EcoHealth 17, 4–12, 2020
https://doi.org/10.1007/s10393-020-01472-1

EcoHealth

Original Contribution

Ecological Fallacy and Aggregated Data: A Case Study of Fried Chicken Restaurants, Obesity and Lyme Disease

Daniel J. Salkeld[1] and Michael F. Antolin[1]

Department of Biology, Colorado State University, Fort Collins, CO 80523

Abstract: Interdisciplinary approaches are merited when attempting to understand the complex and idiosyncratic processes driving the spillover of pathogens from wildlife and vector species to human populations. Public health data are often available for zoonotic pathogens but can lead to erroneous conclusions if the data have been spatially or temporally aggregated. As an illustration, we use human Lyme disease incidence data as a case study to examine correlations between mammalian biodiversity, fried chicken restaurants and obesity rates on human disease incidence. We demonstrate that Lyme disease incidence is negatively correlated with mammalian biodiversity, the abundance of fried chicken restaurants and obesity rates. We argue, however, that these correlations are spurious, representing both an 'ecologic fallacy' and Simpson's paradox, and are generated by the use of aggregated data. We argue that correlations based on aggregated data across large spatial scales must be rigorously examined before being invoked as proof of disease ecology theory or as a rationale for public health policy.

Keywords: Biodiversity–disease hypothesis, Fried chicken, Ecologic fallacy, Aggregate data, Eco-epidemiology, Lyme disease statistics

Introduction

To understand drivers of disease emergence, epidemiologists analyze patterns of the distribution and determinants of pathogen exposure and susceptibility in human populations. Physicians and laboratories that observe and diagnose 'notifiable' diseases are required by law to alert government authorities, a process described as 'passive surveillance' (e.g., Schiffman et al. 2018). The data, based on individual disease cases, are first reported to local health agencies, then to higher levels of government, e.g., state health departments, and finally to national governments. During this process, the individual-based data are aggregated into summaries, in part to protect the privacy of the case-patients. These data are often made publicly available and allow insights into epidemic trends (e.g., is the public health burden increasing, decreasing or stable?), disease distribution (e.g., what is the geographic range, and does seasonal variation affect disease incidence?) and to inform allocation of resources to control the disease (e.g., how many people are infected?).

Disease ecologists also work to understand the distribution of pathogens, their hosts and their interactions with their environment, but typically with more of a bent toward describing patterns of infection in wildlife host and/or vector populations. Ecologists address questions like: How

Published online: February 6, 2020

Correspondence to: Daniel J. Salkeld, e-mail: dan.salkeld@colostate.edu

do habitat and climate influence local risk of exposure to pathogens? Do interactions between wildlife species impact pathogen transmission dynamics?

Reconciling the two approaches—epidemiology and ecology—has challenges. How does a field ecologist, well-versed in trapping small mammals and collecting questing ticks (for example), extend ecological data to predict incidence of vector-borne zoonotic disease in humans? How might an epidemiologist, taking advantage of publicly available passive surveillance data from health departments, incorporate patterns of distribution and abundance of wildlife hosts or vector populations?

One approach would be to exploit the publicly available data on disease incidence and combine them with insights (data) provided by ecological or environmental perspectives. Unfortunately, available data are often collected and/or reported at different spatial and/or temporal scales. For example, information on pathogen prevalence in host or vector populations is limited by logistical and cost constraints to small spatial scales (e.g., several sites for sample collections), but spatiotemporal heterogeneity in host–vector relationships may limit the ability to extrapolate to broader geographical areas (Pepin et al. 2012; Salkeld et al. 2015a; Millins et al. 2016). From the human perspective, physicians' reports of zoonotic disease in case-patient include information on area of residence (e.g., postal codes, or zip-codes in the USA), but not necessarily the actual site of exposure to the disease agent. Information on recent travel by case-patients is often not reported for several reasons: it is not required, it may violate agreements on the confidentiality of the patient information, or simply because it is not known. Many case-patients cannot identify the time and geographic location they were bitten by an arthropod vector, e.g., hard-bodied ticks (e.g., *Ixodes*) may attach, bite and feed on a person for 2–3 days before being detected, and some proportion of tick bites may go entirely unnoticed (Salkeld et al. 2019). Attributing the exact site of exposure is not straightforward, especially if the patient works frequently in outdoor environments or travels widely.

To align the various information sources, data must often be aggregated or summarized to a common scale, and these often conform to the geographic boundaries of political jurisdictions, e.g., counties, states or countries.

Unfortunately, there is a danger of generating spurious correlations when aggregated data are assumed to be able to explain phenomena that occur at an individual basis. Epidemiologists refer to this phenomenon as '*ecological fallacy*,' where inferences about the nature of individuals are deduced from attributes of the group to which those individuals belong (Gordis 2009; Webb and Bain 2011; Pollet et al. 2014). Amusingly (somewhat), given the term, disease ecologists are too often guilty of producing these fallacious relationships. Though it seems reasonable that large-scale patterns likely reflect mechanisms occurring at individual levels, there is no guarantee of this. Indeed, ecological fallacy can actually generate statistically significant patterns that are diametrically opposed to those occurring at the individual level: a phenomenon called Simpson's paradox, when correlations are reversed during data aggregation. Simpson's paradox arises when the causal driver has not been measured or incorporated into analyses (Bickel et al. 1975; Pollet et al. 2015, 2016).

To provide a case study to illustrate how aggregated data can lead to ecological fallacy, we examine data on potential drivers of Lyme disease incidence patterns in the eastern USA. Lyme disease often begins innocuously with a barely noticeable bite from a nymphal black-legged tick (*Ixodes scapularis*), the size of a poppy seed. If the tick is infected and injects the bacterium that causes Lyme disease—*Borrelia burgdorferi* sensu stricto (hereafter *B. burgdorferi*)—during feeding, a symptomatic rash—*erythema migrans*—may develop as the bacteria disseminate. As the infection spreads, a slew of other flu-like symptoms follow: headache, fever, chills and aching muscles and joints. Without timely diagnosis and antibiotic treatment, disease progression is dire, including arthritis, facial palsy, neurological damage and cardiac complications (Stanek et al. 2012; Forrester et al. 2014). With an estimated 300,000 new cases every year, Lyme disease is the most frequently contracted vector-borne disease in the USA (US) and constitutes a massive public health burden (Nelson et al. 2015). Human cases of Lyme disease are reported across the eastern USA, but are most frequent in two recognized foci: the northeast and the upper Midwest regions (Eisen and Eisen 2018, Fig. 1a), even though the black-legged tick vector occurs throughout the eastern USA (Arsnoe et al. 2015; Eisen et al. 2016).

The Lyme disease bacterium is zoonotic, i.e., it is maintained in wildlife and tick populations but can be transmitted to people. In the northeastern USA, *B. burgdorferi* infects a suite of small mammals in the wild, such as white-footed mice (*Peromyscus leucopus*), eastern chipmunks (*Tamias striatus*) and shrews (*Blarina brevicauda* and *Sorex* spp). These rodent hosts, in turn, infect immature black-legged ticks as the ticks take blood meals

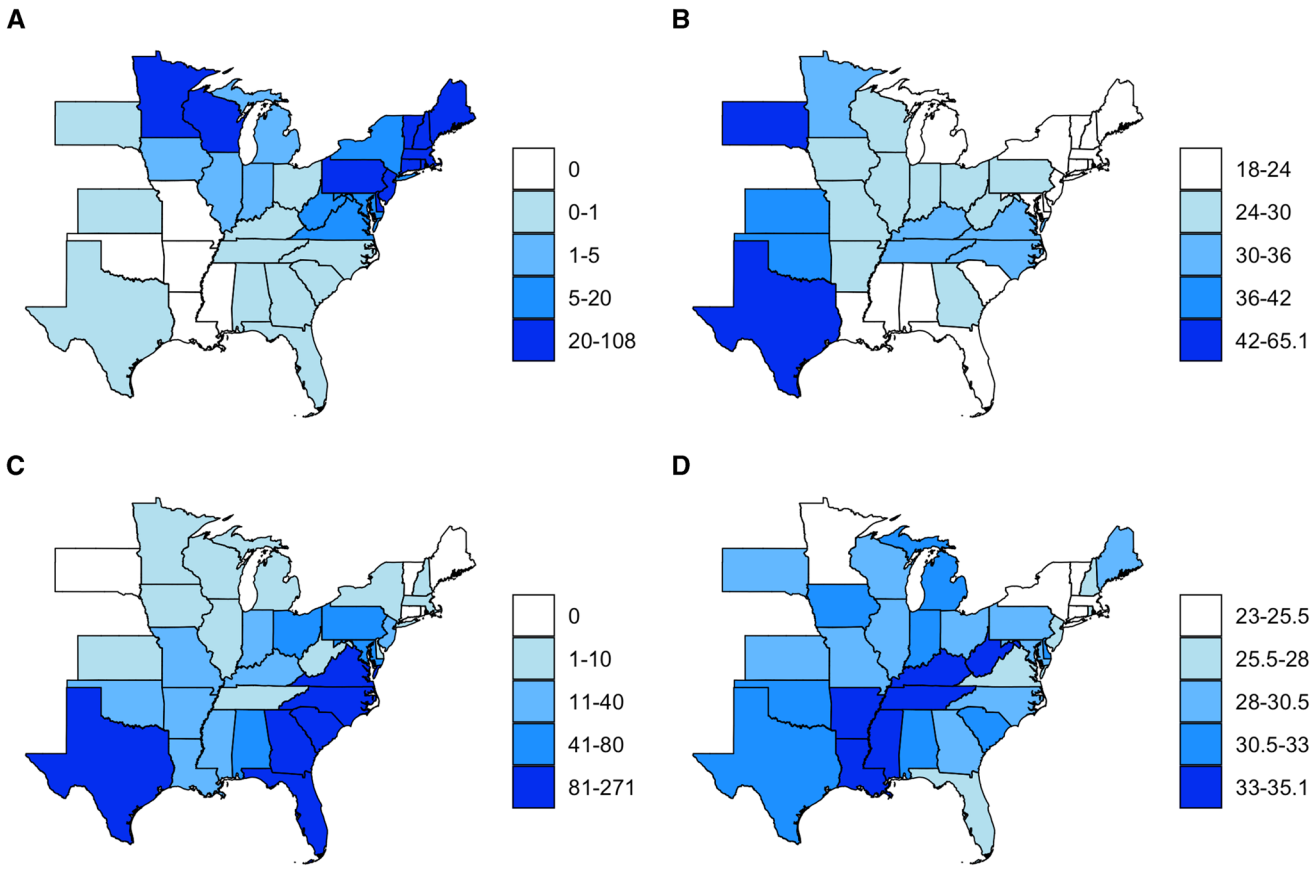

Figure 1. Heat maps of eastern USA, arranged by quintiles, showing **a** states with Lyme disease incidence (ln + 1), **b** mammalian biodiversity (number of species), **c** number of fried chicken restaurants (chain A, representative of both fried chicken restaurant chains) and **d** obesity rates.

from their hosts (LoGiudice et al. 2003). After molting—either from larva to nymph, or from nymph to adult—the now-infected ticks can transmit *B. burgdorferi* to other hosts. When infected black-legged ticks bite humans, *B. burgdorferi* spillover can occur. Spillover describes the process of humans becoming exposed to and infected by pathogens that more commonly infect wildlife species and can be summarized into three constituent parts for vector-borne diseases: (1) the pathogen is transmitted between wildlife host and arthropod vector populations; (2) humans are exposed to the source of infection by the bite of an infected vector; and (3) the human is susceptible to infection. Humans are a dead-end host; no further transmission occurs, so the spillover process at the individual level represents an important component of understanding Lyme disease transmission.

Biodiversity has been postulated to reduce Lyme disease via the 'dilution effect,' a hypothesis that argues that disease risk will decrease as a result of increased species diversity, because, with more host species that are incompetent disease hosts (they are inefficient at infecting ticks, or are refractory), infection prevalence in ticks will decrease (LoGiudice et al. 2003; Pongsiri et al. 2009; Keesing et al. 2010; Civitello et al. 2015). Investigations of Lyme disease and community ecology were some of the formative studies for this hypothesis (e.g., Ostfeld and Keesing 2000; LoGiudice et al. 2003, 2008), though the phenomenon is still debated (e.g., Ogden and Tsao 2009; Randolph and Dobson 2012; Wood and Lafferty 2013; Salkeld et al. 2013; Wilkinson et al. 2018; Halliday and Rohr 2019). We measure biodiversity using species richness of mammals at the state level, gleaned from the mammals' geographic ranges (Turney et al. 2014).

We test the hypothesis that human behavior is an important component of Lyme disease exposure. Food and diet may be regarded as a powerful way to investigate regional behavior and identity (Shortridge 2005 and references therein). Though fried chicken is consumed nationwide in the USA, it is culturally associated with the southeastern USA (Shortridge 2005), where Lyme disease incidence is lower. We use the number of fried chicken restaurants per state to investigate the importance of geo-

graphical patterns of human behavior upon Lyme disease incidence. We also examine the relationship between Lyme disease incidence and state-level obesity rates in order to investigate whether comorbidity factors and health status may be important in Lyme disease epidemiology.

Importantly, we analyze the relationships between Lyme disease, biodiversity, fried chicken restaurants and obesity as an exercise that illustrates how ecologic fallacies can arise and appear feasible.

Methods

All data were obtained from publicly accessible aggregated datasets in 35 US states where Lyme disease is commonly reported (Turney et al. 2014) and is transmitted by the black-legged tick. Data on Lyme disease incidence (number of cases of Lyme disease per 100,000 people in 2013) and mammalian species richness were kindly provided by Virginie Millien and were originally garnered from the US Centers for Disease Control and Prevention (CDC, http://www.cdc.gov/lyme/stats/) and the Smithsonian Institution, National Museum of Natural History, North American Mammals database, available online (http://www.mnh.si.edu/mna/search_name.cfm) (Turney et al. 2014).

We obtained data on restaurant abundance for two fried chicken restaurant chains, here referred to as fried chicken restaurant—A and fried chicken restaurant—C. Data on fried chicken restaurant abundance (number of stores per state) were obtained using a Google web-search. Data on prevalence of self-reported obesity among US adults were obtained from the CDC: http://www.cdc.gov/obesity/data/prevalence-maps.html, with the data provided on http://www.cdc.gov/obesity/data/table-adults.html (accessed on July 13, 2015).

To examine geographic clustering, we analyzed the relationship with subgroups of the state-level data, assembled by geographic proximity: 'Southeast' states include Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee and Texas. 'Virginia and neighbors' include Delaware, Maryland, Virginia and West Virginia. The 'Northeast' includes Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island and Vermont. The 'Upper Midwest' includes Michigan, Minnesota, South Dakota and Wisconsin. The 'Midwest' includes Illinois, Indiana, Iowa, Kansas, Kentucky, Missouri, Ohio and Oklahoma.

To reduce skewness, Lyme disease data were ln + 1 transformed. Analyses were linear regressions, using 'R' for statistical analyses (R Core Team 2014), and code for analyses are available in the appendices.

Results

Overall, at the state level, we detected a negative relationship between biodiversity and Lyme disease incidence ($F_{1,33} = 4.45$, $r^2 = 0.12$, $p = 0.043$; Figs. 1, 2).

Similarly, Lyme disease incidence was lower in states with a higher abundance of fried chicken restaurants, and this negative relationship was consistent for both restaurant chains (fried chicken—A: $F_{1,33} = 6.66$, $r^2 = 0.17$, $p = 0.015$; fried chicken—C: $F_{1,33} = 6.05$, $r^2 = 0.15$, $p = 0.019$; Figs. 1, 2).

State-level obesity rates were also negatively related to Lyme disease incidence ($F_{1,33} = 29.44$, $r^2 = 0.47$, $p < 0.001$; Figs. 1, 2). Obesity rates were not related to the number of fried chicken restaurants (fried chicken—A: $F_{1,33} = 0.15$, $r^2 = 0.004$, $p = 0.70$; fried chicken—C: $F_{1,33} < 0.001$, $r^2 < 0.001$, $p = 0.997$).

When examined as sub-sampled geographical clusters of states, relationships between Lyme disease incidence and mammalian species richness, number of fried chicken restaurants and obesity rates were not statistically significant and often varied in trend, with some positive and some negative slopes (Fig. 3).

Discussion

Our analyses suggest that higher mammalian biodiversity is related to lower Lyme disease incidence at the state level, a finding that has previously been reported (Ostfeld and Keesing 2000).

But further, our analyses also suggest that a fried chicken diet can be good for your health, as higher numbers of fried chicken restaurants are associated with reduced incidence of Lyme disease. This relationship was consistent for two different fried chicken restaurant chains. Though the mechanisms underlying this relationship are not clear and are not directly measured in this investigation, it could be argued that a dietary component found in fried chicken may affect host susceptibility to the Lyme disease spirochete.

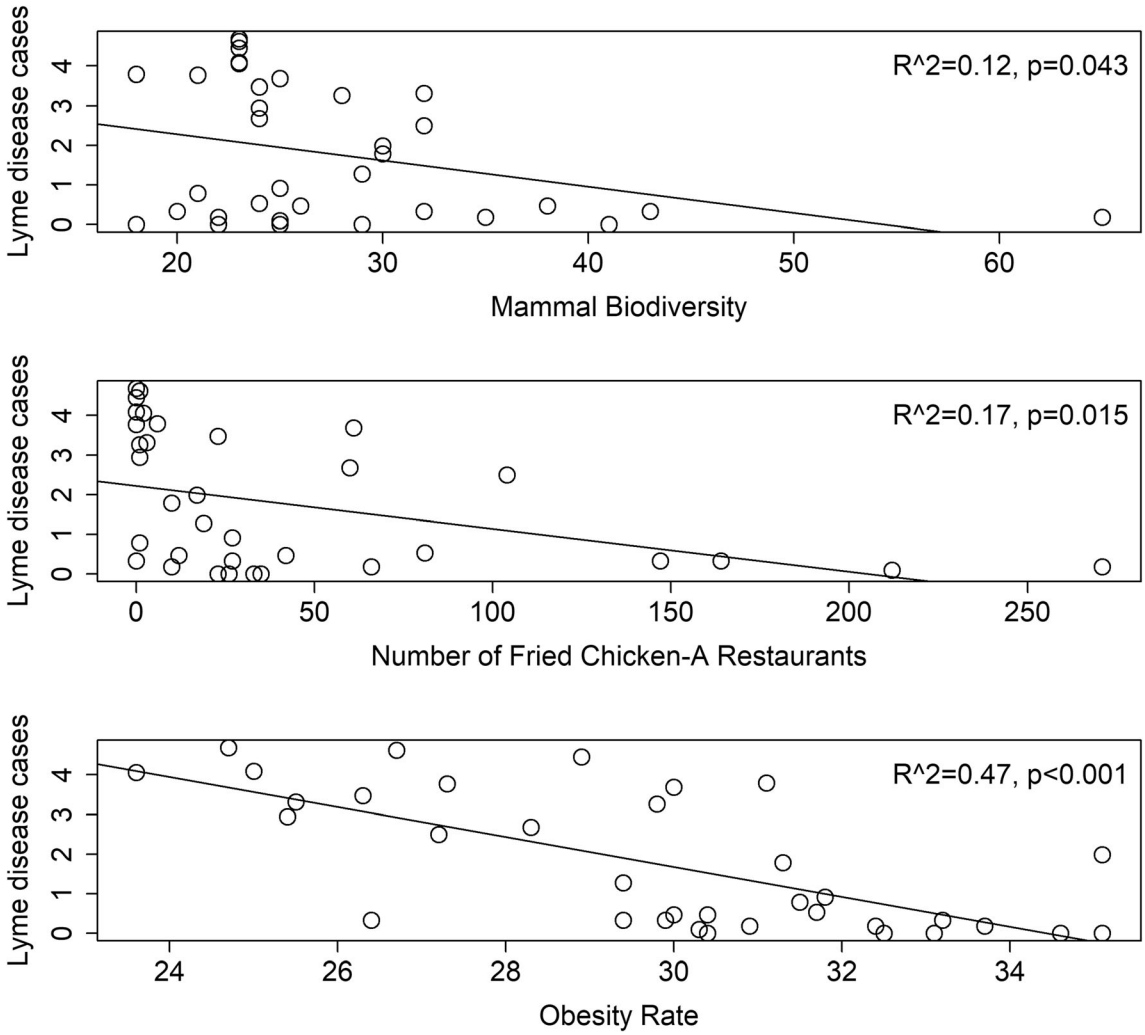

Figure 2. State-level relationships between Lyme disease incidence (ln + 1) and mammalian biodiversity (number of species) (top), number of fried chicken restaurants (chain A, representative of both fried chicken restaurant chains) (middle) and obesity rates (bottom).

Higher obesity rates were linked to reduced Lyme disease infection rates. A relationship between under-nutrition and increased susceptibility to infectious disease has previously been observed (e.g., Hickman et al. 2014). However, our finding conflicts with this pattern and suggests that over-nutrition reduces susceptibility to Lyme disease, which arises from zoonotic spillover disease. One speculative explanation may be a life-history trade-off: with limited resources, a host cannot exert an appropriate immune response upon exposure to infection, but when resources are plentiful the immune response is not limited.

Human behavior strongly influences susceptibility and exposure to infectious disease. The lower rates of Lyme disease incidence in states with higher numbers of fried chicken restaurants and obesity rates may be explained by human behavior. If obesity can be linked to lower rates of physical activity, presumably including outdoor activities such as hiking and gardening, then perhaps there are fewer opportunities for exposure to ticks in outdoor habitats (Porter et al. 2019), though these links require further research (Gascon et al. 2017). Thus, higher obesity rates are associated with lower Lyme disease incidence because of lower exposure rates to the actual pathogen and its vector. A similar mechanism could explain the association with fried chicken restaurants: electing to spend time in fried chicken restaurants implies a reduced exposure to tick habitat.

However, it is most likely that these correlations are entirely spurious and generated only because of the use of aggregated data. Each Lyme disease infection, like any zoonotic spillover event, occurs at the scale of the individual, yet our adopted datasets aggregate data to the state

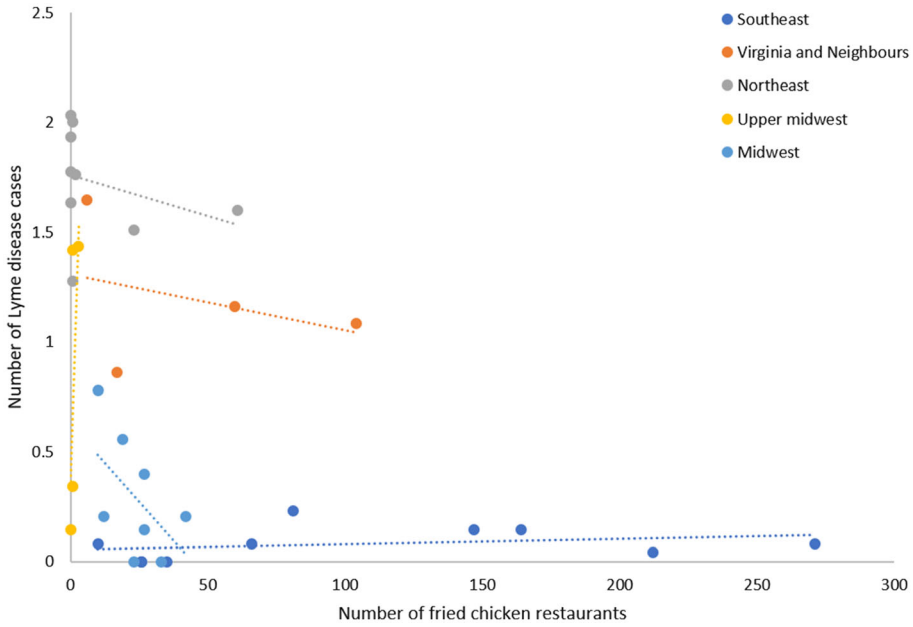

Figure 3. Geographical clusters of state-level data for Lyme disease incidence (log + 1) and the number of fried chicken restaurants—A. The trends vary in direction and strength, demonstrating Simpson's paradox, though none of the relationships are statistically significant.

level. Because the data have been collected at different scales and resolutions, we cannot be sure that correlations between Lyme disease infection, obesity and fried chicken restaurant density are epidemiologically related in any way (Gotway and Young 2002; Gordis 2009). The fact that these relationships tend not to hold when examined as geographical sub-clusters—exhibiting Simpson's paradox—suggests abundant caution should be applied to interpretation of these relationships (Fig. 3; Bickel et al. 1975; Pollet et al. 2014, 2015).

In case we have misled the reader up to this point, our analyses of Lyme disease as a response to mammalian biodiversity, fried chicken restaurants and obesity, are tongue-in-cheek and intended as an illustration of the dangers of advocating correlations based on aggregate data. Correlations can be intriguing and allow hypothesis development, but to ascertain whether there is a causal effect, individual-level data, experimental manipulations and rigorous mechanistic explanations being required.

This study is certainly not the first to preach the dangers of ecological fallacy. Other examples include the relationship between dietary fat consumption and risk of breast cancer, using country-level data (Gordis 2009). Messerli (2012) brilliantly highlighted the ecological fallacy phenomenon in an analysis linking national rates of chocolate consumption with cognitive function measured by numbers of Nobel laureates. Banana and beer production are better predictors than climate change for rates of frog extinctions in South America (Rohr et al. 2008). And, for tick-borne diseases, Nadelman and Wormser (2005) documented the over-looked relationship between Lyme disease, tick vectors and US presidential elections.

Nonetheless, ecologic fallacies regularly appear in the scientific literature. Correlations, using data aggregated at the county, state or multi-state level, have been used to advance the notion that higher biodiversity (of mammals or reptiles, but not birds) is protective against Lyme disease incidence (Ostfeld and Keesing 2000; Turney et al. 2014); that coyote (*Canis latrans*) abundance and invasion into the eastern USA has exacerbated the Lyme disease epidemic in the northeastern USA (Levi et al. 2012); and that socio-economic status affects exposure to Lyme disease and another tick-borne disease (ehrlichiosis, caused by *Ehrlichia* spp. and transmitted by the lone star tick, *Amblyomma americanum*) (Springer and Johnson 2018). While theoretically interesting, many of these correlations remain untested at the individual level and often only reflect expectations of a theoretical model. This can present a concern if the studies' conclusions are adopted to support particular policy ideas (Randolph and Dobson 2012). For example, are humans exposed to Lyme disease at greater rates while recreating in areas of low biological diversity? Similarly, the mechanisms for the correlations require further investigation. Higher coyote harvests and increased Lyme disease incidence are correlated, and perhaps the relationship is causal (Levi et al. 2012), but there is little

direct evidence showing that higher coyote abundance reduces abundance of foxes, or that higher coyote abundance increases the abundance and activity of small mammal hosts that are reservoirs for the Lyme disease bacterium (Way and White 2013). We have focused here on tick-borne diseases and the dilution effect hypothesis because these topics are our research interests. However, the phenomenon of ecologic fallacy is not limited to tick-borne diseases, or even just disease ecology.

Our framework also demonstrates other disadvantages of analyses of aggregated data: (1) ignoring additional key factors for which data may be less accessible, (2) adopting proxies to describe processes and (3) neglecting relevant complexities. Vector ecology is a key mechanistic factor that ought to be included when examining the Lyme disease system (Arsnoe et al. 2015). Black-legged ticks in the northeast quest higher in vegetation compared to black-legged ticks in the south (Arsnoe et al. 2015); this vector behavior is likely a more important contributor to spatial variation in Lyme disease risk than the abundance of fried chicken restaurants. Yet we ignored vector behavior and ecology in this study. Understanding the relevance of geographic scale and tick abundance and of pathogen prevalence in tick populations remains a major challenge in tick-borne disease epidemiology and ecology.

Similarly, using numbers of fried chicken restaurants, or obesity rates, or density of empty rental housing (Springer and Johnson 2018) doesn't necessarily relate to behavior that is relevant to exposure to vectors. The use of aggregated data means that we do not, and cannot, know whether individuals infected with *B. burgdorferi* eat fried chicken less frequently, are less obese, or live in areas of high densities of empty rental housing.

Furthermore, such correlations should be regarded in broader more complex contexts (Strauss et al. 2016; Kilpatrick et al. 2017). For example, diets containing fried chicken have other consequences that may outweigh possible impacts upon Lyme disease risk, e.g., fried chicken consumption is associated with a higher risk of cardiovascular mortality (Sun et al. 2019).

So, why do ecologists fall prey to ecological fallacy? Partly because free and available data are attractive for initial examination of links between environmental or ecological factors and disease incidence or trends. Indeed, one of us (DS) was thrilled by the statistically significant relationship between *B. burgdorferi* prevalence in squirrels and human incidence of Lyme disease at a county level, and used this correlation to support the argument that western gray squirrels are Lyme disease reservoirs in California (Salkeld et al. 2008). In addition, aggregated data can allow a greater spatial scale and sample size for analyses, compared to fieldwork that is limited, logistically, to fewer study sites (Springer and Johnson 2018).

However, we use the data and analyses here to demonstrate that statistical relationships at a macro-level cannot be assumed to mirror statistical relationships at a micro-level (Pollet et al. 2014; Halliday and Rohr 2019). Unless the relationships are ground-truthed at individual levels, and the mechanisms carefully described, these correlations should at best be considered supportive, and more often be regarded as speculative. We would expect that spurious correlations arising from ecologic fallacies will eventually be exposed and overturned by the scientific process, but associated costs are incurred when further research is required, or when findings are prematurely adopted for policy and management. Our case study of Lyme disease, fried chicken and obesity is intended to remind researchers to avoid making inappropriate analyses in the first place. Not for the first time, we advocate interdisciplinary collaborations involving epidemiologists, conservationists, public health agencies and ecologists for furthering an understanding of the ecology and epidemiology of infectious zoonoses (Salkeld et al. 2015b).

Acknowledgements

We wish to thank Anne Kjemtrup, Lynne Gaffikin, James Holland Jones and Mevin Hooten for fierce insights into ecology and epidemiology and especially Jean Tsao for her brilliant offhand remark that inspired this paper. We thank Virginie Millien for providing data and W. Tanner Porter for creating maps and Bay Area Lyme Foundation for financial support. This work is dedicated to the memory of Nate Nieto—a brilliant friend and scientist, one of the best, and loved to bits.

References

Arsnoe IM, Hickling GJ, Ginsberg HS, McElreath R, Tsao JI (2015) Different populations of blacklegged tick nymphs exhibit differences in questing behavior that have implications for human Lyme disease risk. *PLoS ONE* 10:e0127450

Bickel PJ, Hammel EA, O'Connell JW (1975) Sex bias in graduate admissions: data from Berkeley. *Science* 187:398–404

Civitello DJ, Cohen J, Fatima H, Halstead NT, Liriano J, McMahon T, Ortega CN, Sauer EL, Sehgal T, Young S, Rohr JR

(2015) Biodiversity inhibits parasites: broad evidence for the dilution effect. *Proceedings National Academy Sciences, USA* 112:8667–8671

Eisen RJ, Eisen L, Beard CB (2016) County-scale distribution of *Ixodes scapularis* and *Ixodes pacificus* (Acari: Ixodidae) in the continental United States. *Journal of Medical Entomology* 53:349–386

Eisen RJ, Eisen L (2018) The blacklegged tick, *Ixodes scapularis*: an increasing public health concern. *Trends in Parasitology* 34:295–309

Forrester JD, Meiman J, Mullins J, Nelson R, Ertel S-H, Cartter M, Brown CM, Lijewski V, Schiffman E, Neitzel D, Daly ER, Mathewson AA, Howe W, Lowe LA, Kratz NR, Semple S, Backenson PB, White JL, Kurpiel PM, Rockwell R, Waller K, Johnson DH, Steward C, Batten B, Blau D, DeLeon-Carnes M, Drew C, Muehlenbachs A, Ritter J, Sanders J, Zaki SR, Molins C, Schriefer M, Perea A, Kugeler K, Nelson C, Hinckley A, Mead P (2014) Update on Lyme carditis, groups at high risk, and frequency of associated sudden cardiac death – United States. *Morbidity and Mortality Weekly Report (MMWR)* 63:982–983

Gascon M, Zijlema W, Vert C, White MP, Nieuwenhuijsen MJ (2017) Outdoor blue spaces, human health and well-being: A systematic review of quantitative studies. *International Journal of Hygiene and Environmental Health* 220:1207–1221

Gordis L (2009) *Epidemiology*, 4th edn., Philadelphia PA: Saunders Elsevier, p 375

Gotway CA, Young LJ (2002) Combining incompatible spatial data. *Journal of the American Statistical Association* 97:632–648

Halliday FW, Rohr JR (2019) Measuring the shape of the biodiversity-disease relationship across systems reveals new findings and key gaps. *Nature Communications* 10:5032

Hickman D, Jones MK, Zhu S, Kirkpatrick E, Ostrov DA, Wang X, Ukhanova M, Sun Y, Mai V, Salemi M, Karst SM (2014) The effect of malnutrition on norovirus infection. *mBio* 5(2):e01032–13

Keesing F, Belden LK, Daszak P, Dobson A, Harvell CD, Holt R, Hudson P, Jolles A, Jones KE, Mitchell CE, Myers SS, Bogich T, Ostfeld RS (2010) Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature* 468:647–652

Kilpatrick AM, Salkeld DJ, Titcomb G, Hahn MB (2017) Conservation of biodiversity as a strategy for improving human health and well-being. *Philosophical Transactions of the Royal Society, London B.* 372:20160131

Levi T, Kilpatrick AM, Mangel M, Wilmers CC (2012) Deer, predators, and the emergence of Lyme disease. *Proceedings National Academy Sciences, USA* 109:10942–10947

LoGiudice K, Ostfeld RS, Schmidt KA, Keesing F (2003) The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proceedings National Academy Sciences, USA* 100:567–571

Messerli FH (2012) Chocolate consumption, cognitive function, and Nobel laureates. *New England Journal of Medicine* 367:1562–1564

Millins C, Gilbert L, Johnson P, James M, Kilbride E, Birtles R, Biek R (2016) Heterogeneity in the abundance and distribution of *Ixodes ricinus* and *Borrelia burgdorferi* (sensu lato) in Scotland: implications for risk prediction. *Parasites and Vectors* 9:595

Nadelman RB, Wormser G (2005) Poly-ticks: blue state versus red state for Lyme disease. *Lancet* 356:280

Nelson CA, Saha S, Kugeler KJ, Delorey MJ, Shankar MB, Hinckley AF, Mead PS (2015) Incidence of clinician-diagnosed Lyme disease, United States, 2005–2010. *Emerging Infectious Diseases* 21:1625–1631

Ogden NH, Tsao JI (2009) Biodiversity and Lyme disease: dilution or amplification. *Epidemics* 1:196–206

Ostfeld RS, Keesing F (2000) Biodiversity and disease risk: the case of Lyme disease. *Conservation Biology* 14:722–728

Pepin KM, Eisen RJ, Mead PS, Piesman J, Fish D, Hoen AG, Barbour AG, Hamer S, Diuk-Wasser MA (2012) Geographic variation in the relationship between human Lyme disease incidence and density of infected host-seeking *Ixodes scapularis* nymphs in the Eastern United States. *American Journal of Tropical Medicine and Hygiene* 86:1062–1071

Pollet TV, Tybur JM, Frankenhuis WE, Rickard IJ (2014) What can cross-cultural correlations teach us about human nature? *Human Nature* 25:410–429

Pollet TV, Stulp G, Henzi SP, Barrett L (2015) Taking the aggravation out of data aggregation: a conceptual guide to dealing with statistical issues related to the pooling of individual-level observational data. *American Journal of Primatology* 77:727–740

Pongsiri MJ, Roman J, Ezenwa VO, Goldberg TL, Koren HS, Newbold SC, Ostfeld RS, Pattanayak SK, Salkeld DJ (2009) Biodiversity loss and the new global disease ecology. *BioScience* 59:945–954

Porter WT, Motyka PJ, Wachara J, Barrand ZA, Hmood Z, McLaughlin M, Pemberton K, Nieto NC (2019) Citizen science informs human-tick exposure in the Northeastern United States. *International Journal of Health Geographics* 18:9

R Core Team (2014). *R: A language and environment for statistical computing*. Vienna: R Foundation for Statistical Computing. http://www.R-project.org/

Randolph SE, Dobson ADM (2012) Pangloss revisited: a critique of the dilution effect and the biodiversity-buffers-disease paradigm. *Parasitology* 16:1–17

Rohr JR, Raffel TR, Romansic JM, McCallum H, Hudson PJ (2008) Evaluating the links between climate, disease spread, and amphibian declines. *Proceedings National Academy Sciences, USA* 105:17436–17441

Salkeld DJ, Leonhard S, Girard YA, Hahn N, Mun J, Padgett KA, Lane RS (2008) Identifying the reservoir hosts of the Lyme disease spirochete *Borrelia burgdorferi* in California: the role of the western gray squirrel (*Sciurus griseus*). *American Journal of Tropical Medicine and Hygiene* 79:535–540

Salkeld DJ, Padgett KA, Jones JH (2013) A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. *Ecology Letters* 16:679–686

Salkeld DJ, Nieto NC, Carbajales-Dale P, Carbajales-Dale M, Cinkovich SS, Lambin EF (2015) Disease risk and landscape attributes of tick-borne *Borrelia* pathogens in the San Francisco Bay Area. *California. PLoS ONE* 10(8):e0134812

Salkeld DJ, Padgett KA, Jones JH, Antolin MF (2015) Public health perspective on patterns of biodiversity and zoonotic disease. *Proceedings National Academy Sciences, USA* 112:E6261

Salkeld DJ, Porter WT, Loh SM, Nieto NC (2019) Time of year and outdoor recreation affect human exposure to ticks in California, United States. *Ticks and Tick-borne Diseases* 10:1113–1117

Schiffman EK, McLaughlin C, Ray JAE, Kemperman MM, Hinckley AF, Friedlander HG, Neitzel DF (2018) Underreporting of Lyme and other tick-borne diseases in residents of a

high-incidence county, Minnesota, 2009. *Zoonoses Public Health* 65:230–237

Shortridge BG (2005) Apple stack cake for dessert: Appalachian regional foods. *Journal of Geography* 104:65–73

Springer YP, Johnson PTJ (2018) Large-scale health disparities associated with Lyme disease and human monocytic ehrlichiosis in the United States, 2007-2013. *PLoS ONE* 13(9):e0204609

Stanek G, Wormser GP, Strle F (2012) Lyme borreliosis. *Lancet* 379:461–473

Strauss AT, Shocket MS, Civitello DJ, Hite JL, Penczykowski RM, Duffy MA, Cáceres CE, Hall SR (2016) Habitat, predators, and hosts regulate disease in *Daphnia* through direct and indirect pathways. *Ecological Monographs* 86:393–411

Sun Y, Liu B, Snetselaar LG, Robinson JG, Wallace RB, Peterson LL, Bao W (2019) Association of fried food consumption with all cause, cardiovascular, and cancer mortality: prospective cohort study. *The BMJ* 364:k5420

Turney S, Gonzalez A, Millien V (2014) The negative relationship between mammal host diversity and Lyme disease incidence strengthens through time. *Ecology* 95:3244–3250

Way JG, White BN (2013) Coyotes, red foxes, and the prevalence of Lyme disease. *Northeastern Naturalist* 20:655–665

Webb P, Bain C (2011) *Essential epidemiology: an introduction for students and health professionals*, 2nd ed., Cambridge: University Press, p 445

Wilkinson DA, Marshall JC, French NP, Hayman DTS (2018) Habitat fragmentation, biodiversity loss and the risk of novel infectious disease emergence. *Journal of the Royal Society Interface* 15:20180403

Wood CL, Lafferty KD (2013) Biodiversity and disease: a synthesis of ecological perspectives on Lyme disease transmission. *Trends in Ecology and Evolution* 28:239–247