless of population spatial distribution. For intermediate values of D, decreasing spatial aggregation of the population (increased H) decreases the probability that the population will crash (Figure 2.3).

Discussion

We illustrated how spatial aggregation can decrease population resilience to spatially restricted disturbances. We applied the hypergeometric distribution to the setting of disturbance to demonstrate that high levels of spatial aggregation make it possible for

higher levels of population mortality to result from spatially limited disturbances while decreasing the frequency at which a population is affected by disturbance. We used a flow-kick model to demonstrate that because higher levels of spatial aggregation result in less frequent but more severe disturbances, population crashes in response to disturbance are more likely in highly aggregated populations.

Robustness of our findings

The qualitative results of our model are robust to the functional form of the kick. Regardless of whether kicks are proportional or density-dependent, for a constant average kick severity, less frequent and more severe events are more likely to lead to a population crash. This is also true when kicks are of constant magnitude (Meyer et al., 2018). Therefore, we expect that for a many extreme event types, higher levels of population spatial aggregation and more severe, widespread, and frequent extreme events are more likely to lead to population crashes. However, the resilience boundary for density-dependent kicks was further towards high kick severity and frequency than the resilience boundary for proportional kicks. Both of these resilience boundaries are further towards high kick severity and frequency than the resilience boundary for constant kicks (Meyer et al., 2018). Therefore, population crashes may be less likely in response to extreme climate events than to harvesting (as was investigated in Meyer et al., 2018) and population crashes may be even less likely in response to extreme events for which the proportional impact on a population decreases with population size.

The estimates using the analytic approach indicate that populations will be resilient for a smaller amount of parameter space than the simulation approach. This is odd because in the analytical approach, we used criteria that left out some kicks and took some kicks to be smaller than they actually are, which should lead to estimating that a population would be less likely to crash than it actually is. However, one of our analytical criteria for predicting a crash is the possibility of a kick that would bring a population at its carrying capacity below the Allee threshold. These kicks may be sufficiently infrequent that they rarely occur over the 100 time steps in the simulations.

Empirical implications

Migratory populations often exhibit high degrees of spatial aggregation (Rubenstein & Hack, 2013). Our model indicates that this may lead to migratory populations being more vulnerable to extreme climate events than non-migratory populations. Empirically, we see declines in the sizes of migratory populations, and these declines have been hypothesized to be related to climate change (Wilcove & Wikelski, 2008). We provide one mechanism by which migration may be associated with higher vulnerability to climate change.

In addition to providing insight into how the spatial aggregation associated with migration mediates response to extreme climate events, we can use our results to make predictions about how land use change interacts with ECEs to determine population dynamics. If land use change increases the spatial aggregation of a population (e.g. Šálek et al., 2015; Serneels & Lambin, 2001), we expect the population will be more likely to crash in response to a regime of spatially restricted ECEs because more spatially aggregated populations experience more severe disturbances. To the extent that management actions like the implementation of habitat corridors or protected areas increase the size of a population's range (i.e. decrease the spatial aggregation of a population) (Ramiadantsoa et al., 2015; Thomas et al., 2012), we expect that populations will be less likely to crash in response to extreme climate events.

Our results give insight into how spatial aggregation may mediate the effects of changes in the frequency, severity, and spatial extent of ECEs. In general, an increase in severity (μ) of disturbances on the landscape will increase the size of kick experienced by the population (κ). A decrease in the time interval between disturbances on the landscape (ν) will decrease the time interval between kicks experienced by the population (τ). These change have the potential to push a population across the resilience threshold and lead the population to collapse regardless of population spatial distribution. However, population spatial aggregation determines how close the disturbance regime was to the resilience threshold before the change and thus how large of a change in severity and

frequency of kicks is required to cause the population to crash. How a change in spatial extent of disturbance (D)affects populations depends on population spatial aggregation. At the extremes, if the population is spread out across the entire landscape, a change in spatial extent of disturbance only affects the size of kicks (κ) and if the population occupies only one site, a change in spatial extent of disturbance only affects the frequency of kicks (τ). One resulting difference is that when the kicks are sufficiently large ($\kappa > 1 - A/K$), a change in spatial extent of disturbance cannot move the kick regime across the resilience threshold for maximally aggregated populations but can for maximally spread-out populations.

Demonstrating how population spatial aggregation mediates the probability of population persistence in response to ECEs helps us think about conservation of animal populations in the face of climate change in two ways. First, it leads us to predict that highly aggregated populations are most likely to be extirpated by changes in regimes of ECEs, improving our ability to target management action. Second, it helps us ideate conservation solutions. In alignment with previous modeling work finding that highly spatially aggregated populations are vulnerable to spatially autocorrelated disturbance (Kallimanis et al., 2005), our results indicate that preserving or creating habitat that allows for populations to be more spread out across the landscape could be beneficial (Ovaskainen et al., 2021). This may offer support for the 'several small' side of the 'single large or several small' (SLOSS) debate (reviewed in Fahrig, 2020).

Future Directions

It would be valuable to relax our assumption that ECEs and occupied habitat patches occur randomly in space because the spatial patterning of ECEs and habitat occupancy that we observe empirically may affect severity and frequency of disturbances a population experiences. We modeled both the population and disturbance as being located randomly to isolate the effects of population spatial aggregation and spatial extent of disturbance. This provided an analytically tractable way to incorporate space into disturbance models. However, we do not consider habitat selection or spatial patterns in

where disturbances occur. Our results would not change if one of these things occurred randomly in the environment and the other occurred non-randomly. However, if organisms select habitat patches that are less likely to be affected by disturbance (or vice versa) or if the spatial pattern of both patch occupancy and disturbance is set (e.g. all occupied patches are adjacent to each other) the severity and frequency of disturbances experienced by the population would be different relative to the predictions of this paper.

There are several reasons why migrants might differ from non-migrants in their responses to extreme climate events that weren't explored in this paper. We describe a few in the following paragraphs. Future work exploring how other differences between migrants and non-migrants mediate population response to ECEs could evaluate the relative importance of these mechanisms for determining population resilience to ECEs or determine how these mechanisms interact with one another (i.e. use an 'instigator' framing, sensu (Shaw et al., 2024)). This would increase our understanding of how migration mediates population response to climate change and lead to general insights about factors that mediate population response to disturbance.

There may be non-spatial differences in how extreme events affect populations that differ in movement strategy. For example, migrants might be less tolerant of extreme climate conditions than non-migrants (i.e. rates of mortality in response to the same climate conditions may be different (Newton, 2007)). A difference in tolerance could be explored in our model by exploring differences in the value of μ. The impact of extreme events may be especially variable throughout the annual cycle for migrants (e.g. the effect of an extreme event is especially severe during the migratory period (e.g. Overton et al., 2022)). Variability in seasonality of extreme event impact could be implemented in our model by implementing different values of μ for different seasons. Finally, migratory and non-migratory populations may differ in their rate of recovery following disturbance due to differences in fecundity and mortality rates (i.e. pace of life) (Soriano-Redondo et al., 2020), affecting the rate of recovery from a disturbance. This could be implemented in our model by varying the population growth rate in equation 2.1.

There may also be differences in the effects of extreme climate events that are more explicitly movement related. For example, differences in rates of dispersal between

habitat patches may affect response to disturbance at the metapopulation scale. In basic metapopulation theory, the equilibrium proportion of patches occupied is $1 - e/c$ where e is patch level extinction and c is the colonization rate. Thus, a higher colonization rate means that the metapopulation can persist for higher patch extinction rates. On the other hand, there are cases for which higher colonization rates can increase metapopulation extinction risk and local extinctions can decrease metapopulation extinction risk due to the effects of spatial synchrony (Fox et al., 2017). Further, changes in rates of dispersal between patches may evolve in response to changes in rates of local disturbance (Ronce et al., 2000). Therefore, colonization of extirpated patches may be important to consider when predicting population response to disturbance regimes. A metapopulation approach would allow us to consider when dispersal between patches increases resilience to extreme climate events. We predict that if disturbance repeatedly affects the same habitat patches, high dispersal rates may decrease meta-population level resilience because these patches could represent ecological sinks. If disturbance occurs more randomly throughout the environment, high dispersal rates may promote resilience. If animals are able to move out of affected areas, population crashes may be less likely than our model predicts. Therefore, if migratory and non-migratory populations differ in their ability to move in response to information about disturbances, there may be differences in resilience to disturbance regimes.

Summary

In summary, we illustrate that the spatial aggregation of populations mediates the severity and frequency of spatially restricted disturbances experienced by a population. The severity and frequency of experienced disturbances affect the probability that a population will crash in response to a given disturbance regime. This gives us a reason why migratory animal populations may be more vulnerable to extreme climate events. It also provides a mechanistic basis for understanding the interactions between land use change and pulsed disturbances.

References

Barton, P. S., Reboldi, A., Bonat, S., Mateo-Tomás, P., & Newsome, T. M. (2023). Climate-driven animal mass mortality events: Is there a role for scavengers? *Environmental Conservation*, *50*(1), 1–6. https://doi.org/10.1017/S0376892922000388

Fahrig, L. (2020). Why do several small patches hold more species than few large patches? *Global Ecology and Biogeography*, *29*(4), 615–628. https://doi.org/10.1111/geb.13059

Fey, S. B., Gibert, J. P., & Siepielski, A. M. (2019). The consequences of mass mortality events for the structure and dynamics of biological communities. *Oikos*, *128*(12), 1679–1690. https://doi.org/10.1111/oik.06515

Fey, S. B., Siepielski, A. M., Nusslé, S., Cervantes-Yoshida, K., Hwan, J. L., Huber, E. R., Fey, M. J., Catenazzi, A., & Carlson, S. M. (2015). Recent shifts in the occurrence, cause, and magnitude of animal mass mortality events. *Proceedings of the National Academy of Sciences of the United States of America*, *112*(4), 1083–1088. https://doi.org/10.1073/pnas.1414894112

Fox, J. W., Vasseur, D., Cotroneo, M., Guan, L., & Simon, F. (2017). Population extinctions can increase metapopulation persistence. *Nature Ecology & Evolution*, *1*(9), Article 9. https://doi.org/10.1038/s41559-017-0271-y

Fryxell, J., Greever, J., & Sinclair, A. (1988). Why are Migratory Ungulates So Abundant? *The American Naturalist*, *131*, 781. https://doi.org/10.1086/284822

Hansen, B. B., Gamelon, M., Albon, S. D., Lee, A. M., Stien, A., Irvine, R. J., Sæther, B.-E., Loe, L. E., Ropstad, E., Veiberg, V., & Grøtan, V. (2019). More frequent extreme climate events stabilize reindeer population dynamics. *Nature Communications*, *10*(1), 1616. https://doi.org/10.1038/s41467-019-09332-5

Johnson, D. H. (1980). The Comparison of Usage and Availability Measurements for Evaluating Resource Preference. *Ecology*, *61*(1), 65–71. https://doi.org/10.2307/1937156

Jolly, C. J., Dickman, C. R., Doherty, T. S., van Eeden, L. M., Geary, W. L., Legge, S. M., Woinarski, J. C. Z., & Nimmo, D. G. (2022). Animal mortality during fire. *Global Change Biology*, *28*(6), 2053–2065. https://doi.org/10.1111/gcb.16044

Jones, T., Parrish, J. K., Peterson, W. T., Bjorkstedt, E. P., Bond, N. A., Ballance, L. T., Bowes, V., Hipfner, J. M., Burgess, H. K., Dolliver, J. E., Lindquist, K., Lindsey, J., Nevins, H. M., Robertson, R. R., Roletto, J., Wilson, L., Joyce, T., & Harvey, J. (2018). Massive Mortality of a Planktivorous Seabird in Response to a Marine Heatwave. *Geophysical Research Letters*, *45*(7), 3193–3202. https://doi.org/10.1002/2017GL076164

Kallimanis, A. S., Kunin, W. E., Halley, J. M., & Sgardelis, S. P. (2005). Metapopulation Extinction Risk under Spatially Autocorrelated Disturbance. *Conservation Biology*, *19*(2), 534–546. https://doi.org/10.1111/j.1523-1739.2005.00418.x

Kauffman, M. J., Aikens, E. O., Esmaeili, S., Kaczensky, P., Middleton, A., Monteith, K. L., Morrison, T. A., Mueller, T., Sawyer, H., & Goheen, J. R. (2021). Causes, Consequences, and Conservation of Ungulate Migration. *Annual Review of Ecology, Evolution, and Systematics*, *52*(1). https://doi.org/10.1146/annurev-ecolsys-012021-011516

Lamberti, G. A., Levesque, N. M., Brueseke, M. A., Chaloner, D. T., & Benbow, M. E. (2020). Editorial: Animal Mass Mortalities in Aquatic Ecosystems: How Common and Influential? *Frontiers in Ecology and Evolution*, *8*. https://doi.org/10.3389/fevo.2020.602225

Law, R., Murrell, D. J., & Dieckmann, U. (2003). Population Growth in Space and Time: Spatial Logistic Equations. *Ecology*, *84*(1), 252–262. https://doi.org/10.1890/0012-9658(2003)084[0252:PGISAT]2.0.CO;2

Louzao, M., Gallagher, R., García-Barón, I., Chust, G., Intxausti, I., Albisu, J., Brereton, T., & Fontán, A. (2019). Threshold responses in bird mortality driven by extreme wind events. *Ecological Indicators*, *99*, 183–192. https://doi.org/10.1016/j.ecolind.2018.12.030

Meyer, K., Hoyer-Leitzel, A., Iams, S., Klasky, I., Lee, V., Ligtenberg, S., Bussmann, E., & Zeeman, M. L. (2018). Quantifying resilience to recurrent ecosystem

disturbances using flow–kick dynamics. *Nature Sustainability*, *1*(11). https://doi.org/10.1038/s41893-018-0168-z

Newton, I. (2007). Weather-related mass-mortality events in migrants. *Ibis*, *149*(3), 453–467. https://doi.org/10.1111/j.1474-919X.2007.00704.x

Ovaskainen, O., Somervuo, P., & Finkelshtein, D. (2021). Mathematical and simulation methods for deriving extinction thresholds in spatial and stochastic models of interacting agents. *Methods in Ecology and Evolution*, *12*(1), 162–169. https://doi.org/10.1111/2041-210X.13498

Overton, C. T., Lorenz, A. A., James, E. P., Ahmadov, R., Eadie, J. M., Mcduie, F., Petrie, M. J., Nicolai, C. A., Weaver, M. L., Skalos, D. A., Skalos, S. M., Mott, A. L., Mackell, D. A., Kennedy, A., Matchett, E. L., & Casazza, M. L. (2022). Megafires and thick smoke portend big problems for migratory birds. *Ecology*, *103*(1). https://doi.org/10.1002/ecy.3552

Parrish, J. K., & Edelstein-Keshet, L. (1999). Complexity, Pattern, and Evolutionary Trade-Offs in Animal Aggregation. *Science*, *284*(5411), 99–101. https://doi.org/10.1126/science.284.5411.99

Piatt, J. F., Parrish, J. K., Renner, H. M., Schoen, S. K., Jones, T. T., Arimitsu, M. L., Kuletz, K. J., Bodenstein, B., García-Reyes, M., Duerr, R. S., Corcoran, R. M., Kaler, R. S. A., McChesney, G. J., Golightly, R. T., Coletti, H. A., Suryan, R. M., Burgess, H. K., Lindsey, J., Lindquist, K., … Sydeman, W. J. (2020). Extreme mortality and reproductive failure of common murres resulting from the northeast Pacific marine heatwave of 2014-2016. *PLOS ONE*, *15*(1), e0226087. https://doi.org/10.1371/journal.pone.0226087

Picardi, S., Abrahms, B. L., & Merkle, J. A. (2024). Scale at the interface of spatial and social ecology. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *379*(1912), 20220523. https://doi.org/10.1098/rstb.2022.0523

Ramiadantsoa, T., Ovaskainen, O., Rybicki, J., & Hanski, I. (2015). Large-Scale Habitat Corridors for Biodiversity Conservation: A Forest Corridor in Madagascar. *PLOS ONE*, *10*(7), e0132126. https://doi.org/10.1371/journal.pone.0132126

Roberts, S. D., Van Ruth, P. D., Wilkinson, C., Bastianello, S. S., & Bansemer, M. S. (2019). Marine Heatwave, Harmful Algae Blooms and an Extensive Fish Kill Event During 2013 in South Australia. *Frontiers in Marine Science*, *6*. https://doi.org/10.3389/fmars.2019.00610

Ronce, O., Perret, F., & Olivieri, I. (2000). Evolutionarily Stable Dispersal Rates Do Not Always Increase with Local Extinction Rates. *The American Naturalist*, *155*(4), 485–496. https://doi.org/10.1086/303341

Rubenstein, D. I., & Hack, M. A. (2013). Migration. In *Encyclopedia of Biodiversity* (pp. 309–320). Elsevier. https://doi.org/10.1016/B978-0-12-384719-5.00095-2

Šálek, M., Drahníková, L., & Tkadlec, E. (2015). Changes in home range sizes and population densities of carnivore species along the natural to urban habitat gradient. *Mammal Review*, *45*(1), 1–14. https://doi.org/10.1111/mam.12027

Serneels, S., & Lambin, E. F. (2001). Impact of land-use changes on the wildebeest migration in the northern part of the Serengeti–Mara ecosystem. *Journal of Biogeography*, *28*(3), 391–407. https://doi.org/10.1046/j.1365-2699.2001.00557.x

Shaw, A. K., Bisesi, A. T., Wojan, C., Kim, D., Torstenson, M., Naven Narayanan, Lutz, P., Ales, R., & Shao, C. *Proceedings of the Royal Society B: Biological Sciences*, *291*(2031), 20240803. https://doi.org/10.1098/rspb.2024.0803

Soriano-Redondo, A., Gutiérrez, J. S., Hodgson, D., & Bearhop, S. (2020). Migrant birds and mammals live faster than residents. *Nature Communications*, *11*(1). https://doi.org/10.1038/s41467-020-19256-0

Thomas, C. D., Gillingham, P. K., Bradbury, R. B., Roy, D. B., Anderson, B. J., Baxter, J. M., Bourn, N. A. D., Crick, H. Q. P., Findon, R. A., Fox, R., Hodgson, J. A., Holt, A. R., Morecroft, M. D., O'Hanlon, N. J., Oliver, T. H., Pearce-Higgins, J. W., Procter, D. A., Thomas, J. A., Walker, K. J., … Hill, J. K. (2012). Protected areas facilitate species' range expansions. *Proceedings of the National Academy of Sciences*, *109*(35), 14063–14068. https://doi.org/10.1073/pnas.1210251109

Wilcove, D. S., & Wikelski, M. (2008). Going, going, gone: Is animal migration disappearing? *PLoS Biology*, *6*(7). https://doi.org/10.1371/journal.pbio.0060188

Yang, D., Yang, A., Yang, J., Xu, R., & Qiu, H. (2021). Unprecedented Migratory Bird Die-Off: A Citizen-Based Analysis on the Spatiotemporal Patterns of Mass Mortality Events in the Western United States. *GeoHealth*, *5*(4), e2021GH000395. https://doi.org/10.1029/2021GH000395

Tables

Table 2.1: Parameter definitions and value(s) used.

Symbol	Definition [units]	Value/Range
$\boldsymbol{T}$	Total number of sites	100
$\boldsymbol{D}$	Number of sites affected by a disturbance	1-100
$\boldsymbol{H}$	Number of sites occupied by a population	1-100
$\boldsymbol{S}$	Number of occupied sites affected by a disturbance	Varies
$\boldsymbol{N}$	Population size	Varies
$\boldsymbol{K}$	Carrying capacity	100
$\boldsymbol{A}$	Allee threshold	20
T	Time interval between kicks	Varies
$\boldsymbol{\tau_S}$	Time interval between kicks that affect S occupied sites	Varies
$\boldsymbol{\tau_{S_i \leq S}}$	Time interval between kicks that affect at least S_i occupied sites	Varies
$\boldsymbol{\nu}$	Time interval at which disturbances occur on the landscape	1
K	Size of kick	Varies
$\boldsymbol{\kappa_S}$	Size of kick where S occupied sites are affected	Varies

M	proportion of the population in affected cells that is killed	1

Figures

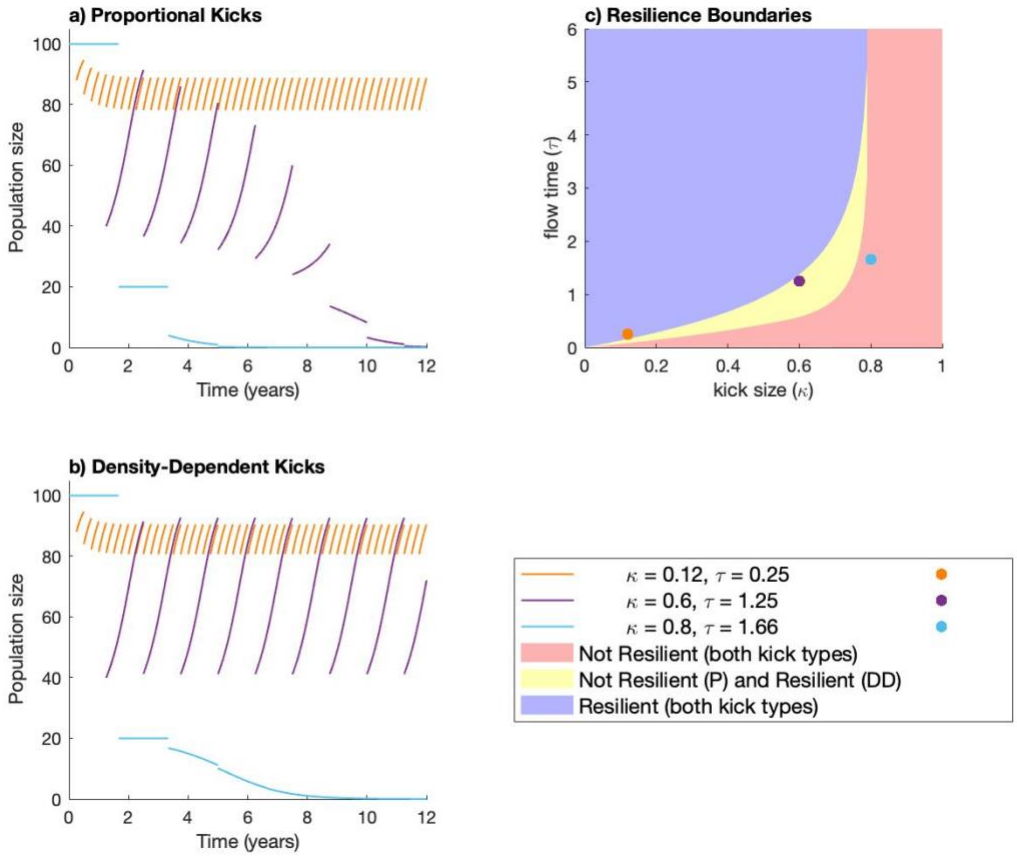

Figure 2.1: Example deterministic kick trajectories and resilience boundaries for proportional and density-dependent kicks. Three exemplar combinations of κ and τ values with equivalent average kick severity ($\kappa/\tau = 0.48$) are displayed: (κ, τ)=(0.12, 0.25), (0.6,1.25) and (0.8,1.66). (a) Kick trajectories for the three exemplar kick regimes when kicks are proportional. The population crashes for (κ, τ)=(0.6,1.25), and (0.8,1.66) and does not crash for (κ, τ)=(0.12, 0.25). (b) Kick trajectories for the three exemplar kick regimes when kicks are density-dependent. The population crashes for (κ, τ)= (0.8,1.66) and does not crash for (κ, τ)=(0.12, 0.25) and (0.6,1.25). (c) Resilience boundaries for proportional, and density-dependent kicks. The red region represents (κ, τ) values that lead to crashes for both kick types. The yellow region represents (κ, τ) values that lead to crashes for proportional but not density-dependent kicks. The blue region represents (κ, τ) values that do not lead to crashes for either kick type. The exemplar (κ, τ) are shown as points.

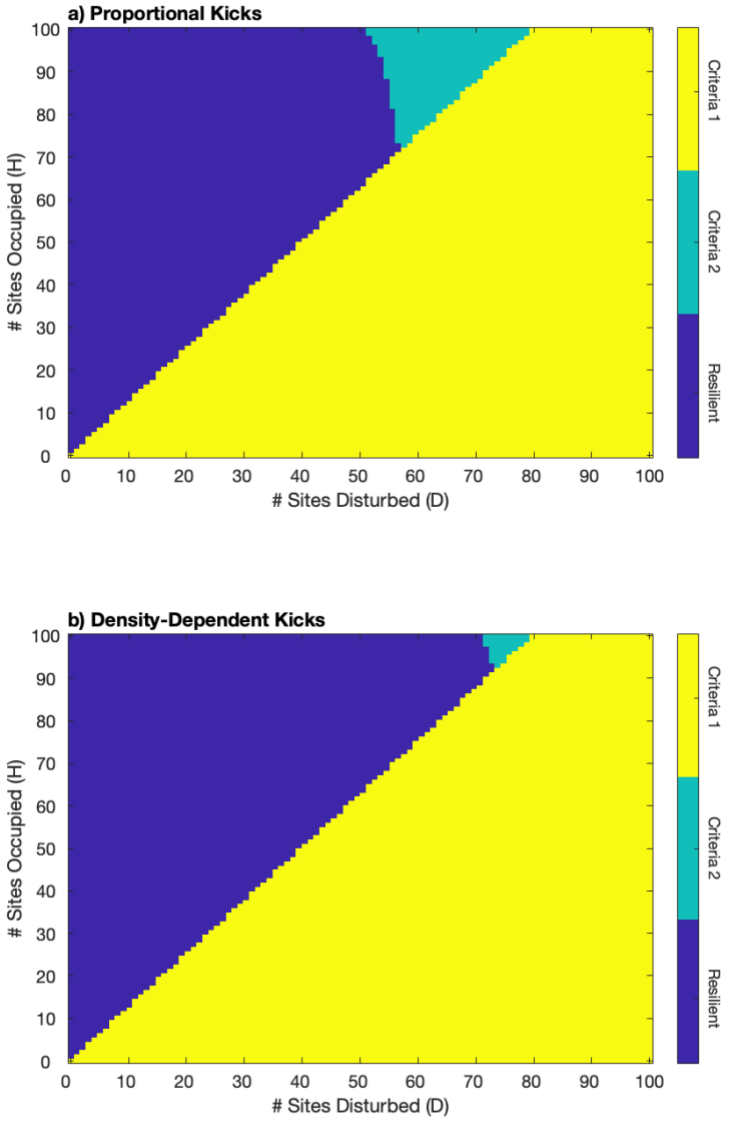

Figure 2.2: Analytical estimates of the likelihood that a population will crash in response to a kick regime in which τ=1, μ=1 and D and H are varied for (a) proportional and (b) density-dependent kicks. The yellow areas represent *D,H* values for which a single possible kick could bring the population below the Allee threshold (Criteria 1). The green areas represent *D,H* values for which there exists some possible $S = S_i$ for which (κ_{S_i},$\tau_{S_i \leq S}$) is in the 'not resilient' region of (κ, τ) parameter space (Criteria 2). The blue areas represent *D,H* values for which neither of these criteria are met and we therefore estimate the population to be resilient to the kick regime.

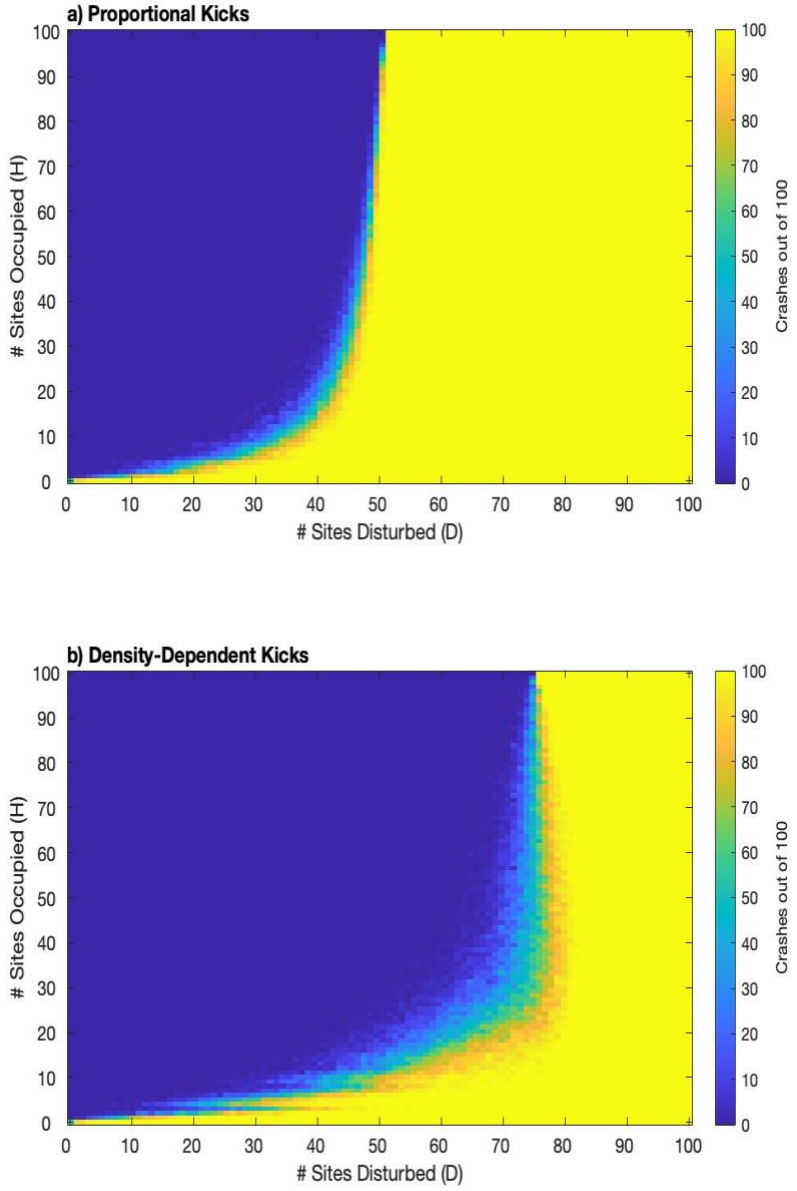

Figure 2.3 Heat map of how the number of population crashes out of 100 trials depends on spatial distribution of the population (*H*) and spatial extent of extreme event (*D*) for a) proportional and (b) density-dependent kicks when $\tau=1$ and $\mu=1$.

Chapter 3

Pathogen evolution following spillover from a resident to a migrant host population depends on interactions between host life history speed and cost of infection

Abstract

Changes to migration routes and phenology create novel contact patterns among hosts and pathogens. These novel contact patterns can lead to pathogens spilling over between resident and migrant populations. Predicting the consequences of such pathogen spillover events requires understanding how pathogen evolution depends on host movement behavior. Following spillover, pathogens may evolve changes in their transmission rate and virulence phenotypes because different strategies are favored by resident and migrant host populations. There is conflict in current theoretical predictions about what those differences might be. Some theory predicts lower pathogen virulence and transmission rates in migrant populations because migrants have lower tolerance to infection. Other theoretical work predicts higher pathogen virulence and transmission rates in migrants because migrants have more contacts with susceptible hosts. We aim to understand how differences in tolerance to infection and host pace of life act together to determine the direction of pathogen evolution following pathogen spillover from a resident to a migrant population. We constructed a spatially implicit model in which we investigate how pathogen strategy changes following the addition of a migrant population. We investigate how differences in tolerance to infection and pace of life between residents and migrants determine the effect of spillover on pathogen evolution and host population size. When the paces of life of the migrant and resident hosts are equal, larger costs of infection in

the migrants lead to lower pathogen transmission rate and virulence following spillover. When the tolerance to infection in migrant and resident populations are equal, faster migrant paces of life lead to increased transmission rate and virulence following spillover. However, the opposite can also occur: when the migrant population has lower tolerance to infection, faster migrant paces of life can lead to decreases in transmission rate and virulence. Predicting the outcomes of pathogen spillover requires accounting for both differences in tolerance to infection and pace of life between populations. It is also important to consider how movement patterns of populations affect host contact opportunities for pathogens. These results have implications for wildlife conservation, agriculture, and human health.

Introduction

Changes to animals' patterns of habitat use and movement induced by climate and land use change create novel contact patterns among hosts and pathogens (Altizer et al., 2013). Such novel contact patterns can lead to pathogen spillover events, with implications for the health of wildlife, livestock, and humans (Altizer et al., 2011). Changes to patterns of seasonal migration, a type of movement in which animals make an annual round trip between habitats (Dingle & Drake, 2007), have affected disease dynamics by altering contact patterns between resident and migrant populations (e.g. in monarchs, Satterfield et al., 2018; elk, Rayl et al., 2021; salmon, Ashander et al., 2012). Predicting the effect of pathogen spillover events between resident and migrant hosts requires an understanding of how these spillover events affect the evolution of pathogen transmission and virulence.

Before considering virulence in the context of migration and residency, we consider the selective pressures that act on pathogens generally. It is generally thought that pathogens experience a tradeoff between transmission rate (β) and virulence (μ) (Anderson & May, 1982; Cressler et al., 2016; Lipsitch & Moxon, 1997). These parameters are used in SIS (susceptible-infected-susceptible) models which are often used to study directly transmitted infectious diseases for which infection does not confer immunity (Hethcote, 2000) and can be used to describe environmentally transmitted diseases under some conditions (Benson et al., 2021). We define virulence as pathogen-induced host mortality and note that although virulence is often taken to be a pathogen property, it is an emergent property of a host-pathogen-environment system (Turner et al., 2021). We refer to the component of virulence attributed to the pathogen as the pathogen's virulence phenotype (Read, 1994). High transmission rates can be achieved by reproducing rapidly within a host but this may come with high host mortality costs (Acevedo et al., 2019). Long-term transmission opportunities from a host can be maintained by reproducing slower at lower cost to the host, but this may come with lower transmission rates (Acevedo et al., 2019). The total number new infections from a single host (R_0) depends on infection duration and the per capita rate at which infected individuals produce new infections (βS; the transmission rate multiplied by the number of susceptible individuals, which we call 'infection rate') (Nelson & May, 2017). The

tradeoff between transmission rate and virulence means that infection duration and infection rate are negatively correlated.

Properties of host populations affect which pathogen strategies lead to the highest pathogen fitness (Ewald, 1983; Restif et al., 2001). Tolerance to infection is one host property that determines host response to a given pathogen burden (McCarville & Ayres, 2018). If a host has lower tolerance to infection (a higher mortality rate for a given pathogen burden), this reduces the infection duration associated with a given transmission rate, which may select for a lower virulence and transmission rate pathogen phenotype (Altizer, 2001; Altizer et al., 2011, 2018). Host and population properties that increase the number of opportunities for contact with susceptible individuals may select for higher virulence pathogen phenotypes by increasing the infection rate associated with a given virulence phenotype (e.g. high levels of salmon density in aquaculture settings select for more virulent salmon lice phenotypes Ugelvik et al., 2017). Migrant and resident populations may differ in properties that select on pathogen virulence phenotypes. Migrants and residents may differ in their tolerance to infection. The energetic costs of migration may decrease tolerance to infection, leading to culling of hosts infected with highly virulent pathogens (Altizer et al., 2011, Table 3.2). Thus, for a given pathogen phenotype, virulence may be higher in migrants, which may select for lower virulence and transmission rate pathogen phenotypes. It may also be the case that migration selects for more infection tolerant hosts, which could select for more virulent pathogen phenotypes (Altizer et al., 2011, Table 3.2). Therefore, differences in tolerance between migrants and residents may lead to higher or lower virulence pathogen phenotypes evolving in migrant host populations.

Migrants and residents may also differ in their rates of contact with susceptible hosts and pathogen-independent mortality rates. Much of the empirical work on pathogen virulence in resident and migrant hosts (Table 3.2) has shown that pathogens infecting migrants have less virulent phenotypes. In many of these examples, residents have more contact opportunities with susceptible hosts since they are in agricultural settings with high host population densities (Krauss et al., 2010; Morgan et al., 2007). More frequent contact opportunities with susceptible hosts likely selects for more virulent phenotypes. It

may not be generally true that migrants have fewer contact opportunities than residents. Migrants often aggregate at high densities (Rubenstein & Hack, 2013), which could lead to high contract rates with susceptible individuals (Krauss et al., 2010). Migrants may also have faster paces of life (higher fecundity and mortality rates) than residents (Soriano-Redondo et al., 2020). Faster pace of life may select for more virulent pathogen phenotypes because higher fecundity rates (Ewald, 1983) and higher pathogen-independent mortality rates (e.g. due to faster pace of life) (Restif et al., 2001) can both select for more virulent pathogen phenotypes. Higher host fecundity rates select for more virulent pathogen phenotypes by increasing the rate at which susceptible hosts are added to the population, thus increasing the rate of contact with susceptible hosts (Ewald, 1983). Higher mortality from factors other than infection by the focal pathogen reduces infection duration, selecting for higher transmission rate pathogen phenotypes (Restif et al., 2001).

We investigate how pathogen virulence phenotype changes following spillover from residential hosts to migratory hosts using a proof-of-concept model (Servedio et al., 2014). We have compiled some examples of host-pathogen systems for which our model is relevant in Table 3.2, which include host populations of the same or different species. In particular, we consider how differences in tolerance to infection and pace of life between migrants and residents interact to determine whether spillover leads to an increase or decrease in pathogen virulence and transmission phenotype. Both pace of life and tolerance to infection can vary between species or between populations of the same species (de Roode et al., 2008; Mathot & Frankenhuis, 2018; Power & Mitchell, 2004). Past work on how the evolution of virulence depends on host migration has either considered differences in contact opportunities (Poulin & de Angeli Dutra, 2021) or differences in tolerance (Altizer, 2001; Osnas et al., 2015; Ugelvik et al., 2017) between migrants and residents. These mechanisms operate in opposite directions and we consider both. We explore a range of parameter values that allows us to discover the range of outcomes possible when these mechanisms operate together, improving our understanding of how spillover from a resident to a migrant population affects the evolution of pathogen transmission rate and virulence phenotypes.

Methods

The goal of our model is to investigate how host population properties related to migration strategy affect which pathogen strategies dominate at equilibrium. The foundation of our model is a spatially implicit SIS (susceptible - infected - susceptible) model in which there are multiple pathogen strategies that vary in transmission and virulence phenotypes and host populations vary in their pace of life and tolerance to infection (see Table 3.1 for model parameters and variables). Our choices to model pathogen dynamics as density dependent and explore differences in tolerance between migratory and non-migratory hosts are related to relevant empirical case studies described in Table 3.2. We use a numerically simulated adaptive dynamics approach (Best et al., 2017; Shaw et al., 2019) in order to identify how the dominant pathogen strategy depends on host traits. We begin by asking how the pathogen strategy that dominates at equilibrium depends on host pace of life and tolerance to infection in a single population in order to propose a source for the differences in pathogen strategy between migratory and non-migratory populations that are sometimes seen in empirical systems (Table 3.2). We then ask how pathogen strategy changes following spillover from a resident into a migrant population where the migrant and resident populations can differ in their pace of life, tolerance to infection, both, or neither. We focus on how the predictions about pathogen strategy evolution following spillover differ from what could be predicted in the single population case.

Pathogen Strategy

Pathogen strategy α (where α is an integer between 0 and 10) gives a pathogens transmission and virulence phenotype. Each pathogen strategy is defined by a transmission rate (β_α) that is a linear function

$$\beta_\alpha = 0.0005*\alpha \quad (3.1)$$

of α. This transmission rate is related to the virulence of the pathogen, taken to be the pathogen-induced rate of host mortality in some host population X. This is the difference between the mortality rates of hosts in population X infected with a pathogen of strategy

α ($\mu_{X,\alpha}$) and susceptible individuals in population X ($\mu_{X,S}$). The mortality rate of infected hosts is taken to be a quadratic function of transmission rate (Alizon & van Baalen, 2005)

$$\mu_{X,\alpha} = \left(\sqrt{\mu_{X,S}} + m\left(\frac{\beta_\alpha}{\beta_{10}}\right)\right)^2 \quad (3.2)$$

where m ($0 \leq m \leq 2$) and the mortality rate of susceptible individuals ($\mu_{X,S}$) are host properties. The parameter m (hereafter referred to as the host's tolerance to infection) governs how steep the relationship between virulence and transmission rate is (Figure A1) (McCarville & Ayres, 2018). High values of m mean low tolerance to infection and low values of m mean high tolerance to infection.

Single population model

Infection

In an SIS model of infection dynamics in some population X ($X = R$ or M where R stands for resident and M stands for migrant) , susceptible hosts become infected by pathogen strategy α at a rate proportional to the number of susceptible hosts in the population (S_X) and the number of hosts in the population infected with pathogen strategy α ($I_{X,\alpha}$), i.e. transmission is direct and density dependent as seen in many pathogens in Table 3.2. Infected hosts recover at a rate γ ($\gamma = 0.1$) and immediately become susceptible again. Hosts can become infected with any of the 11 pathogen strategies in our model. We ignore coinfection with multiple pathogen strategies for simplicity despite its potential importance (Rigaud et al., 2010).

Fecundity

Per-capita host fecundity rate is independent of infection status and all individuals are born into the susceptible class. The density-independent component of the per-capita fecundity rate is given by f_X ($0 \leq f_X \leq 3$) and the density-dependent per-capita fecundity rate in population X, $f(S_X, I_X)$, is given by

$$f(S_X, I_X) = f_X(1 - \delta(S_X + I_X)) \quad (3.3)$$

where δ ($\delta = 0.001$) is the strength of density dependence. Fecundity occurs continuously during the breeding season, which lasts for half of each simulation year.

Mutation

The role of mutation in this model to explore the strategy space and identify the strategies that dominate at equilibrium after competition with other strategies. As the pathogen is transmitted to a new host, it can mutate to each adjacent strategy with some small probability p ($p = 0.001$). That is, if a host is infected with strategy $\alpha = 4$, it will typically transmit the same strategy ($\alpha = 4$) with probability $1 - 2p$, but will sometimes transmit to a lower strategy ($\alpha = 3$) with probability p, or a higher strategy ($\alpha = 5$) with probability p. When $\alpha = 0$ or $\alpha = 10$, there is only one adjacent strategy. In these edge cases, the rate of mutation into the adjacent class remains p, but transmission will result in infection with the original pathogen strategy with probability $1 - p$ instead of $1 - 2p$. We show the differential equations for all infection classes in the single population model, but for simplicity, omit the edge cases in presenting the full two population model.

Model

Bringing together the infection dynamics with fecundity and pathogen strategy-specific mortality, the rate of change of the susceptible individuals in population *X* in the breeding season is given by

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10} \beta_\alpha S_X I_{X,\alpha} + \sum_{\alpha=0}^{10} \gamma I_{X,\alpha} + f_X \left(1 - \delta\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right)\right)\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right) - \mu_{X,S} S_X \quad (3.4a).$$

The rate of change of the individuals infected by strategy α in population *X* is given by

$$\frac{dI_{X,\alpha}}{dt} = (1-2p)\beta_\alpha S_X I_{X,\alpha} + p\beta_{\alpha-1} S_X I_{X,\alpha-1} + p\beta_{\alpha+1} S_X I_{X,\alpha+1} - \gamma I_{X,\alpha} - \mu_{X,\alpha} I_{X,\alpha}$$

(3.4b).

for $1 \leq \alpha \leq 9$ and by

$$\frac{dI_{X,0}}{dt} = (1-p)\beta_0 S_X I_{X,0} + p\beta_1 S_X I_{X,1} - \gamma I_{X,0} - \mu_{X,0} I_{X,0} \quad (3.4c)$$

when $\alpha = 0$ and

$$\frac{dI_{X,10}}{dt} = (1-p)\beta_{10}S_X I_{X,10} + p\beta_9 S_X I_{X,9} - \gamma I_{X,10} - \mu_{X,10} I_{X,10} \quad (3.4d)$$

when $\alpha = 10$. During the non-breeding season, the equations for the infected classes remain the same, but the equation for the susceptible class

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10} \beta_\alpha S_X I_{X,\alpha} + \sum_{\alpha=0}^{10} \gamma I_{X,\alpha} - \mu_{X,S} S_X \quad (3.4e)$$

does not include the fecundity term.

Two population model

In the two-population model, a resident population *R* and a migrant population *M* share an environment and can infect each other during the breeding season. During the non-breeding season, since the two populations do not share an environment, they cannot infect each other. This modelling choice is related to the fact that migrant and resident populations that share a pathogen often overlap for only part of the year (Table 3.2). Individuals are born into the same population as their parents and remain in the same population following infection and recovery. During the breeding season, the rates of change for the susceptible resident (X=R) and migrant populations (X=M) are given by

$$\frac{dS_X}{dt} = -\sum_{\alpha=0}^{10} \beta_\alpha S_X (I_{R,\alpha} + I_{M,\alpha}) + \sum_{\alpha=0}^{10} \gamma I_{X,\alpha} + f_X \left(1 - \delta\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right)\right)\left(S_X + \sum_{\alpha=0}^{10} I_{X,\alpha}\right) - \mu_{X,S} S_X \quad (3.5a).$$

The rate of change of the infected populations of the resident (X=R) and migrant (X=M) populations are given by

$$\frac{dI_{X,\alpha}}{dt} = (1-2p)\beta_\alpha S_X (I_{R,\alpha} + I_{M,\alpha}) + p\beta_{\alpha-1} S_X (I_{R,\alpha-1} + I_{M,\alpha-1}) + p\beta_{\alpha+1} S_X (I_{R,\alpha+1} + I_{M,\alpha+1}) - \gamma I_{X,\alpha} - \mu_{X,\alpha} I_{X,\alpha} \quad (3.5b)$$

during the breeding season. During the non-breeding season, the rates of change for the susceptible resident and migrant populations are given by equation 3.4e and the rate of change for the infected resident and migrant populations are given by equations 3.4b-d where $X = R$ or M.

Pace of Life

To isolate the effect of host pace of life on pathogen evolution independent of the effects on population size, we find pairs of mortality and fecundity rates that yield the same equilibrium population sizes N^* ($100 \leq N^* \leq 1000$) in the absence of infection. To do this, we consider the host population dynamics in the absence of infection, given by

$$\frac{dN}{dt} = f(1 - \delta N)N - \mu N \tag{3.6a}$$

in the breeding season and

$$\frac{dN}{dt} = -\mu N \tag{3.6b}$$

in the non-breeding season, where each season lasts for half of a year. Next, we look for the equilibrium population size (N^*), i.e., the population size at the beginning of the breeding season such that the growth during the breeding season is equal to the population decline during the non-breeding season. Solving (3.6a-b), we find that

$$N^* = \frac{(f-\mu)(1-e^{\mu-(f/2)})}{f\delta(e^{\mu/2}-e^{\mu-(f/2)})} \tag{3.6c}$$

To find different fecundity (f) and mortality (μ) rate pairs that yield a particular population size, we fix values of $N^*$and f and solve numerically for μ using vpasolve in MATLAB. We also run analyses in which we vary fecundity (f) and mortality (μ) rate separately in order to determine the effect of each rate separately and identify the effect of varying them together. While the pace of life syndrome idea can encompass many physiological and behavioral traits (Mathot & Frankenhuis, 2018), we restrict our attention here to the effects of fecundity and mortality rate.

Simulations

We begin by initializing a one-population model of the resident population with 100 individuals in the susceptible class, 10 individuals in the middle infection class ($\alpha = 5$), and no individuals in any other class. We run simulations until there is no class whose populations at the end of the non-breeding season changes in number by more than ε between two years ($\varepsilon = 0.001$). When this stability condition has been met, we calculate

which pathogen strategy dominates in the resident population alone. We also initialize a migrant population with N^*susceptible individuals and no infection. We use these stable populations as the starting condition for a two-population model. When stability is reached in the two-population simulation, we calculate what pathogen strategy dominates and how many individuals are in the migrant and resident populations. We explore simulations in which $N^* = 800$ as a representative example. When not taken as variables, we set the fecundity rate of the host population to $f_x = 1$ and the tolerance value to $m = 0.8$. These parameter choices were made because they led to intermediate levels of virulence at equilibrium in a single population, allowing virulence to increase or decrease following the addition of a second population. We also performed a sensitivity analysis to assess the generality of our findings (Appendix B).

Calculating pathogen strategy

We chose average pathogen strategy at equilibrium as our metric because when multiple pathogen strategies are present at equilibrium they are adjacent to one another. To calculate the average pathogen strategy in a single population X ($V(X)$), we take the sum of α multiplied by the proportion of infected individuals infected with pathogen strategy α over all values of α

$$V(X) = \sum_{\alpha=0}^{10} I_\alpha \alpha \Big/ \sum_{\alpha=0}^{10} I_\alpha \quad (3.7).$$

To find the change in mean pathogen strategy resulting from adding in the migrant population, we calculate the difference in average pathogen strategy between the resident population at equilibrium before and after the addition of the migrant population.

Results

The addition of a migrant population can lead to an increase, decrease or no change in the pathogen strategy (i.e., the pathogen transmission rate and virulence phenotype) depending on the tolerance to infection in the migrant population and the pace of life of

the migrants. We first explain how tolerance to infection and pace of life operate separately and together in a single host population to determine pathogen strategy. Next, we consider these mechanisms in the context of spillover in a two-population model with residents and migrants and show how changes in relative population size can generate results that cannot be predicted solely from the single-population model.

Pathogen strategy evolution in a single host population

The pathogen strategy (α =0,…,10) that evolves decreases monotonically as the host's tolerance to infection decreases (as m increases) (i.e. the transmission and virulence phenotypes of pathogens are lower for lower host tolerance to infection) (Figure 3.1a). The pathogen strategy that evolves increases monotonically with the pace of life of the host population (i.e. the transmission and virulence phenotypes of pathogens are higher for faster host paces of life) (Figure 3.1b). When fecundity and mortality rates are considered separately, the pathogen strategy that evolves increases monotonically with mortality rate (Figure B2) and generally does not change as a function of fecundity rate (Figure B4). When taken together, the effects of the host's tolerance to infection and pace of life are additive (Figure 3.1c).

Change in pathogen strategy following spillover from a resident into a migrant population

We consider what happens when migrants and residents (1) differ only in their tolerance to infection, (2) differ only in their paces of life and (3) differ in both tolerance to infection and pace of life.

We begin by considering the case where migrants differ from residents in their tolerance to infection but have the same pace of life (f_M =1 and m_M varies) (Figure 3.2). Following spillover into a migrant population, one might expect that the pathogen strategy would shift towards whatever strategy is favored in that migrant population. Indeed, the qualitative pattern of the change in virulence phenotype follows the pattern of

the single population simulations (Figure 3.1a). There is a monotonic decrease in pathogen strategy as migrant tolerance to infection decreases. The pathogen strategy increases when the migrant's tolerance to infection is higher than the residents and decreases when the migrant's tolerance to infection is lower (Figure 3.2a). Pathogen strategy changes more when the migrant has a higher tolerance to infection than residents than when the migrant has a lower tolerance to infection because of the effect of spillover on population sizes.

As the migrant's tolerance to infection decreases, the size of the migrant population decreases and the size of the resident population increases (Figure 3.2b). When the migrant's tolerance to infection is extremely high, the resident population is extirpated due to the increase in pathogen virulence and transmission phenotypes. In these cases, the pathogen strategy shifts all the way to what is optimal in the migrant population alone. When the migrant's tolerance to infection is lower than the resident's, the migrants make up a smaller proportion of the total number of hosts than the residents and the pathogen strategy does not move as far toward what is optimal in the migrant population. When $m_M = m_R$, the migrants and residents have the same population size because they are both equally affected by the pathogens. Although in this case there is almost no change in pathogen strategy (Figure 3.2a), we see a decrease in resident population size because infection is density-dependent and the addition of the migrant population increases host density for half of the year.

We now consider the case in which migrants and residents differ in their pace of life, but have the same tolerance to infection (m_M = 0.8 and f_M varies)(Figure 3.3). As would be predicted from the single population case (Figure 3.1b), we see that spillover into a migrant with a slower pace of life leads to a decrease in pathogen strategy and spillover into a migrant population with a faster pace of life leads to an increase in pathogen strategy. When we consider mortality rate and fecundity rate separately, as would be expected from the single population case (Figures B2 and B4), spillover into a migrant population with a lower mortality rate leads to a decrease in pathogen strategy (Figure B3a), spillover into a migrant population with a higher mortality rate leads to an increase in pathogen strategy (Figure B3a), and the fecundity rate of the migrant

population has next to no effect on pathogen strategy (Figure B5a). However, unlike in the single population case (Figure 3.1b) and unlike the case in which migrant mortality rate but not migrant fecundity rate is varied (Figure B3a), the relationship we see between migrant pace of life and change in pathogen strategy is not monotonic (Figure 3.4a). When migrants have a very slow pace of life, there is no change in pathogen strategy. Then, as migrant pace of life gets faster, there is a larger decrease in pathogen strategy until a certain point (f_M=0.6) past which we see a monotonic increase of pathogen strategy as migrant pace of life gets faster.

This non-monotonicity can be explained by considering how migrant and resident population sizes vary with migrant pace of life (Figure 3.3b) and especially how population sizes vary with migrant fecundity rate (Figure B5b). When migrants have very slow paces of life (low fecundity rates), their population is extirpated and they exert no selection pressure on pathogen strategy. This contrasts with the large migrant population sizes when migrant mortality rates are lower than residents and migrant and resident fecundity rates are equal (Figure B3b). Then, as migrant fecundity rates get faster, migrants make up a larger portion of the population, leading to larger influence on pathogen strategy. Since initially migrants have a lower average pathogen strategy than the residents, this leads to a larger decrease in pathogen strategy. However, as migrant pace of life continues to increase, the difference between what pathogen strategies are optimal in each population separately also gets smaller (Figure 3.1b), leading to a smaller decrease in pathogen strategy. The combination of these effects leads to a peak in the size of the decrease in pathogen strategy for an intermediate pace of life. When $f_R < f_M$, increases in f_M lead to both larger differences in optimal pathogen strategy between migrants and residents (Figure 3.1b) and increases in the size of the migrant population (Figure 3.3b). This differs from the case in which migrants have higher mortality rates than residents but equal fecundity rates, in which higher migrant mortality rates lead to smaller migrant population sizes (Figure B3b). Thus, we see a monotonic increase in change in pathogen strategy as pace of life gets faster when $f_R < f_M$ with increases in pathogen strategy that are larger in magnitude than if only migrant mortality rate was varied. Thus, although migrant mortality rates are largely responsible for the direction of

the change in pathogen strategy following spillover, migrant fecundity rates qualitatively affect the magnitude of the change through the effect of fecundity on population size. Finally, we consider the case in which migrants and residents differ both in pace of life and tolerance to infection (Figure 3.4). We find that the effects of these two factors on pathogen strategy are not simply additive as they were in the single population case (Figure 3.1c). Although a larger tolerance to infection always leads to a larger increase in pathogen strategy (lines with small m_M are typically above those with larger m_M in Figure 3.4a), a faster pace of life sometimes leads to an increase in pathogen strategy and sometimes leads to a decrease (some lines in Figure 3.4a increase, others decrease). The cases where a faster migrant pace of life leads to a larger decrease in pathogen strategy, even when $f_R < f_M$ are the cases where the migrant's tolerance to infection is lower.

As with the non-monotonicity in Figure 3.3a, this can be understood by considering how migrant pace of life influences the proportion of the total number of hosts that are migrants at equilibrium along with the difference in optimal pathogen strategy between residents and migrants (Figure 3.1c). When $m_R < m_M$, there are cases when the pathogen strategy that would evolve in the migrant is lower than that in residents even when the migrant's pace of life is faster (Figure 3.1c). In these cases, adding in the migrant exerts a downward pull on pathogen strategy. Meanwhile, the proportion of migrants at equilibrium increases monotonically with migrant fecundity rate meaning that the migrant population exerts a larger pull on pathogen strategy (Figure 3.4b). Thus, when the migrant population has a lower tolerance to infection, faster paces of life mean that more of the difference between the pathogen strategy that is optimal in the migrant and resident populations is realized as a change in pathogen virulence phenotype. This explains why the change in pathogen strategy continues to decrease even when $f_R < f_M$ in cases where $m_R < m_M$ (Figure 3.4a). When the tolerance to infection and pace of life mechanisms act together, faster pace of life doesn't always lead to higher pathogen strategy values, but instead can serve to amplify the direction of the effect caused by differences in tolerance to infection.

Discussion

Predicting the outcome of disease spillover events between migrant and resident populations is increasingly important as altered movement patterns lead to novel contact opportunities (Altizer et al., 2013). This requires understanding how pathogen virulence and transmission rate phenotypes evolve following spillover. There are many host-pathogen systems in which some hosts migrate and others do not (Table 3.2). However, there are very few systems for which we understand how pathogen virulence phenotypes differ between migratory and non-migratory host populations (Alexander, 2007; Altizer, 2001; Altizer et al., 2004; Ugelvik et al., 2017). There are even fewer systems for which we have information about the trajectory of pathogen evolution following a spillover event (Hawley et al., 2013). Finally, we found no empirical systems for which we know how pathogen strategy differs in the case where migratory and non-migratory host populations come into repeated contact versus the case where migratory and non-migratory host populations are separate. The lack of empirical data on a phenomenon of relevance in a broad range of host-pathogen systems necessitates the development of general theory.

Despite limited theoretical work about the evolution of virulence following spillover between resident and migrant host populations, we can form intuition from studies on pathogen evolution in other contexts. Some theory predicts that pathogens should evolve lower rates of transmission and lower virulence phenotypes in migrant populations if migratory hosts have lower tolerance to infection or fewer susceptible contacts (Osnas et al., 2015). We might also expect that pathogens should evolve phenotypes with higher rates of transmission and virulence in migratory hosts if migratory hosts have more contacts with susceptible hosts or higher tolerance (Ewald, 1983; Poulin & de Angeli Dutra, 2021). Differences in tolerance to infection and contact rate with susceptible individuals have not been considered simultaneously.

We began by considering how tolerance to infection and pace of life affected pathogen strategy evolution in a single population. In agreement with previous work, we found that faster pace of life increased the virulence and transmission rates of the pathogen phenotypes that evolved, lower tolerance to infection decreased the virulence

and transmission rates of the pathogen phenotypes that evolved and that the effects of these two factors were additive when combined. When we investigated spillover, pathogen effects on host population size led to results that could not be directly predicted from the single population case. When migrants have a faster pace of life, spillover into a migrant population leads to the evolution of pathogen phenotypes with higher rates of transmission and virulence. This leads to a decrease in resident population size. This is an example of pathogen-mediated apparent competition, in which spillover reduces the abundance of one of the host populations (Power & Mitchell, 2004). When migrants have lower tolerance to infection, spillover into a migrant population leads to the evolution of pathogen phenotypes with lower rates of transmission and virulence. When these differences are combined, with migrants having lower tolerance to infection and faster paces of life, the size of the decrease in pathogen transmission and virulence phenotypes can increase with faster paces of life, reversing the direction of the effect of migrant pace of life when values of tolerance to infection are equal. Thus, the direction of the effect of pace of life on the evolution of virulence following a spillover event depends on differences in tolerance to infection.

Although our study was primarily concerned with pathogen evolution, feedbacks with host population response to infection were important. Other ways in which coevolution between hosts and pathogens following spillover might affect pathogen evolution would require consideration of the phylogenetic distance between host populations. The only host traits we vary are pace of life and tolerance to infection and the only pathogen traits we vary are transmission rate and virulence phenotype. Neither the evolution of host resistance to infection nor pathogen host breadth are included. How these factors operate during spillover may depend on phylogenetic relatedness between host populations. The relationship between phylogenetic distance between host populations and resistance to infection is complicated because as phylogenetic distance increases, nonhost resistance increases and evolved resistance decreases (Antonovics et al., 2013). Adding considerations of resistance and host breadth as a function of phylogenetic distance between hosts to our model would add valuable nuance.

Our model is based on the commonly held and broadly theoretically and empirically supported assumption that there is a tradeoff between transmission rate and virulence (Acevedo et al., 2019; Alizon & van Baalen, 2005; Bonneaud et al., 2020; Cressler et al., 2016; de Roode et al., 2008; Lipsitch & Moxon, 1997; Turner et al., 2021). There are cases in which this tradeoff might not apply. For example, when pathogens infect a host that is a 'dead-end' in terms of transmission or when transmission occurs primarily through vectors, transmission might be disconnected from virulence (Farrell & Davies, 2019). Predicting the outcome of spillover in these cases would require a different model.

Future theoretical work should consider whether different ways to implement virulence and contact rate with susceptible individuals yield qualitatively different predictions. We considered virulence only as increased host mortality and considered differences in pace of life as drivers of differences in contact rates with susceptible hosts. Future spatially explicit models could consider mechanisms explicitly related to movement. For example, sub-lethal costs to movement ability might drive pathogen evolution differently than mortality costs. Manipulating contact rate through host movement or aggregation may yield different predictions than manipulating pace of life. Modelling movement mechanisms could increase our understanding of the effects of host migration on pathogen evolution separate from the associations between migration and pace of life and tolerance to infection.

As migration patterns change, it is increasingly important to understand and predict the trajectory of spillover events between migrant and resident populations, including the evolutionary trajectory of pathogen strategy. Predicting the trajectories of spillover events involving migratory species is important to maintaining wildlife, livestock, and human health in a changing world (Altizer et al., 2011).

References

Acevedo, M. A., Dillemuth, F. P., Flick, A. J., Faldyn, M. J., & Elderd, B. D. (2019). Virulence-driven trade-offs in disease transmission: A meta-analysis*. *Evolution*, *73*(4). https://doi.org/10.1111/evo.13692

Alexander, D. J. (2007). An overview of the epidemiology of avian influenza. *Vaccine*, *25*(30), 5637–5644. https://doi.org/10.1016/j.vaccine.2006.10.051

Alizon, S., & van Baalen, M. (2005). Emergence of a convex trade-off between transmission and virulence. *The American Naturalist*, *165*(6). https://doi.org/10.1086/430053

Altizer, S. (2001). Migratory behaviour and host-parasite co-evolution in natural populations of monarch butterflies infected with a protozoan parasite. *Evol. Ecol. Res.*, *3*.

Altizer, S., Bartel, R., & Han, B. A. (2011). Animal migration and infectious disease risk. *Science*. https://doi.org/10.1126/science.1194694

Altizer, S., Becker, D. J., Epstein, J. H., Forbes, K. M., Gillespie, T. R., Hall, R. J., Hawley, D. M., Hernandez, S. M., Martin, L. B., Plowright, R. K., Satterfield, D. A., & Streicker, D. G. (2018). Food for contagion: Synthesis and future directions for studying host –parasite responses to resource shifts in anthropogenic environments. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1745). https://doi.org/10.1098/rstb.2017.0102

Altizer, S., Hochachka, W. M., & Dhondt, A. A. (2004). Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American house finches. *Journal of Animal Ecology*, *73*(2), 309–322. https://doi.org/10.1111/j.0021-8790.2004.00807.x

Altizer, S., Ostfeld, R. S., Johnson, P. T. J., Kutz, S., & Harvell, C. D. (2013). Climate change and infectious diseases: From evidence to a predictive framework. *Science*. https://doi.org/10.1126/science.1239401

Anderson, R. M., & May, R. M. (1982). Coevolution of hosts and parasites. *Parasitology*, *85*(2), 411–426. https://doi.org/10.1017/S0031182000055360

Antonovics, J., Boots, M., Ebert, D., Koskella, B., Poss, M., & Sadd, B. M. (2013). The Origin of Specificity by Means of Natural Selection: Evolved and Nonhost Resistance in Host–Pathogen Interactions. *Evolution*, *67*(1), 1–9. https://doi.org/10.1111/j.1558-5646.2012.01793.x

Ashander, J., Krkošek, M., & Lewis, M. A. (2012). Aquaculture-induced changes to dynamics of a migratory host and specialist parasite: A case study of pink salmon and sea lice. *Theoretical Ecology*, *5*(2), 231–252. https://doi.org/10.1007/s12080-011-0122-4

Benson, L., Davidson, R. S., Green, D. M., Hoyle, A., Hutchings, M. R., & Marion, G. (2021). When and why direct transmission models can be used for environmentally persistent pathogens. *PLOS Computational Biology*, *17*(12), e1009652. https://doi.org/10.1371/journal.pcbi.1009652

Best, A., Ashby, B., White, A., Bowers, R., Buckling, A., Koskella, B., & Boots, M. (2017). Host-parasite fluctuating selection in the absence of specificity. *Proceedings. Biological Sciences*, *284*(1866), 20171615. https://doi.org/10.1098/rspb.2017.1615

Bonneaud, C., Tardy, L., Hill, G. E., McGraw, K. J., Wilson, A. J., & Giraudeau, M. (2020). Experimental evidence for stabilizing selection on virulence in a bacterial pathogen. *Evolution Letters*, *4*(6), 491–501. https://doi.org/10.1002/evl3.203

Carbó-Ramírez, P., & Zuria, I. (2015). Immune Condition and Blood Parasites in Three Sparrow Species with Different Migratory Status in Central Mexico. *Avian Biology Research*, *8*(3), 167–174. https://doi.org/10.3184/175815515X14371521830098

Cressler, C. E., McLEOD, D. V., Rozins, C., Van Den Hoogen, J., & Day, T. (2016). The adaptive evolution of virulence: A review of theoretical predictions and empirical tests. *Parasitology*, *143*(7), 915–930. https://doi.org/10.1017/S003118201500092X

de Roode, J. C., Yates, A. J., & Altizer, S. (2008). Virulence-transmission trade-offs and population divergence in virulence in a naturally occurring butterfly parasite.

Proceedings of the National Academy of Sciences, *105*(21), 7489–7494. https://doi.org/10.1073/pnas.0710909105

Dingle, H., & Drake, V. A. (2007). What Is Migration? *BioScience*, *57*(2), 113–121. https://doi.org/10.1641/B570206

Endo, A., & Nishiura, H. (2018). The Role of Migration in Maintaining the Transmission of Avian Influenza in Waterfowl: A Multisite Multispecies Transmission Model along East Asian-Australian Flyway. *Canadian Journal of Infectious Diseases and Medical Microbiology*, *2018*, 1–7. https://doi.org/10.1155/2018/3420535

Ewald, P. W. (1983). Host- parasite relations, vectors, and the evolution of disease severity. *Annual Review of Ecology and Systematics. Vol. 14*. https://doi.org/10.1146/annurev.es.14.110183.002341

Farrell, M. J., & Davies, T. J. (2019). Disease mortality in domesticated animals is predicted by host evolutionary relationships. *Proceedings of the National Academy of Sciences*, *116*(16), 7911–7915. https://doi.org/10.1073/pnas.1817323116

Hawley, D. M., Osnas, E. E., Dobson, A. P., Hochachka, W. M., Ley, D. H., & Dhondt, A. A. (2013). Parallel Patterns of Increased Virulence in a Recently Emerged Wildlife Pathogen. *PLOS Biology*, *11*(5), e1001570. https://doi.org/10.1371/journal.pbio.1001570

Hethcote, H. W. (2000). The Mathematics of Infectious Diseases. *SIAM Review*, *42*(4), 599–653. https://doi.org/10.1137/S0036144500371907

Hurtado, P. (2008). The Potential Impact of Disease on the Migratory Structure of a Partially Migratory Passerine Population. *Bulletin of Mathematical Biology*, *70*(8), 2264–2282. https://doi.org/10.1007/s11538-008-9345-y

Krauss, S., Stallknecht, D. E., Negovetich, N. J., Niles, L. J., Webby, R. J., & Webster, R. G. (2010). Coincident ruddy turnstone migration and horseshoe crab spawning creates an ecological 'hot spot' for influenza viruses. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1699), 3373–3379. https://doi.org/10.1098/rspb.2010.1090

Krkošek, M., Lewis, M. A., & Volpe, J. P. (2005). Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proceedings of the Royal Society B: Biological Sciences*, *272*(1564), 689–696. https://doi.org/10.1098/rspb.2004.3027

Lipsitch, M., & Moxon, E. R. (1997). Virulence and transmissibility of pathogens: What is the relationship? *Trends in Microbiology*, *5*(1). https://doi.org/10.1016/S0966-842X(97)81772-6

Maina, L. G. M., Maingi, N., Ng'ang'a, C. J., Waruiru, R. M., & Gakuya, F. (2022). Diversity, prevalence, and intensity of gastrointestinal helminth infections in migratory, resident, and sedentary plains zebras (Equus quagga) in Masai Mara National Reserve and Lake Nakuru National Park, Kenya. *Veterinary Parasitology: Regional Studies and Reports*, *33*, 100750. https://doi.org/10.1016/j.vprsr.2022.100750

Mathot, K. J., & Frankenhuis, W. E. (2018). Models of pace-of-life syndromes (POLS): A systematic review. *Behavioral Ecology and Sociobiology*, *72*(3), 41. https://doi.org/10.1007/s00265-018-2459-9

McCarville, J., & Ayres, J. (2018). Disease tolerance: Concept and mechanisms. *Current Opinion in Immunology*, *50*, 88–93. https://doi.org/10.1016/j.coi.2017.12.003

Morgan, E. R., Lundervold, M., Medley, G. F., Shaikenov, B. S., Torgerson, P. R., & Milner-Gulland, E. J. (2006). Assessing risks of disease transmission between wildlife and livestock: The Saiga antelope as a case study. *Biological Conservation*, *131*(2), 244–254. https://doi.org/10.1016/j.biocon.2006.04.012

Morgan, E. R., Medley, G. F., Torgerson, P. R., Shaikenov, B. S., & Milner-Gulland, E. J. (2007). Parasite transmission in a migratory multiple host system. *Ecological Modelling*, *200*(3), 511–520. https://doi.org/10.1016/j.ecolmodel.2006.09.002

Nelson, P. G., & May, G. (2017). Coevolution between mutualists and parasites in symbiotic communities may lead to the evolution of lower virulence. *American Naturalist*, *190*(6). https://doi.org/10.1086/694334

Osnas, E. E., Hurtado, P. J., & Osnas, E. E. (2015). Evolution of pathogen virulence across space during an epidemic. *American Naturalist*, *185*(3). https://doi.org/10.1086/679734

Poulin, R., & de Angeli Dutra, D. (2021). Animal migrations and parasitism: Reciprocal effects within a unified framework. *Biological Reviews*. https://doi.org/10.1111/brv.12704

Power, A. G., & Mitchell, C. E. (2004). Pathogen Spillover in Disease Epidemics. *The American Naturalist*, *164*(S5), S79–S89. https://doi.org/10.1086/424610

Rayl, N. D., Merkle, J. A., Proffitt, K. M., Almberg, E. S., Jones, J. D., Gude, J. A., & Cross, P. C. (2021). Elk migration influences the risk of disease spillover in the Greater Yellowstone Ecosystem. *Journal of Animal Ecology*, *90*(5), 1264–1275. https://doi.org/10.1111/1365-2656.13452

Read, A. F. (1994). The evolution of virulence. *Trends in Microbiology*, *2*(3), 73–76. https://doi.org/10.1016/0966-842X(94)90537-1

Restif, O., Hochberg, M. E., & Koella, J. C. (2001). Virulence and age at reproduction: New insights into host–parasite coevolution. *Journal of Evolutionary Biology*, *14*(6), 967–979. https://doi.org/10.1046/j.1420-9101.2001.00355.x

Rigaud, T., Perrot-Minnot, M.-J., & Brown, M. J. F. (2010). Parasite and host assemblages: Embracing the reality will improve our knowledge of parasite transmission and virulence. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1701), 3693–3702. https://doi.org/10.1098/rspb.2010.1163

Rubenstein, D. I., & Hack, M. A. (2013). Migration. In *Encyclopedia of Biodiversity* (pp. 309–320). Elsevier. https://doi.org/10.1016/B978-0-12-384719-5.00095-2

Satterfield, D. A., Maerz, J. C., Hunter, M. D., Flockhart, D. T. T., Hobson, K. A., Norris, D. R., Streit, H., de Roode, J. C., & Altizer, S. (2018). Migratory monarchs that encounter resident monarchs show life-history differences and higher rates of parasite infection. *Ecology Letters*, *21*(11). https://doi.org/10.1111/ele.13144

Servedio, M. R., Brandvain, Y., Dhole, S., Fitzpatrick, C. L., Goldberg, E. E., Stern, C. A., Cleve, J. V., & Yeh, D. J. (2014). Not Just a Theory—The Utility of Mathematical Models in Evolutionary Biology. *PLOS Biology*, *12*(12), e1002017. https://doi.org/10.1371/journal.pbio.1002017

Shaw, A. K., Craft, M. E., Zuk, M., & Binning, S. A. (2019). Host migration strategy is shaped by forms of parasite transmission and infection cost. *Journal of Animal Ecology*, *88*(10). https://doi.org/10.1111/1365-2656.13050

Smith, J., Smith, N., Yu, L., Paton, I. R., Gutowska, M. W., Forrest, H. L., Danner, A. F., Seiler, J. P., Digard, P., Webster, R. G., & Burt, D. W. (2015). A comparative analysis of host responses to avian influenza infection in ducks and chickens highlights a role for the interferon-induced transmembrane proteins in viral resistance. *BMC Genomics*, *16*(1), Article 1. https://doi.org/10.1186/s12864-015-1778-8

Soriano-Redondo, A., Gutiérrez, J. S., Hodgson, D., & Bearhop, S. (2020). Migrant birds and mammals live faster than residents. *Nature Communications*, *11*(1). https://doi.org/10.1038/s41467-020-19256-0

Torstenson, M., & Shaw, A. K. (2024). Model code associated with: Pathogen evolution following

spillover from a resident to a migrant host population depends on interactions between host life

history speed and cost of infection. In Journal of Animal Ecology. Zenodo. https://doi.org/10.5281/zenodo.10723759

Turner, W. C., Kamath, P. L., Van Heerden, H., Huang, Y. H., Barandongo, Z. R., Bruce, S. A., & Kausrud, K. (2021). The roles of environmental variation and parasite survival in virulence-transmission relationships. *Royal Society Open Science*, *8*(6). https://doi.org/10.1098/rsos.210088

Ugelvik, M. S., Skorping, A., Moberg, O., & Mennerat, A. (2017). Evolution of virulence under intensive farming: Salmon lice increase skin lesions and reduce host growth in salmon farms. *Journal of Evolutionary Biology*, *30*(6), 1136–1142. https://doi.org/10.1111/jeb.13082

Tables

Table 3.1: Model symbols, definitions, and values (where applicable)

Symbol	Definition [units]	Value(s)
$\boldsymbol{\alpha}$	Pathogen strategy [unitless]	Integers between 0 and 10
$\boldsymbol{\beta_\alpha}$	Transmission rate for pathogen strategy α [individual^{-1} time^{-1}]	$0.0005 * \alpha$
$\boldsymbol{\gamma}$	Recovery rate [time^{-1}]	0.1
$\boldsymbol{m_X}$	Steepness of association between transmission rate and host mortality rate in population X (host tolerance to infection) [time$^{-1/2}$]	Varied (0-2)
$\boldsymbol{f_X}$	Density-independent component of the fecundity rate for individuals in population X [time^{-1}]	Varied (0.1-2)
$\boldsymbol{N_X}$	Number of individuals in population X [individuals]	Varies
$\boldsymbol{N^*}$	Number of individuals when the population is at equilibrium without infection [individuals]	Varied (100-1000)
$\boldsymbol{\mu_{X,S}}$	Mortality rate of susceptible individuals in population X [time^{-1}]	Varied based on f_X and N^* values
$\boldsymbol{\delta}$	Strength of density dependence [individuals^{-1}]	0.001
$\boldsymbol{\mu_{X,\alpha}}$	Mortality rate for individuals in population X infected with pathogen strategy α [time^{-1}]	$\left(\sqrt{\mu_{X,S}} + m\left(\frac{\beta_\alpha}{0.005}\right)\right)^2$
$\boldsymbol{p}$	Probability of mutation [unitless]	0.001
$\boldsymbol{\varepsilon}$	Stability cutoff [individuals]	0.001

$\boldsymbol{S_X}$	Number of susceptible individuals in population X [individuals]	Varies
$\boldsymbol{I_{X,\alpha}}$	Number of individuals in population X infected with pathogen transmission strategy α [individuals]	Varies
$\boldsymbol{f(S_X, I_X)}$	Density-dependent fecundity rate [individuals time^{-1}]	$f_X(1-\delta(S_X+I_X))$
$\boldsymbol{V(X)}$	Mean pathogen strategy value in population X [unitless]	Evolves

Table 3.2 Examples of systems in which migratory and non-migratory hosts share a pathogen/ parasite and transmission is density dependent

Host species	**Pathogen/ Parasite species**	**Transmission and recovery dynamics**	**Migration and resident dynamics**	**Virulence details- Differences in Pathogen Phenotype (if known)**	**Virulence details- Differences in Host tolerance/ resistance (if known)**	**Spillover details (if known)**	**References**
House finches	Bacteria (Mycoplasma gallisepticum)	Direct transmission and short-term environmental transmission. Transmission has been modeled using SIS and SIR (susceptible-infected-recovered) models.	In the eastern U.S. population, migrants and residents share breeding grounds, migrants travel south-west for the winter. The western U.S. population in not migratory.	Initial virulence of strains in western population lower than in eastern population. Virulence in both populations increased following spillover. The virulence in the two populations is now similar.	The costs of long distance migration may decrease resistance. Non-migrants may experience decreased tolerance in the winter due to cold.	Initial spillover into eastern population presumably from poultry. Spillover from eastern into western population in the 2000s.	(Altizer et al., 2004; Hawley et al., 2013; Hurtado, 2008)

Migratory waterfowl and poultry	Avian influenza viruses	Transmission details are uncertain. Has been modeled as SIS.	Domestic poultry are sedentary. Poultry on flyways can overlap with migrating waterfowl.	Lower virulence pathogen phenotypes in wild migratory birds than in poultry.	Ducks have high levels of tolerance. Vaccination can increase tolerance/resistance in poultry.	Initial transmission to poultry seems to be from wild waterfowl. Spillback from poultry to wild migratory birds has occurred.	(Alexander, 2007; Endo & Nishiura, 2018; Smith et al., 2015)
Saiga and domestic sheep	Foot and mouth disease virus	Direct Transmission. Has been modelled using SLIR (susceptible-latent-infected-recovered).	Saiga migrate and come into contact with sedentary sheep during part of their migration.	Unknown	Saiga have higher infection induced mortality than domestic ruminants.	Transmission from domestic ruminants to saiga is widely assumed. Anecdotal evidence of spillback from saiga to livestock.	(Morgan et al., 2006)
Atlantic salmon and pink salmon	Salmon lice	Direct transmission. Free-swimming stages are transmitted to	Farmed salmon are sedentary; wild salmon migrate between rivers for breeding and ocean for maturation and	Higher virulence pathogen phenotypes in farmed fish than wild ones.	Unknown	Both spillover (wild to farmed) and spillback (farmed to wild) occurs.	(Ashander et al., 2012; Krkošek et al., 2005; Ugelvik et al., 2017)

		fish without an intermediate host.	pass by farms en route.				
Monarch butterflies	Protozoan (Ophryocystis elektroscirrha)	Transmission is typically maternal but can be environmental. Infected individuals cannot recover, so best modeled as SI.	Eastern population migrates farther than western population. Resident population in Florida. Some overlap between migratory and non-migratory individuals.	Parasites from resident populations are more virulent than those from migratory ones.	Migrant hosts more resistant to infection. Migration may lead to culling of hosts infected with more virulent pathogen phenotypes.	Spillover from residents to migrants occurs when they share habitat	(Altizer, 2001; Altizer et al., 2011; Satterfield et al., 2018)
Zebra	Helminths	Environmental transmission.	Separate populations of resident, migrant, and sedentary zebra.	Unknown	Possible that migrant and sedentary zebra populations have lower resistance than the resident population.	Unknown	(Maina et al., 2022)

Sparrows	Nematodes	Environmental transmission.	Migrant and resident species that overlap during migration	Unknown	Residents had lower resistance than migrants.	Unknown	(Carbó-Ramírez & Zuria, 2015)

Figures

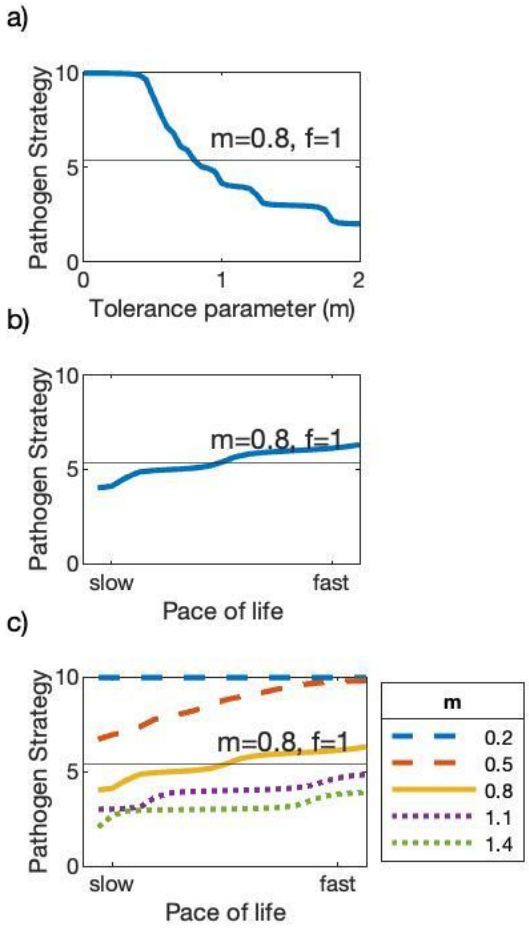

Figure 3.1 Pathogen Strategies in One Population. In a single population in which N^*=800 a) the pathogen strategy that evolves decreases monotonically as tolerance to infection decreases (m increases) (f =1), b) the pathogen strategy that evolves increases monotonically as fecundity (f) increases (m=0.8) and c) the pathogen strategy that evolves increases monotonically as fecundity (f) increases and decreases monotonically as tolerance to infection decreases (m increases) when both tolerance to infection and pace of life vary. The pathogen strategy that evolves in the resident population in the two population simulation (m=0.8 and f=1) is shown as a horizontal line in all panels for easy comparison with the pathogen strategy that evolves in the migrant population in the two population simulations.

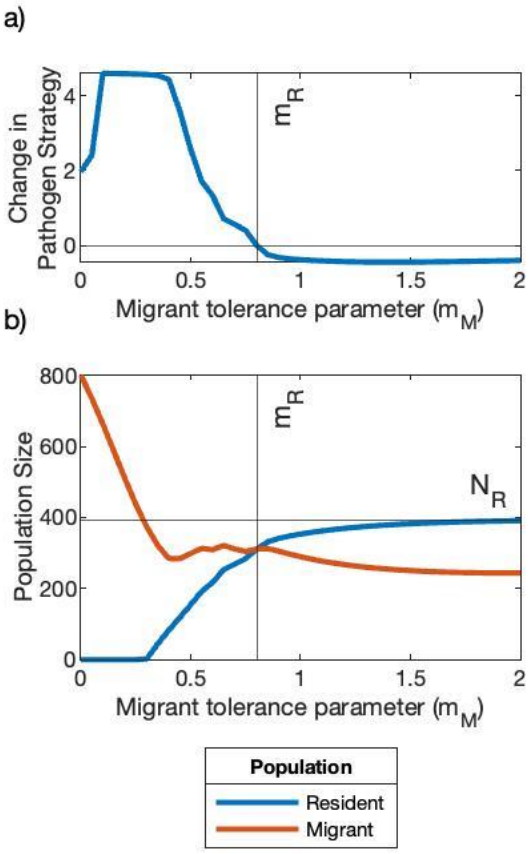

Figure 3.2 Migrant and Resident Populations Differ in Tolerance to Infection. Following spillover from a resident into a migrant population in which N^* =800 for both populations, $f_R = f_M = 1$ and m_R=0.8 a) the change in pathogen strategy generally decreases monotonically as migrant tolerance to infection decreases (m_M increases). When $m_M = m_R$ (m_R is shown as a vertical line for reference), there is almost no change in pathogen strategy. b) The size of the resident population increases as migrant tolerance to infection decreases (m_M increases) and size of the migrant population generally decreases as migrant tolerance to infection decreases (m_M increases). The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

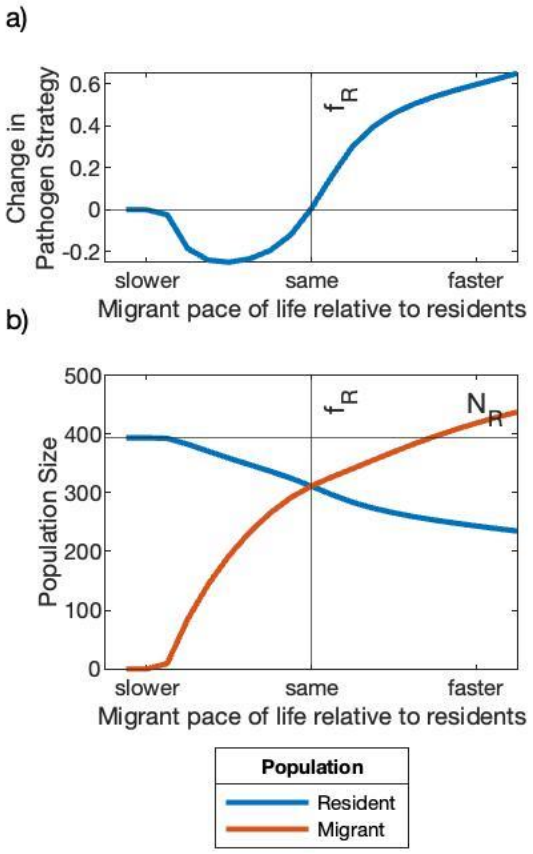

Figure 3.3 Migrant and resident populations differ in pace of life. Following spillover from a resident into a migrant population in which N ∗=800 for both populations, $m_R = m_M = 0.8$ and f_R=1 a) the change in pathogen strategy following spillover decreases and then increases as migrant fecundity rate (f_M) increases. When $f_M = f_R$ (f_R is shown as a vertical line for reference), there is almost no change in pathogen strategy. b) The size of the resident population following spillover decreases as migrant fecundity rate (f_M) increases and size of the migrant population generally increases as migrant fecundity rate (f_M) increases. The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

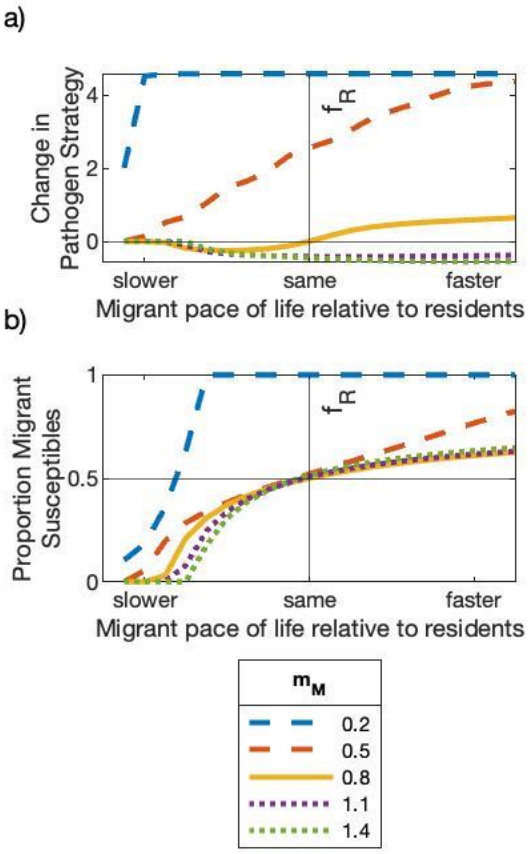

Figure 3.4 Migrant and resident populations differ in tolerance to infection and pace of life. Following spillover from a resident into a migrant population in which N^* =800 for both populations, m_R=0.8 and f_R=1 (with f_R shown as a vertical line for reference) a) the change in pathogen strategy following spillover decreases as migrant tolerance to infection decreases (m_M increases) and can either increase or decrease as migrant fecundity rate (f_M) increases. b) The proportion of susceptible individuals at equilibrium that are migrants typically increases as migrant fecundity rate (f_M) increases and as migrant tolerance to infection increases. A horizontal line is shown where migrants and residents make up an equal proportion of the susceptible population for reference.

Future Directions

Ecological theory development allows us to explore relationships between mechanisms and patterns (Shaw et al. 2024) and is therefore useful for developing an understanding of how animal migration mediates the consequences of a variety of global change factors. In this thesis, I explore three possible mechanism-pattern pairs. There are a number of ways to expand upon these projects to improve our understanding of how migration mediates the consequences of global change.

First, the models developed in this thesis could be extended or combined. For example, in Shao et al. 2025, we investigated how the predictions of Chapter 3 change with the addition of resistance to infection following recovery. We could also combine the models in Chapters 1 and 2 in order to understand the combined effects of phenological change and extreme climate events. Empirically, we have seen that phenological shifts that seem adaptive in light of warmer spring temperatures can lead to higher mortality due to exposure to extreme climate events (Shipley et al. 2020), and this phenomenon would be interesting to explore using theory.

Additionally, new models could be built to explore other ways in which migration might mediate the consequences of global change for populations, communities and ecosystems. For example, when focusing on migratory populations, models could be built to explore the extent to which migrants movement abilities confer resilience to global change or exploit new ecological opportunities (Ljungström et al. 2021, Tsujii et al. 2021, Stratmann et al. 2023). When considering consequences for communities and ecosystems, models could be built to understand the trophic and transport functions of migration in the context of global change (Bauer and Hoye 2014).

Finally, these models could be used to inform empirical research and conservation. Empirical studies could evaluate the extent to which the predictions and assumptions of the models presented in this thesis hold in the real world (Grainger et al. 2021). Conservation efforts could be targeted towards populations that the models in this thesis predict to be most vulnerable to environmental change and in some cases targeted towards ameliorating the factors that lead to this vulnerability. Environmental health efforts could use model predictions to focus on preventing spillover events that are especially likely to lead to the evolution of highly virulent pathogens.

The intertwining crises of climate change and biodiversity loss have made it urgent that we understand how ecosystems respond to environmental change so that we can predict the

consequences of ongoing global change and design conservation and restoration strategies to maintain ecological function in a changing world. Animal movement, including migration, plays an important role in response to environmental change. Mathematical models allow us to gain clarity about how migration mediates the response of populations, communities and ecosystems to global change. This helps to advance our understanding of the biology of global change, which in turn helps to prepare for the future.

References

Bauer, S., and B. J. Hoye. 2014. Migratory Animals Couple Biodiversity and Ecosystem Functioning Worldwide. Science 344:1242552.

Grainger, T. N., A. Senthilnathan, P.-J. Ke, M. A. Barbour, N. T. Jones, J. P. DeLong, S. P. Otto, M. I. O'Connor, K. E. Coblentz, N. Goel, J. Sakarchi, M. C. Szojka, J. Levine, and R. M. Germain. 2021. An empiricist's guide to using ecological theory. The American Naturalist.

Ljungström, G., T. J. Langbehn, and C. Jørgensen. 2021. Light and energetics at seasonal extremes limit poleward range shifts. Nature Climate Change 11:530–536.

Shao, C., M. Torstenson, and A. K. Shaw. 2025. Effect of resistant compartment on pathogen strategy in partially migratory populations. PLOS ONE 20:e0316640.

Shaw, A. K., A. T. Bisesi, C. Wojan, D. Kim, M. Torstenson, null Naven Narayanan, P. Lutz, R. Ales, and C. Shao. 2024. Six personas to adopt when framing theoretical research questions in biology. Proceedings of the Royal Society B: Biological Sciences 291:20240803.

Shipley, J. R., C. W. Twining, C. C. Taff, M. N. Vitousek, A. Flack, and D. W. Winkler. 2020. Birds advancing lay dates with warming springs face greater risk of chick mortality. Proceedings of the National Academy of Sciences of the United States of America 117.

Stratmann, T. S. M., M. Forrest, W. Traylor, N. Dejid, K. A. Olson, T. Mueller, and T. Hickler. 2023. Movement drives population dynamics of one of the most mobile ungulates on Earth: Insights from a mechanistic model. Ecology 104:e4071.

Tsujii, K., M. Otsuki, T. Akamatsu, K. Amakasu, M. Kitamura, T. Kikuchi, A. Fujiwara, H. Shirakawa, K. Miyashita, and Y. Mitani. 2021. Annual variation of oceanographic conditions changed migration timing of bowhead whales Balaena mysticetus in the southern Chukchi Sea. Polar Biology 44:2289–2298.

Appendix A

Strength of seasonality and type of migratory cue determine the fitness consequences of changing phenology for migratory animals

Supplemental Methods

When we allow $a \neq 1$, for tractability, we choose to start the annual cycle at some time t when the original function for habitat 2 crosses over the function for habitat 1 (a time when migration from habitat 1 to habitat 2 occurs) and end the annual cycle at the next time t when the function for habitat 2 crosses over the function for habitat 1. The time cues when $a \neq 1$ take the form

$$t_Z = \tan^{-1}\left(\frac{\sin(b)}{\cos(b)-a}\right) + (z-1)\pi \quad \text{(A1)}$$

where z is an integer.

For our analysis, we must split into two conditions. In the first condition, we start the annual cycle at t_1, which in the first condition marks a time when migration from habitat 1 to habitat 2 should occur. This condition applies when $a < 1$ and either $0 < b < \cos^{-1}(a)\ mod\ 2\pi$ or $2\pi - \cos^{-1}(a) < b < 2\pi\ mod\ 2\pi$. In the second condition, we start the annual cycle at t_2, which in the second condition marks a time when migration from habitat 1 to habitat 2 should occur. This condition applies when $a \geq 1$ and when $a < 1$ and $\cos^{-1}(a) < b < 2\pi - \cos^{-1}(a)\ mod\ 2\pi$.

The cumulative habitat quality experienced before the phenological change when migration is optimally timed is the same for both cue types. In condition 1,

$$Q_t^* = Q_e^* = 2\cos\left(b + \tan^{-1}\left(\frac{\sin(b)}{a-\cos(b)}\right)\right) - \frac{2a}{\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} \quad \text{(A2a)}$$

In condition 2,

$$Q_t^* = Q_e^* = \frac{2(1+a^2-2a\cos(b))}{(a-\cos(b))\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} \quad \text{(A2b)}$$

Because migration occurs at the same time in both conditions but for a given migration time, movement in the two conditions goes in opposite directions, $Q_t^* = Q_e^*$ in condition 1 is equal to $-Q_t^* = -Q_e^*$ in condition 2. The equation for $Q_t^* = Q_e^*$ in condition 1 is positive when condition 1 applies and negative when condition 2 applies. The equation for $Q_t^* = Q_e^*$ in condition 2 is positive when condition 2 applies and negative when condition 1 applies. This is to say that overall,

$$Q_t^* = Q_e^* = \left|\frac{2(1+a^2-2a\cos(b))}{(a-\cos(b))\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}}\right| \quad \text{(A2c)}$$

(Figure A1). As in the case where the amplitudes of both habitat functions are the same, this value gets larger when b is closer to $\pi \; mod \; 2\pi$. In this case, this value also gets larger for larger values of a.

One way that this equation is different from the equation describing $Q_t^* = Q_e^*$ when $a = 1$ is that it can still take a positive value when there is no horizontal translation between the two habitat quality functions. When $b = 0$,

$$Q_t^* = Q_e^* = |2 - 2a| \quad \text{(A3)}$$

which is larger than zero whenever $a \neq 1$.

When we allow $a \neq 1$, the cumulative habitat quality experienced when using cues that were optimal in the original habitat system following a phenological shift is not necessarily equal for migrants that use temporal cues and environmental cues. When migrants use temporal cues, this value is given by

$$Q_t^{\wedge} = 2\cos\left(b + c + \tan^{-1}\left(\frac{\sin(b)}{a-\cos(b)}\right)\right) - \frac{2a}{\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} \quad \text{(A4a)}$$

in condition 1 and by

$$Q_t^{\wedge} = -2\cos\left(b + c + \tan^{-1}\left(\frac{\sin(b)}{a-\cos(b)}\right)\right) + \frac{2a}{\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} \quad \text{(A4b)}$$

in condition 2. The variation of $Q_t^{\wedge}$ as a function of *a* and *c* when $b = \pi$ is shown in Figure A2.

When migrants use environmental cues, we end up with further sub-conditions. This is because for migration from habitat 2 in this case, we not only need to find a time at which the new habitat 2 function is equal to the habitat quality cue, we need to find a time that would lead to optimal migration if we set $c = 0$. However, when $b = \pi$,

$$Q_e^{\wedge} = \sqrt{\cos(b + \cot^{-1}((a - \cos(b))\csc(b)))^2} - \cos(\mathrm{b} + \mathrm{c} + \cot^{-1}((\mathrm{a} - \cos(\mathrm{b}))\csc(\mathrm{b}))) + \mathrm{a}(-\cos(\mathrm{b} + \mathrm{c} + \sin^{-1}(\sin(\mathrm{b} + \cot^{-1}((\mathrm{a} - \cos(\mathrm{b}))\csc(\mathrm{b})))))) + \frac{1}{\sqrt{1+\frac{\sin(\mathrm{b})^2}{(\mathrm{a}-\cos(\mathrm{b}))^2}}}) \quad \text{(A5)}$$

(Figure A3).

The difference between the cumulative habitat quality experienced before and after the phenological shift may also take different values depending on cue type. When the migrant uses temporal cues and $b = \pi$, this value is given by

$$Q_t^{\wedge} - Q_t^* = -2\cos\left(b + c + \tan^{-1}\left(\frac{\sin(b)}{a-\cos(b)}\right)\right) + \frac{2a}{\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} - \frac{2(1+a^2-2a\cos(b))}{(a-\cos(b))\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} \quad \text{(A6)}$$

(Figure A4).

When the migrant uses environmental cues and $b = \pi$, this value is given by

$$Q_e^{\wedge} - Q_e^* = \sqrt{\cos(b + \cot^{-1}((a - \cos(b))\csc(b))^2} - \cos\left(b + c + \cot^{-1}\left((a - \cos(b))\csc(b)\right)\right) - \frac{2\left(1+a^2-2a\cos(b)\right)}{(a-\cos(b))\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}} + \mathrm{a}(-\cos\left(b + c + \cot^{-1}\left((a - \cos(b))\csc(b)\right)\right) + \frac{1}{\sqrt{1+\frac{\sin(b)^2}{(a-\cos(b))^2}}}) \quad \text{(A7)}$$

(Figure A5).

Supplemental Figures

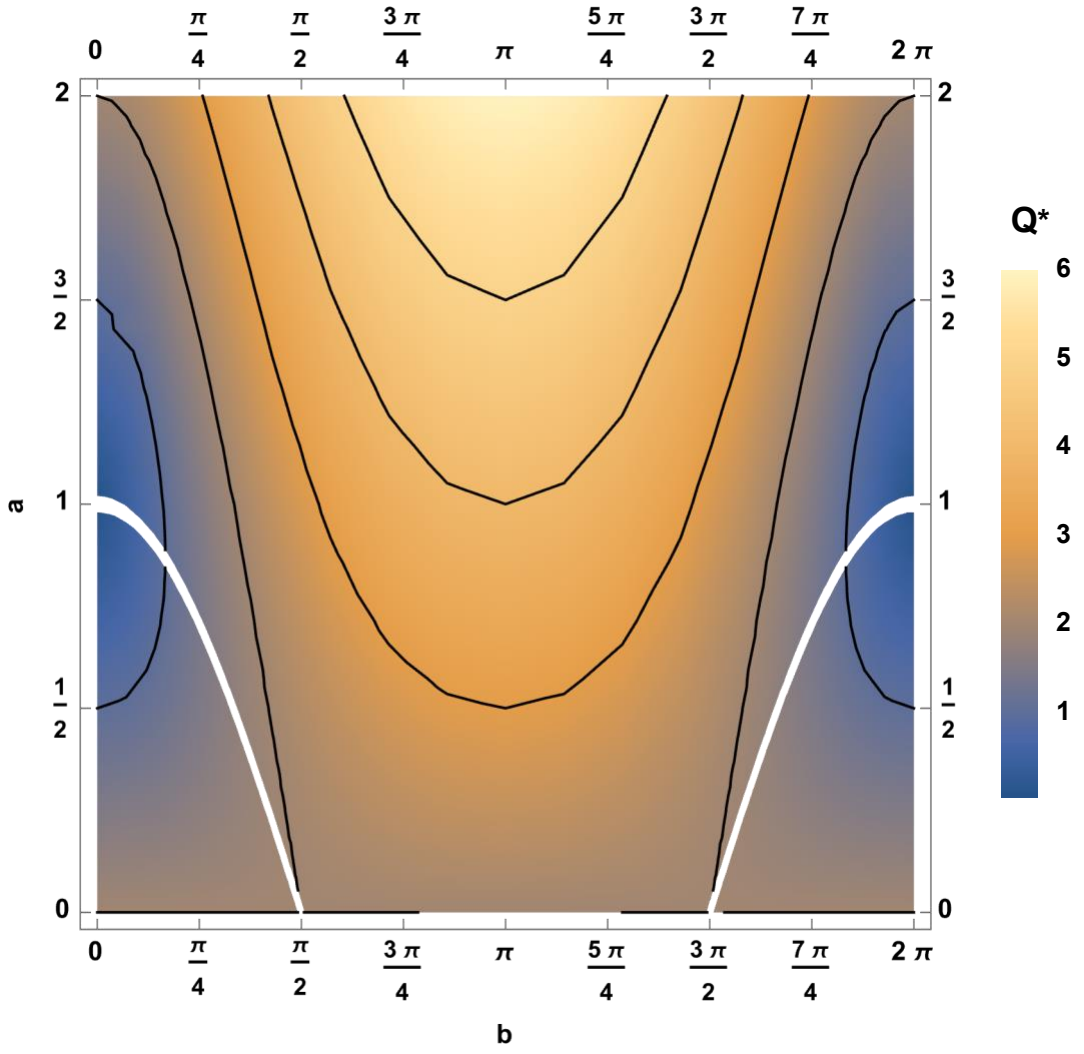

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Figure A1 The cumulative habitat quality experienced by a migrant using optimal time (Q_t^*) or environmental cues (Q_e^*) as a function of the amplitude of habitat 1 (a) and the horizontal displacement between the two habitats (b).

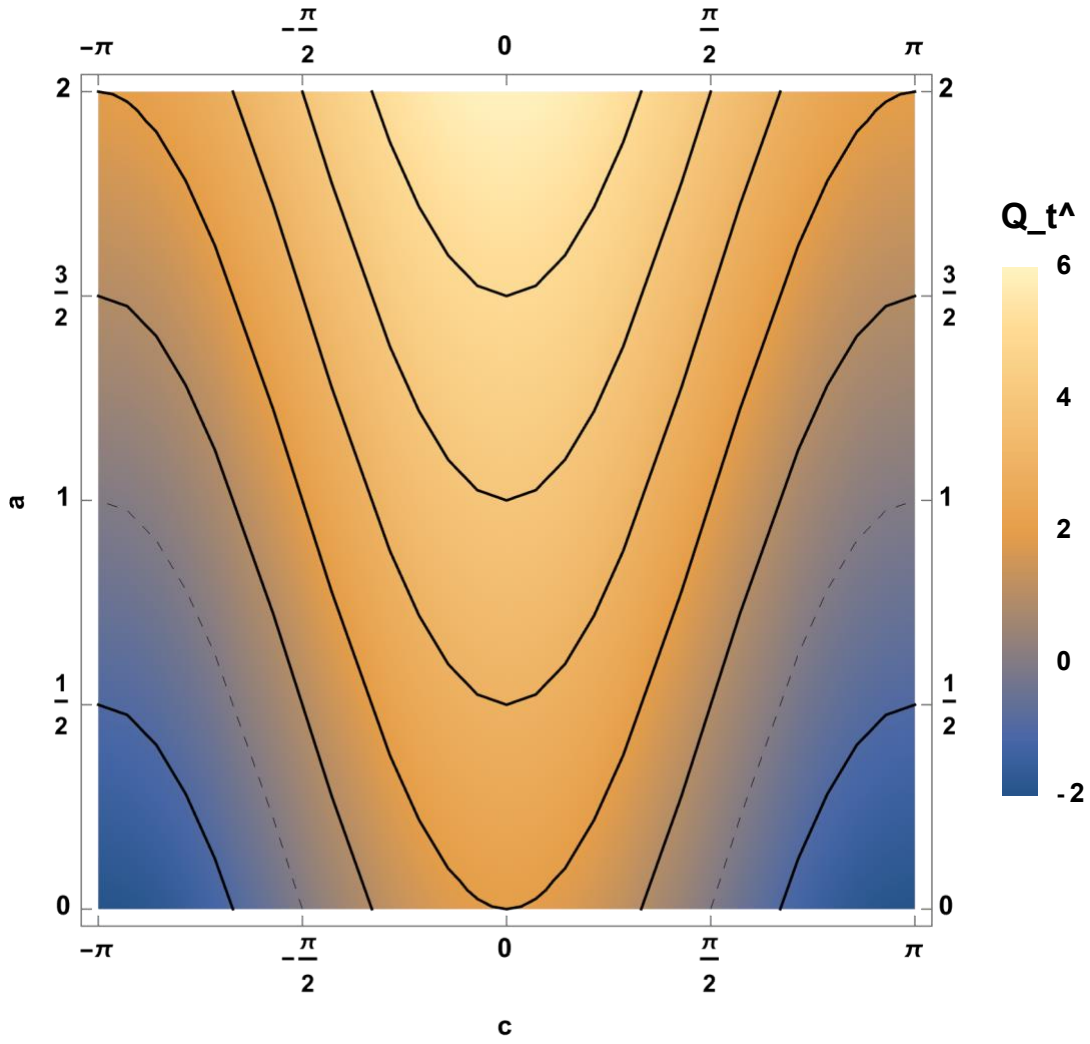

Figure A2 The cumulative habitat quality experienced by a migrant using previously optimal temporal cues ($Q_t^{\wedge}$) as a function of the size of the horizontal shift to habitat 2 (c) and the amplitude of habitat 1 (a) when the original horizontal displacement between the two habitats is equal to π ($b = \pi$). The isocline on which $Q_t^{\wedge} = 0$ is shown with a dashed line.

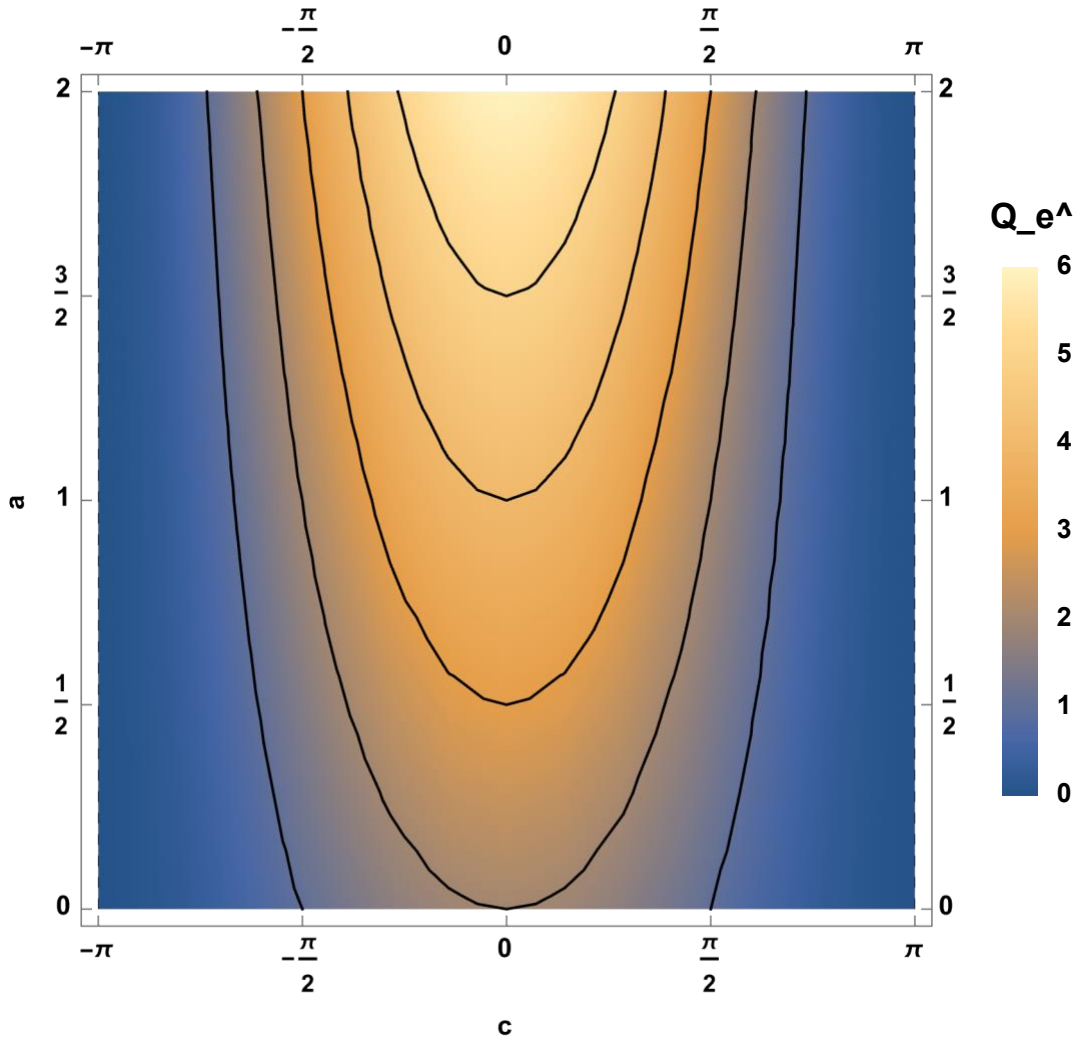

Figure A3 The cumulative habitat quality experienced by a migrant using previously optimal environmental cues ($\hat{Q_e}$) as a function of the size of the horizontal shift to habitat 2 (c) and the amplitude of habitat 1 (a) when the original horizontal displacement between the two habitats is equal to π ($b = \pi$).

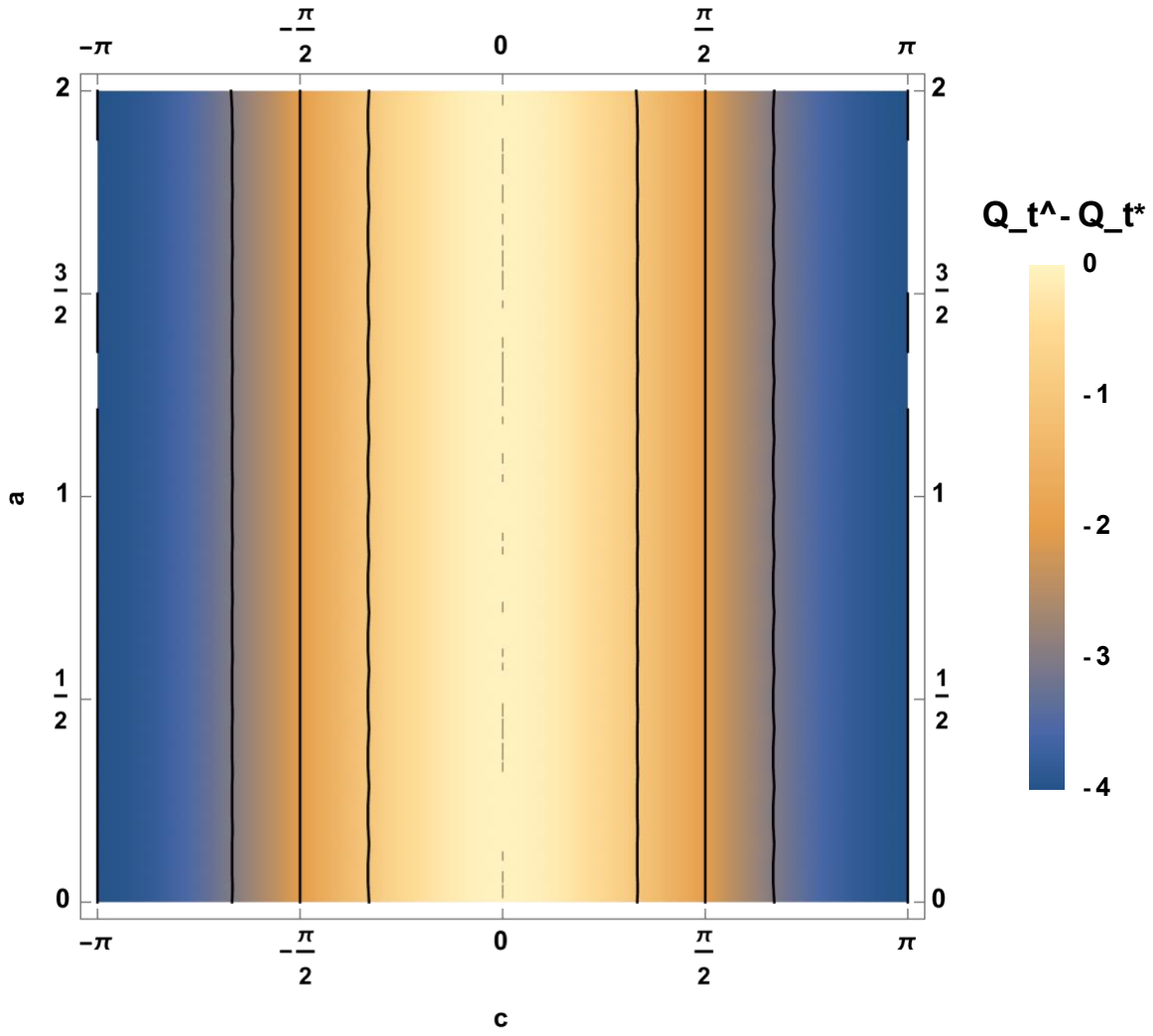

Figure A4 The difference between the cumulative habitat quality experienced by a migrant using temporal cues before and after the horizontal shift to habitat 2 ($Q_t^{\wedge} - Q_t^*$) as a function of the size of the horizontal shift to habitat 2 (c) and the amplitude of habitat 1 (a) when the original horizontal displacement between the two habitats is equal to π ($b = \pi$). The isocline on which $Q_t^{\wedge} - Q_t^* = 0$ is shown with a dashed line.

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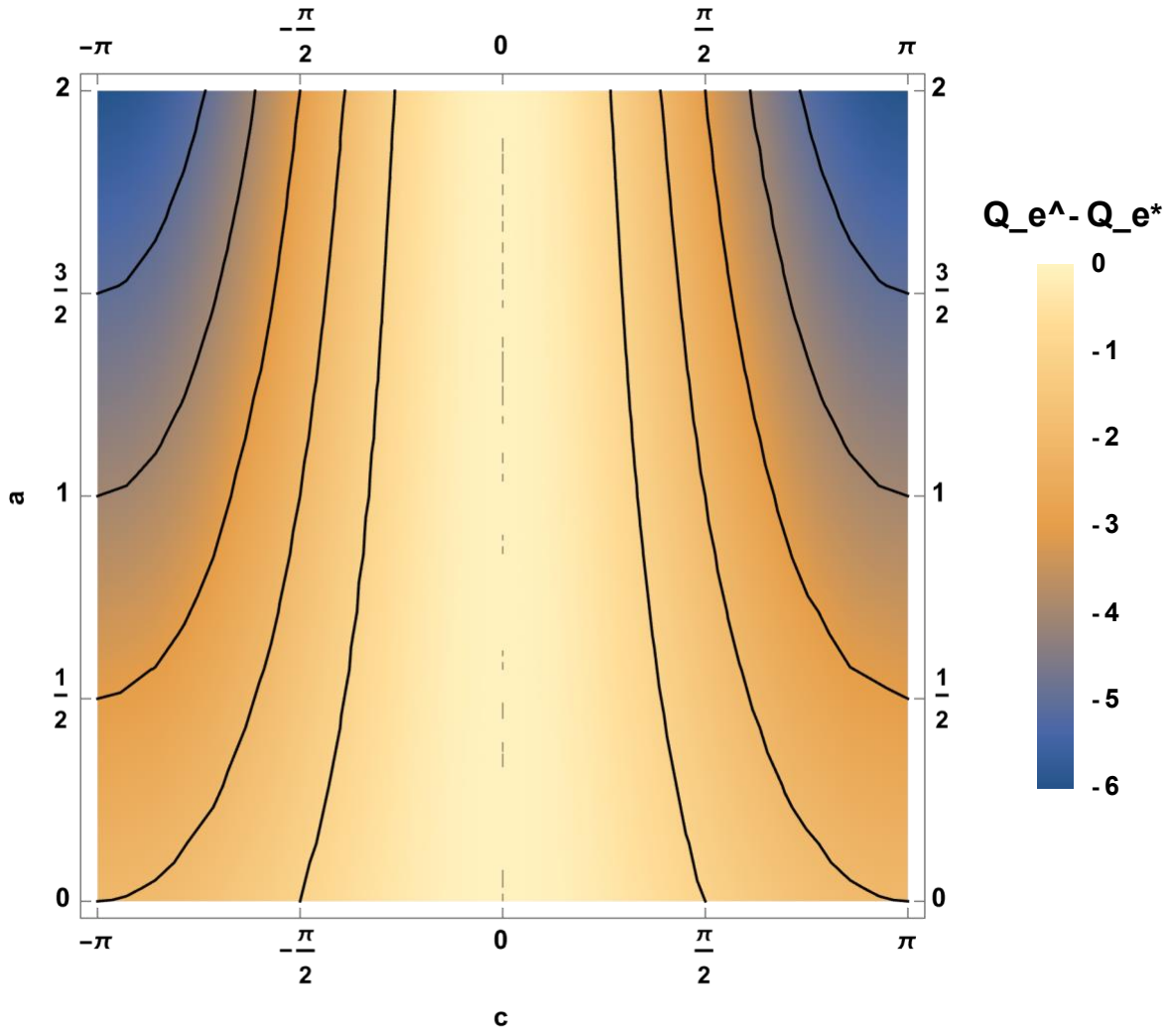

Figure A5 The difference between the cumulative habitat quality experienced by a migrant using environmental cues before and after the horizontal shift to habitat 2 ($Q_e^{\wedge} - Q_e^*$) as a function of the size of the horizontal shift to habitat 2 (c) and the amplitude of habitat 1 (a) when the original horizontal displacement between the two habitats is equal to π ($b = \pi$). The isocline on which $Q_e^{\wedge} - Q_e^* = 0$ is shown with a dashed line.

Appendix B

Pathogen evolution following spillover from a resident to a migrant host population depends on interactions between host life history speed and cost of infection

Sensitivity Analysis

Methods

We performed a sensitivity analysis using Latin Hypercube Sampling following the methodology of (Marino et al., 2008) in order to determine the robustness of our results about how the change in virulence depends on host tolerance and pace of life. We generated 10,000 combinations of values for the following parameters: equilibrium population size without infection ($400 < N < 1600$), the density dependence parameter ($0.0001 < d < 0.01$), the resident tolerance parameter ($0.01 < m_R < 2$), the baseline fecundity rate in the resident population ($0.1 < f_R < 2$), the baseline mortality rate in the resident population ($0.01 < \mu_R < 0.2$), the maximum transmission rate ($0.003 < \beta_{max} < 0.01$), and the recovery rate ($0.05 < \gamma < 0.2$). For each of these sets of parameters, we performed four simulations. First, we ran a simulation in which migrant and residence tolerance are equal, but migrant pace of life is slower than resident pace of life (i.e. migrant baseline fecundity rate f_M is some value $0.001 < \epsilon_f < 0.1$ smaller than f_R and migrant baseline mortality rate μ_M is some value $0.0001 < \epsilon_\mu < 0.01$ smaller than μ_R). Second, we ran a simulation in which migrant and residence tolerance are equal, but migrant pace of life is faster than resident pace of life (i.e. migrant baseline fecundity rate f_M is some value $0.001 < \epsilon_f < 0.1$ larger than f_R and migrant baseline mortality rate μ_M is some value $0.0001 < \epsilon_\mu < 0.01$ larger than μ_R), Third, we ran a

simulation in which migrant and resident pace of life are equal, but migrant tolerance is higher than resident tolerance (i.e. the migrant tolerance parameter m_M is some value $0.001 < \epsilon_m < 0.1$ smaller than m_R). Fourth, we ran a simulation in which migrant and resident pace of life are equal, but migrant tolerance is lower than resident tolerance (i.e. the migrant tolerance parameter m_M is some value $0.001 < \epsilon_m < 0.1$ larger than m_R). For each of these simulations, we determined how much virulence changed when the migrant population was added in.

Results

In general, the patterns that we found across the sensitivity analysis aligned with the main results we present in the manuscript (>75% for each of the four sets of sensitivity analyses; see figure B6). When migrant and residence tolerance are equal, but migrant pace of life is slower than resident pace of life (i.e. migrant baseline fecundity rate f_M is some value $0.001 < \epsilon_f < 0.1$ smaller than f_R and migrant baseline mortality rate μ_M is some value $0.0001 < \epsilon_\mu < 0.01$ smaller than μ_R) there is generally a decrease in virulence following spillover (Figure B6a) . When migrant and residence tolerance are equal, but migrant pace of life is faster than resident pace of life (i.e. migrant baseline fecundity rate f_M is some value $0.001 < \epsilon_f < 0.1$ larger than f_R and migrant baseline mortality rate μ_M is some value $0.0001 < \epsilon_\mu < 0.01$ larger than μ_R), there is generally an increase in virulence following spillover (Figure B6b). When migrant and resident pace of life are equal, but migrant tolerance is higher than resident tolerance (i.e. the migrant tolerance parameter m_M is some value $0.001 < \epsilon_m < 0.1$ smaller than m_R), there is generally an increase in virulence following spillover (Figure B6c). When migrant and resident pace of life are equal, but migrant tolerance is lower than resident tolerance (i.e. the migrant tolerance parameter m_M is some value $0.001 < \epsilon_m < 0.1$ larger than m_R), there is generally a decrease in virulence (Figure B6d).

Supplemental Figures

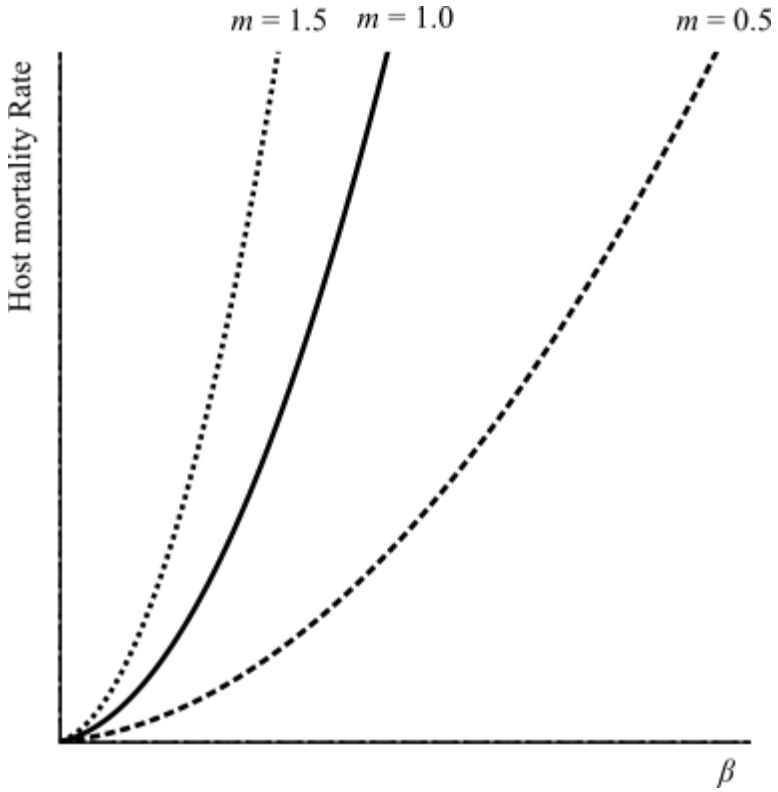

Figure B1*The relationship between transmission and mortality rate.* The host mortality rate increases quadratically with the transmission rate of the pathogen the host is infected with. The value of *m,* which represents the host's tolerance to infection determines how steep this relationship is.

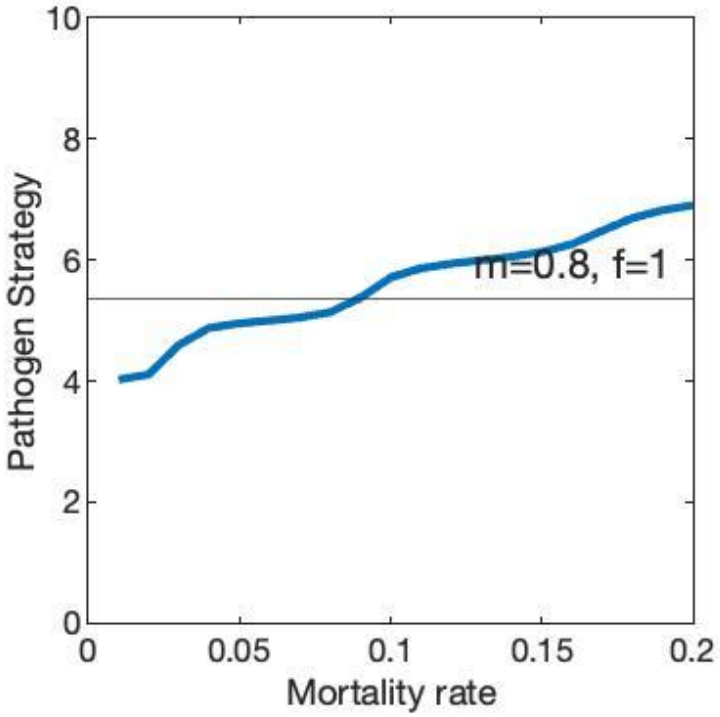

Figure B2 *Pathogen strategies in one population as a function of host mortality rate.* In a single population in which N^*=800, m=0.8 and f=1 a) the pathogen strategy that evolves increases monotonically as host mortality rate (μ) increases. The pathogen strategy that evolves in the resident population in the two population simulation (m=0.8 and f=1) is shown as a horizontal line for easy comparison with the pathogen strategy that evolves in the migrant population in the two population simulations.

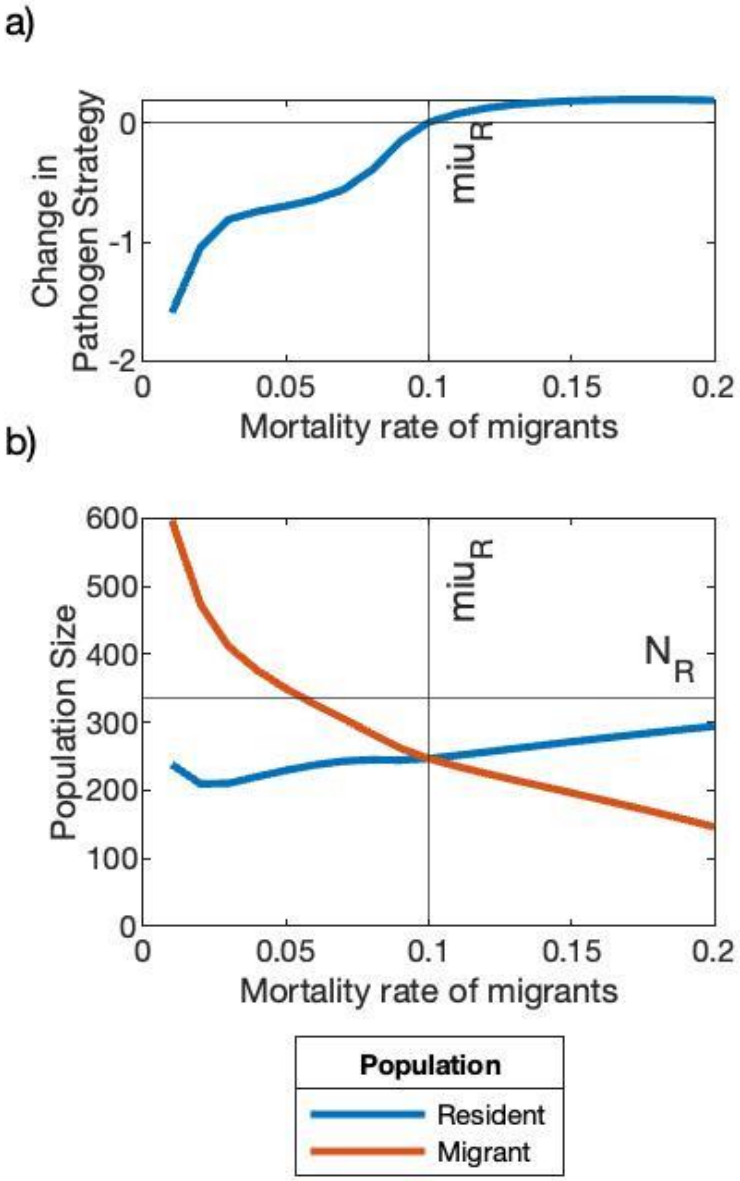

Figure B3 *Migrant and resident populations differ in mortality rate.* Following spillover from a resident into a migrant population in which $N*$=800 for both populations, $m_R = m_M = 0.8$, $f_R = f_M$=1 and $\mu_R = 0.09$ a) the change in pathogen strategy following spillover increases as migrant mortality rate (μ_M) increases. b) The size of the resident population following spillover increases as migrant mortality rate (μ_M) increases and size of the migrant population decreases as migrant mortality rate (μ_M) increases. The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

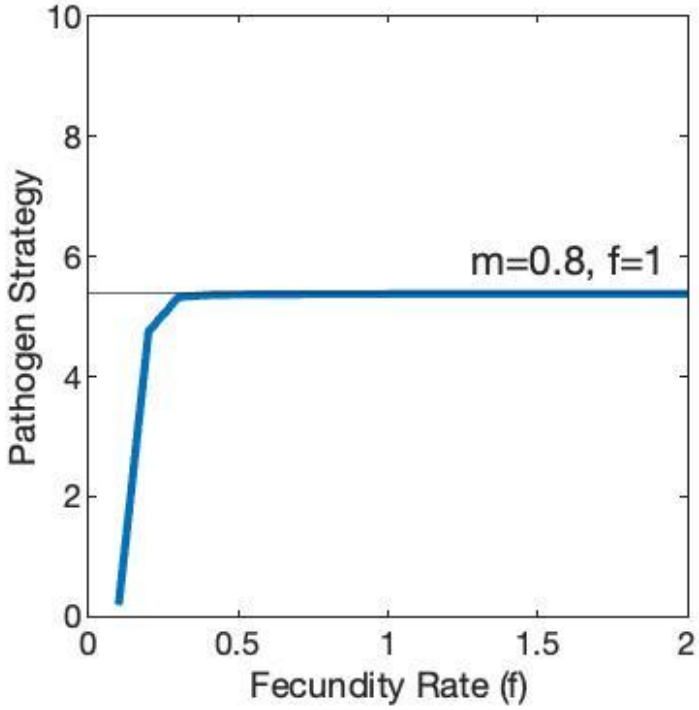

Figure B4 *Pathogen strategies in one population as a function of host fecundity rate.* In a single population in which N^*=800, m=0.8 and μ=0.09 a) the pathogen strategy that evolves generally does not change as host mortality rate (μ) increases. The pathogen strategy that evolves in the resident population in the two population simulation (m=0.8 and f=1) is shown as a horizontal line in all panels for easy comparison with the pathogen strategy that evolves in the migrant population in the two population simulations.

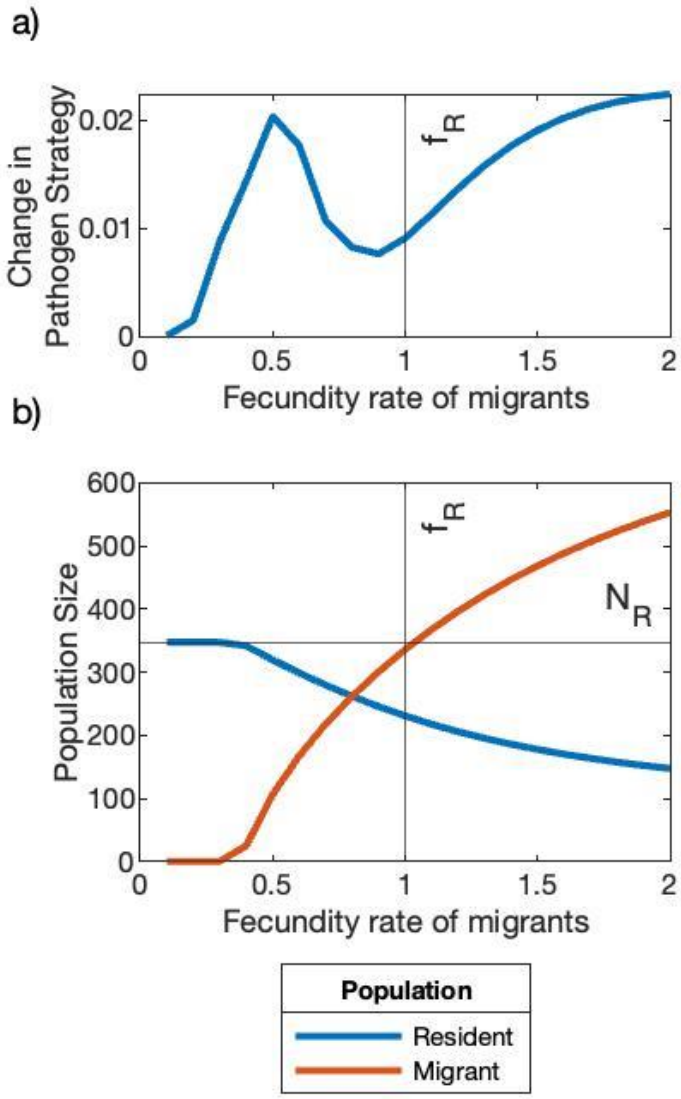

Figure B5 *Migrant and resident populations differ in fecundity rate.* Following spillover from a resident into a migrant population in which $N *$=800 for both populations, $m_R = m_M = 0.8$, f_R=1 and $\mu_R = \mu_M =$ 0.09 a) there is generally minimal change in pathogen strategy following spillover. b) The size of the resident population following spillover decreases as migrant fecundity rate (f_M) increases and size of the migrant population increases as migrant fecundity rate (f_M) increases. The number of resident individuals before spillover is shown as the horizontal line labelled N_R for reference.

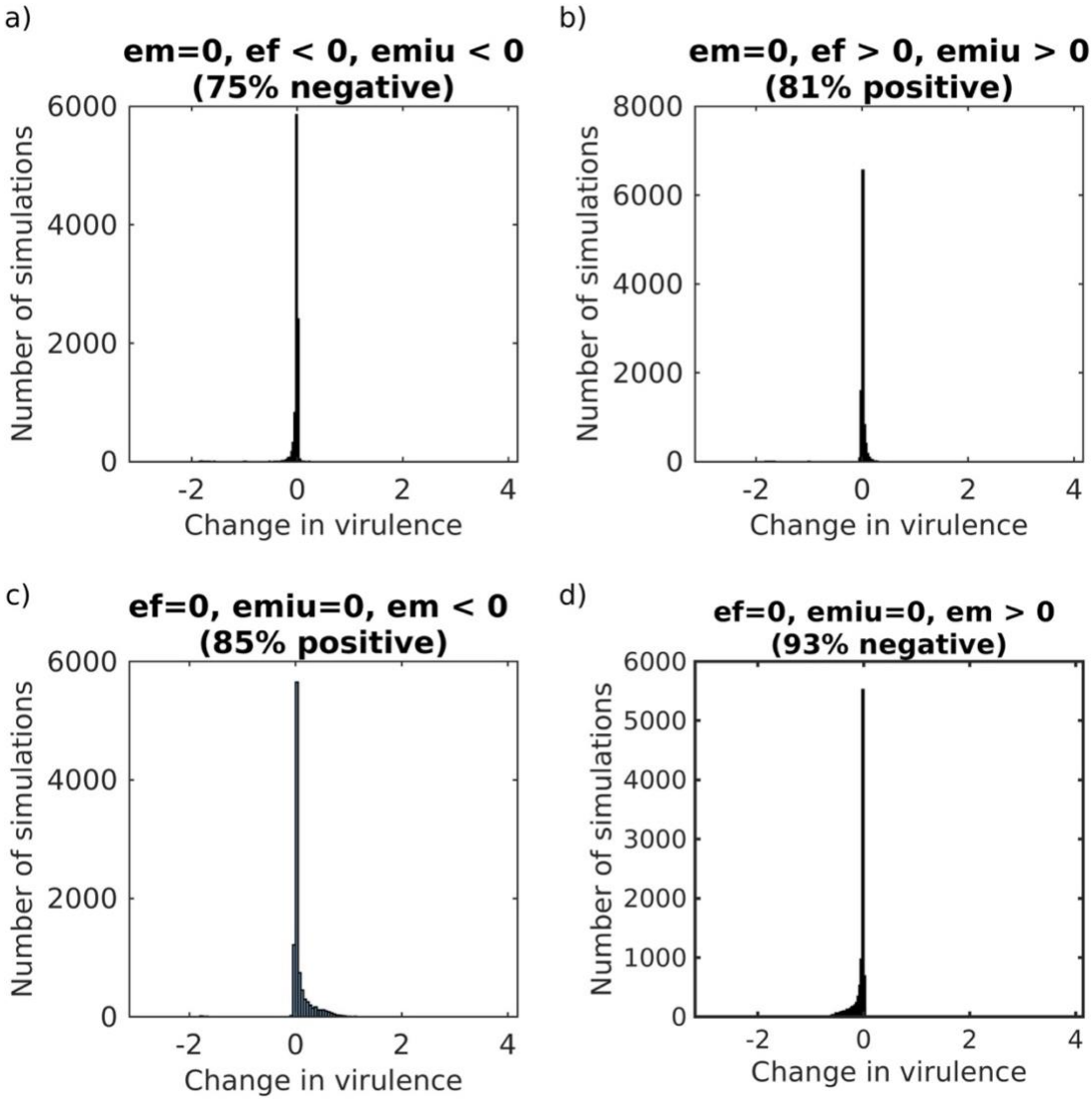

Figure B6 *Sensitivity analysis results* In each panel, we display a histogram representing the number of simulations (out of 10,000) in our sensitivity analysis for which we see a given change in virulence. a) When migrant and resident tolerance to infection are equal and migrant life history speed is slower than resident life history speed, there is generally a decrease in virulence. b) When migrant and resident tolerance to infection are equal and migrant life history speed is faster than resident life history speed, there is generally an increase in virulence. c) When migrant and resident pace of life are equal and migrant tolerance to infection is higher than resident tolerance to infection there is generally an increase in virulence. d) When migrant and resident pace of life are equal and migrant tolerance to infection is lower than resident tolerance to infection there is generally a decrease in virulence. The percentage of simulations that align with our main qualitative findings are given in parentheses in the subpanel labels.

References

Marino, S., Hogue, I. B., Ray, C. J., & Kirschner, D. E. (2008). A methodology for performing global uncertainty and sensitivity analysis in systems biology. *Journal of Theoretical Biology*, *254*(1), 178–196. https://doi.org/10.1016/j.jtbi.2008.04.011

Appendix C

The code that was used to generate the results in these chapters is publicly available on Zenodo. The citations and links to this code are provided here.

Chapter 1:

Torstenson, M. S., & Shaw, A. K. (2024). Model code associated with: Strength of seasonality and type of migratory cue determine the fitness consequences of changing phenology for migratory animals. In Oikos. Zenodo. https://doi.org/10.5281/zenodo.13695479

Chapter 2:

Torstenson, M., & Shaw, A. K. (2024). Model code associated with: Population response to extreme climate events depends on population spatial distribution. Zenodo. https://doi.org/10.5281/zenodo.14510659

Chapter 3:

Torstenson, M., & Shaw, A. K. (2024). Model code associated with: Pathogen evolution following spillover from a resident to a migrant host population depends on interactions between host life history speed and cost of infection. In Journal of Animal Ecology. Zenodo. https://doi.org/10.5281/zenodo.10723759