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Are we overestimating risk of enteric pathogen spillover from wild birds to humans?

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ABSTRACT

Enteric illnesses remain the second largest source of communicable diseases worldwide, and wild birds are suspected sources for human infection. This has led to efforts to reduce pathogen spillover through deterrence of wildlife and removal of wildlife habitat, particularly within farming systems, which can compromise conservation efforts and the ecosystem services wild birds provide. Further, *Salmonella* spp. are a significant cause of avian mortality, leading to additional conservation concerns. Despite numerous studies of enteric bacteria in wild birds and policies to discourage birds from food systems, we lack a comprehensive understanding of wild bird involvement in transmission of enteric bacteria to humans. Here, we propose a framework for understanding spillover of enteric pathogens from wild birds to humans, which includes pathogen acquisition, reservoir competence and bacterial shedding, contact with people and food, and pathogen survival in the environment. We place the literature into this framework to identify important knowledge gaps. Second, we conduct a meta-analysis of prevalence data for three human enteric pathogens, *Campylobacter* spp., *E. coli*, and *Salmonella* spp., in 431 North American breeding bird species. Our literature review revealed that only 3% of studies addressed the complete system of pathogen transmission. In our meta-analysis, we found a *Campylobacter* spp. prevalence of 27% across wild birds, while prevalence estimates of pathogenic *E. coli* (20%) and *Salmonella* spp. (6.4%) were lower. There was significant bias in which bird species have been tested, with most studies focusing on a small number of taxa that are common near people (e.g. European starlings *Sturnus vulgaris* and rock pigeons *Columba livia*) or commonly in contact with human waste (e.g. gulls). No pathogen prevalence data were available for 65% of North American breeding bird species, including many commonly in contact with humans (e.g. black-billed magpie *Pica hudsonia* and great blue heron *Ardea herodias*), and our metadata suggest that some under-studied species, taxonomic groups, and guilds may represent equivalent or greater risk to human infection than heavily studied species. We conclude that current data do not provide sufficient information to determine the likelihood of enteric pathogen spillover from wild birds to humans and thus preclude management solutions. The primary focus in the literature on pathogen prevalence likely overestimates the probability of enteric pathogen spillover from wild birds to humans because a pathogen must survive long enough at an infectious dose and be a strain that is able to colonize humans to cause infection. We propose that future research should focus on the large number of under-studied species commonly in contact with people and food production and demonstrate shedding of bacterial strains pathogenic to humans into the environment where people may contact them. Finally, studies assessing the duration and intensity of bacterial shedding and survival of bacteria in the environment in bird faeces will help provide crucial missing information necessary to calculate spillover probability. Addressing these essential knowledge gaps will support policy to reduce enteric pathogen spillover to humans and enhance bird conservation efforts that are currently undermined by unsupported fears of pathogen spillover from wild birds.

Key words: agroecology, *Campylobacter* spp., *E. coli*, enteric illness, food safety, *Salmonella* spp., wild birds

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I. INTRODUCTION

Enteric pathogens cause millions of illnesses and hundreds of thousands of deaths worldwide each year (Scallan *et al.*, 2011; Batz, Hoffmann & Morris, 2012; Havelaar *et al.*, 2015). The majority of these cases involve three enteric bacteria – *Salmonella* spp., *Escherichia coli*, and *Campylobacter* spp. – that originate in human, livestock, or wildlife waste (Havelaar *et al.*, 2015). Among wildlife, birds have been the focus of many studies concerning enteric pathogens for several reasons. First, *Salmonella* spp. can cause mass die-offs of songbirds among other taxa, causing large conservation concerns (Fichtel, 1978; Daoust *et al.*, 2000; Tizard, 2004; Hall & Saito, 2008). Hall & Saito (2008) estimated that *Salmonella* spp. were involved in 21.5% of passerine and 5.4% of total bird mortality events in the United States from 1985 to 2004. Second, wild birds are highly mobile and can carry pathogens across large distances, especially during migration, which produces a risk of spreading pathogens beyond local outbreaks (Hussong *et al.*, 1979; Altizer, Bartel & Han, 2011; Gardner *et al.*, 2011; Callaway, Edrington & Nisbet, 2014). For example, sandhill cranes (*Grus canadensis*), which are commonly infected with *Campylobacter* spp. during migration, increase enteric bacteria levels in water bodies where they forage (Pacha *et al.*, 1988; Lu *et al.*, 2013; Vogel *et al.*, 2013). Indeed, the only foodborne illness outbreak traced back to a wild bird source occurred from migrating sandhill cranes stopping over at a pea farm in Alaska (Gardner *et al.*, 2011). Third, wild birds are extremely abundant across human-inhabited landscapes (e.g. urban landscapes, agroecosystems), potentially leading to high contact rates with people and food.

Wild birds are thought to transmit enteric pathogens to humans *via* several routes. First, people may contact wild birds directly through hunting and consuming the contaminated meat (Navarro-Gonzalez *et al.*, 2016) or *via* intentional interactions with birds (e.g. feeding geese at a park). For example, many studies test carcasses of hunted waterfowl or gamebirds and often find high enteric pathogen prevalence (number positive/number tested) (e.g. Luechtefeld *et al.*, 1980; Nebola, Borilova & Steinhauserova, 2007), which may be consumed and cause infection if improperly prepared (Navarro-Gonzalez *et al.*, 2016). Perhaps more commonly, people may contact surfaces contaminated with faeces from wild birds in locations where birds aggregate (e.g. parks, playgrounds, and beaches) (Strachan *et al.*, 2013; Abdollahpour *et al.*, 2015; Cody *et al.*, 2015). Canada geese (*Branta canadensis*) can reach high densities at urban parks or beaches where people, particularly young children, may contact faeces either directly with hands or indirectly with clothing or toys (Feare *et al.*, 1999). Children, in particular, are more likely to then place hands in mouths and ingest enteric bacteria (Feare *et al.*, 1999; Strachan *et al.*, 2013). Wild birds may also contaminate drinking, irrigation, or recreational water. An investigation of an *E. coli* O157:H7 outbreak at Battle Ground Lake in Washington State, USA found identical pulsed-field gel electrophoresis/restriction fragment length polymorphism patterns between isolates from wild duck faeces, water samples, and case patients, suggesting the ducks may have introduced the bacteria to the recreational water (Samadpour *et al.*, 2002). Finally, wild birds can infect livestock or defecate on crops, leading to foodborne illness (Carlson *et al.*, 2011; Gardner *et al.*, 2011).

To date, most literature has focused on a small number of bird species in a narrow range of habitat settings [e.g. European starlings (*Sturnus vulgaris*) and rock pigeons (*Columba livia*) in cities or intensified livestock operations (Carlson *et al.*, 2011, 2015; Haesendonck *et al.*, 2016; Marenzoni *et al.*, 2016)]. Conversely, few data are available for the majority of wild bird species, including many of those commonly found in contact with people or agriculture [e.g. American robins (*Turdus migratorius*)]. Further, most studies provide data limited to enteric pathogen prevalence (proportion of individuals infected) and not transmission *per se* (movement of the pathogen). Indeed, a systematic review of 442 modelling studies covering 85 zoonotic pathogens conducted by Lloyd-Smith *et al.* (2009) found that disease ecology literature often fails to account for multi-host ecology of pathogens, with only six studies examined including a mechanistic model of zoonotic spillover. This constrains our ability conclusively to identify sources of pathogens and weakens assessment of risks that wildlife, including birds, pose to human health. For example, mallard ducks (*Anas platyrhynchos*) often have high prevalence of *Campylobacter* spp. [e.g. 9.2–52.2% in Colles *et al.*, 2011 and 34% in Luechtefeld *et al.*, 1980]. Yet, experimental infection data suggest *Campylobacter* spp. are highly host-adapted, and mallards are poor reservoir hosts for non-mallard strains (Atterby *et al.*, 2018). In fact, Colles *et al.* (2011) found only 1 of 109 *Campylobacter* isolates from wild mallard ducks were a sequence type associated with human disease, suggesting a low risk of transmission despite high prevalence. Further, the few studies that attempt to trace human cases to their origin suggest that although prevalence may be high, crossover is rare (Strachan *et al.*, 2013; Cody *et al.*, 2015; Seguino *et al.*, 2018). For example, Seguino *et al.* (2018) found that wild bird isolates accounted for only 0.23% of human *C. jejuni* and *C. coli* infections. Thus, reliance on prevalence data alone may be overestimating the risk of enteric pathogen spillover between wild birds and humans.

Although many reviews at least briefly discuss enteric pathogen transmission between wildlife, livestock, and/or humans (e.g. Hancock *et al.*, 1998; Haag-Wackernagel & Moch, 2004; Hubálek, 2004; Tizard, 2004; Wassenaar, 2011; Clark, 2014; Navarro-Gonzalez *et al.*, 2016), they lack a robust framework from which to develop future risk models (e.g. frameworks provided by Plowright *et al.*, 2017; Cross *et al.*, 2019; Washburne *et al.*, 2019). Further, the lack of systematic or meta-analytic approaches used in prior syntheses could lead to erroneous conclusions about pathogen prevalence and transmission if the narrow subset of species and habitats considered are subject to selection bias and are not representative of broader trends (Haddaway & Watson, 2016). Therefore, to establish a better understanding of the relationship between wild birds and human enteric illness, we developed a framework for understanding transmission, which has largely been ignored in the literature (Lloyd-Smith *et al.*, 2009), and summarize what is currently known throughout. Our conceptual framework builds upon those provided by Lloyd-Smith *et al.* (2009) and Plowright *et al.* (2017) and captures the complex processes involved in regulating the spillover of enteric pathogens from wild birds to humans, including pathogen exposure, reservoir competence, contact with people or food, bacterial survival in the environment, and transmission to human hosts. Second, we conducted a comprehensive meta-analysis of enteric pathogen prevalence in 431 North American breeding birds that focused on three enteric pathogens with large human health burdens known to occur frequently in wild birds: *Campylobacter* spp., *E. coli*, and *Salmonella* spp. We conducted this meta-analysis using prevalence data since it is the most commonly reported proxy for transmission risk. Throughout our conceptual framework and meta-analysis, we assess ideas about biological and ecological variables that may affect risk that are commonly found throughout the literature (e.g. juvenile birds will have higher prevalence because of adaptive immunity). We synthesize our results to identify the most important future research avenues needed to quantify the risk wild birds pose to human health.

II. LITERATURE SEARCH AND ANALYSIS

(1) Literature search

We began by acquiring papers concerning aspects of transmission of enteric pathogens from wild birds to livestock and/or humans, most of which focused on prevalence data. We searched the literature for studies reporting data on *Campylobacter* spp., *E. coli*, and *Salmonella* spp. in North American breeding birds (see online Supporting information, Table S1 and Data S1). First, we gathered a list of North American breeding birds from the USGS Breeding Bird Survey (Sauer *et al.*, 2017) and supplemented this list with any species observed on West Coast farms by Smith *et al.* (2019), which yielded a list of 431 species (Table S2 and Data S2). We assigned each bird species to a taxonomic order and family using the Birds of North America online database taxonomy as of November 2018 (Table S2; Data S2; Rodewald, 2015). We then assigned each species to a diet guild and foraging strata using De Graaf, Tilghman & Anderson (1985), Rodewald (2015), and Wilman *et al.* (2014). We next searched the ISI *Web of Knowledge* for studies reporting the presence of *Campylobacter* spp., *E. coli*, and/or *Salmonella* spp. or other aspects of transmission for each North American breeding bird species and saved all review papers acquired through the search. Our search terms included "'Salmonel*" OR "E* coli" OR "Campylobacter" AND "[bird common name]" OR "[bird scientific name]"'. We searched for additional papers in references in all review papers acquired through the search and selected primary publications reporting estimates for understudied species. Due to a lack of data for most species included in our meta-analysis (see Section IV.2), we gathered estimates from studies conducted outside of North America if they included estimates for North American breeding birds (Figs S1 & S2). We had six criteria for inclusion in prevalence analyses in our meta-analysis section. The paper must (1) report if one or more of the 431 North American breeding birds was/were tested for *Campylobacter* spp., *E. coli*, and/or

Salmonella spp., (2) present primary data that were not duplicated from other studies included in the meta-analysis, (3) report the bird species tested (e.g. *Larus* spp. was not sufficient but *Larus argentatus* was), (4) report on natural infections (i.e. no experimental infection data), (5) report data from free-ranging wild birds (we did not include estimates from farm, long-term rehabilitation centre, or laboratory animals for prevalence estimates), and (6) be in English, Spanish, or French or have all data extractable from an English language abstract. We gathered data on generic *E. coli* when available but considered it an unsuitable proxy for pathogenic *E. coli* and marked it 'Reject (7)' in our study log (Data S1). Data were further considered unsuitable for generating pathogen prevalence estimates but suitable for reporting presence/absence of bacteria if they: (8) did not report the number of individuals tested or positive (including only reporting number of isolates) or (9) only reported data on birds collected after death to avoid overestimating prevalence in birds that died from enteric pathogens (excluding hunted birds which we assumed to be a random sample of wild bird populations), or were brought to a rehabilitation centre within 24 h of testing to avoid overestimating prevalence due to infections acquired after capture or underestimating prevalence due to treatment. A total of 211 papers fitted our full nine criteria for inclusion (Figs S1 and S2; Table S1; Data S1).

We gathered binary data from each study on 30 variables we classified as related to exposure ($N = 6$ variables), reservoir competence ($N = 14$ variables), contact with humans or food ($N = 4$ variables), or bacterial survival and transmission ($N = 6$ variables); bacterial species included; prevalence; substance tested; condition (live, sick, etc.) at testing; bacterial identification method(s); habitat setting(s) of study; and geographical location (Table S1; Data S1). For each study and species reported, we gathered data on number of individuals that tested positive, total number of individuals tested, and whether anti-bacterial resistance was reported (Tables S2 and S3; Data S2 and S3). If *Campylobacter* spp. or *Salmonella* spp. serovar (bacterial groups with unique cell surface antigen variants) were reported, we recorded how many individuals had each species or serovar. *E. coli* were separated into pathogenic and generic forms.

(2) Statistical analysis

(*a*) *Meta-regressions*

We estimated pathogen prevalence in two ways. First, we estimated prevalence for each bacterium in each bird species by summing the total number of individuals with positive samples divided by the total number of individuals tested across studies (Data S2). We then estimated overall prevalence by summing number of positive individuals/number of individuals tested for each pathogen. Second, we estimated pathogen prevalence using random effects models in the rma.mv function in the metafor package in R (Viechtbauer, 2010; R Core Team, 2018) for individual bird species. We included study as a random effect and estimated pathogen prevalence across bird species by including study and species nested within family as random effects. We only estimated species prevalence using random effects models when data came from two or more studies and we estimated that sufficient observations were available based on the Thrusfield (2007) formula. To estimate if sufficient observations were available with the Thrusfield (2007) formula, we assumed an infinite study population, used the expected overall pathogen prevalence calculations described above, used a confidence interval of 95%, and used 5% desired absolute precision. In the main text we present prevalence estimates derived from models including study as a random effect, while also providing estimates calculated by summing across studies in the supporting information.

We tested for differences in pathogen prevalence by sex and age using log risk ratios in the escalc function in the metafor package. We tested for differences in prevalence by age for *Campylobacter* spp., pathogenic *E. coli*, and *Salmonella* spp., but limited analyses on sex to *Salmonella* spp. due to data availability (Tables S4 and S5). Next, we compared the prevalence of *Salmonella* spp. for three species with the most estimates across studies (European starling, house sparrow (*Passer domesticus*), and rock pigeon, all introduced to North America and, therefore, not protected) by type of sample tested for bacteria [cloacal swab, faeces, blood, and dissected internal organs ('necropsy')] using mixed-effects models in the rma.mv function in the metafor package including study as a random effect. We conducted pairwise comparisons using Tukey HSD tests. We hypothesized that studies that tested internal organs would find higher prevalence of pathogens because a bird would not have to be shedding bacteria in order to obtain a positive result; this, in turn, may erroneously suggest that commonly necropsied birds have higher prevalence than protected natives that generally cannot be necropsied. Finally, we conducted comparisons of pathogen prevalence by order, diet guild, and foraging strata using mixed-effects models in the rma.mv function in the metafor package in R, followed by Tukey HSD tests for pairwise differences. Study and species nested within family were used as random effects to account for multiple observations from some studies and taxonomic relatedness, respectively. We suggest caution in interpreting P values from our analyses for two reasons. First, the data available are largely biased to a small number of commonly studied species (see Section IV.2) that may not be representative of most wild birds. Second, some pathogen–bird combinations have very few observations compared to others. Thus, lack of statistical differences in pathogen prevalence between some bird species or groups might often reflect low power, based on small sample sizes, rather than no differences in underlying biology. Conversely, data on *Salmonella* spp. prevalence were comparatively common in the literature, giving us greater power to detect differences.

(*b*) *Representativeness of the literature*

We evaluated representation of the wild bird species studied across the literature using a two-part comparison. First, we compared the taxonomic orders and species studied for each

pathogen to the percentage of species each taxon represented in the North American Breeding Bird Survey list of reported birds (Sauer *et al.*, 2017). Second, we searched the eBird database for reported sightings of each species as a measure of relative abundance (Sullivan *et al.*, 2009). Then, using estimated prevalence and the minimal sample size needed to determine prevalence with 5% precision, we classified species into those with no pathogen observations, those with 1+ observations but insufficient numbers to determine prevalence for any pathogens, and those with enough data to determine prevalence for one, two, or three of the pathogens. We then calculated the percentage of species falling into each category. Finally, we summed the total sightings in eBird of species in each category to calculate the relative abundances of individuals in each group. For the second comparison, we accessed a farm bird database from Smith *et al.* (2019) that surveyed birds on 52 small-scale, diversified organic farms (23 that integrated livestock and 29 crop-only) and two cattle feedlots. Organic produce farming is often thought to be a hotspot of enteric pathogen transmission, and increasingly, farmers are encouraged to remove wildlife habitat from their farms (Beretti & Stuart, 2008). Adhering to these Good Agricultural Practice recommendations can be extremely cost prohibitive to small-scale growers (Bovay & Sumner, 2018). Therefore, understanding risk of enteric pathogen spillover within this system is particularly important. We compared our meta-data to the percentage of species in each taxon represented within this farm population and the average on-farm densities each taxon represented. We repeated our analyses described above for percent of North American breeding bird species and relative abundances using eBird with the farm bird data and classified farm bird species into those with no pathogen observations, those with 1+ observations but insufficient numbers to determine prevalence for any pathogens, and those with enough data to determine prevalence for one, two, or three of the pathogens. We compared the proportion of observations of each species for each of the four comparisons (North American breeding birds, eBird abundances, farm bird species, and farm bird abundances) with their proportions in the data collected for the meta-analysis using Chi-square goodness-of-fit tests.

III. CONCEPTUAL FRAMEWORK FOR UNDERSTANDING SPILLOVER OF ENTERIC PATHOGENS FROM WILD BIRDS TO HUMANS

Lloyd-Smith *et al.* (2009) developed a framework for understanding zoonotic spillover that included prevalence of infection in animal reservoirs, the rate of human contact with reservoirs, and the probability that humans become infected when contact occurs. Plowright *et al.* (2017) expanded upon these ideas, noting that a hierarchical series of barriers must align for spillover to occur. The framework of Plowright *et al.* (2017) included pathogen pressure (determined by reservoir distribution, pathogen prevalence, and pathogen release), human and vector behaviour leading to route and dose of exposure, and attributes of recipient hosts which affect the probability and severity of infection (genetics, physiological, and immunological factors). Here, we expand these frameworks to create a wild bird–enteric pathogen-specific framework for understanding the factors that influence the likelihood of enteric pathogens contacting and infecting humans (Fig. 1A). To accomplish this, we consider factors that influence wild bird exposure to enteric pathogens, reservoir competence, contact with humans and food, and probability of pathogens surviving in the environment, colonizing, and causing disease in a human host. Briefly, for wild birds to become transporters or reservoirs of a pathogen, they must first be exposed to bacteria in the environment. Exposure may vary based on ecological traits, including habitat associations and foraging traits. At the same time, an individual's susceptibility to being colonized by a pathogen will vary based on reservoir competence, which can differ based on a number of physiological factors. Lastly, for enteric pathogens to be transmitted from wild birds to humans, humans must either come into direct contact with the pathogens (e.g. from wild bird meat or faeces through direct hand-to-mouth contact) or indirectly from crops or water contaminated with faeces. If directly consumed, the bacteria must be a strain that can effectively colonize and cause disease in a human host and be ingested at an infectious dose. If indirectly consumed, the bacteria must also survive in the environment and through food preparation for long enough to remain at an infectious dose. Many factors influence each stage of the pathogen life cycle and the subsequent likelihood of wild birds transmitting enteric pathogens that may cause disease in humans. We describe these processes in more detail below. We suggest that research approaches that integrate all four stages will be most informative, and studies that only consider prevalence are likely to overestimate risk of enteric pathogen spillover from wild birds to humans.

(1) Exposure

Wild birds must come into contact with bacteria in the environment to be colonized by enteric pathogens. If pathogen prevalence varies by landscape context, habitat associations may make species that tend to inhabit pathogen-sparse landscapes less likely to encounter pathogens than species that inhabit pathogen-rich landscapes (Taff *et al.*, 2016). Within a species, individuals that inhabit pathogen-rich *versus* pathogen-poor landscapes may experience different exposure levels and have variable pathogen prevalence and shedding intensity (Barron *et al.*, 2015; Taff *et al.*, 2016). Several landscape contexts are thought to be hotspots for transmission. A large body of literature has demonstrated transmission of enteric pathogens from refuse sites to gull and corvid species, including *Salmonella* serovars known to infect humans (Butterfield *et al.*, 1983; Ito, Totake & Ogawa, 1988; Tizard, 2004). Wild birds can also acquire and transmit bacteria in water bodies (Levesque *et al.*, 2000; Fogarty *et al.*, 2003; Lu *et al.*, 2013). Species that interact with livestock

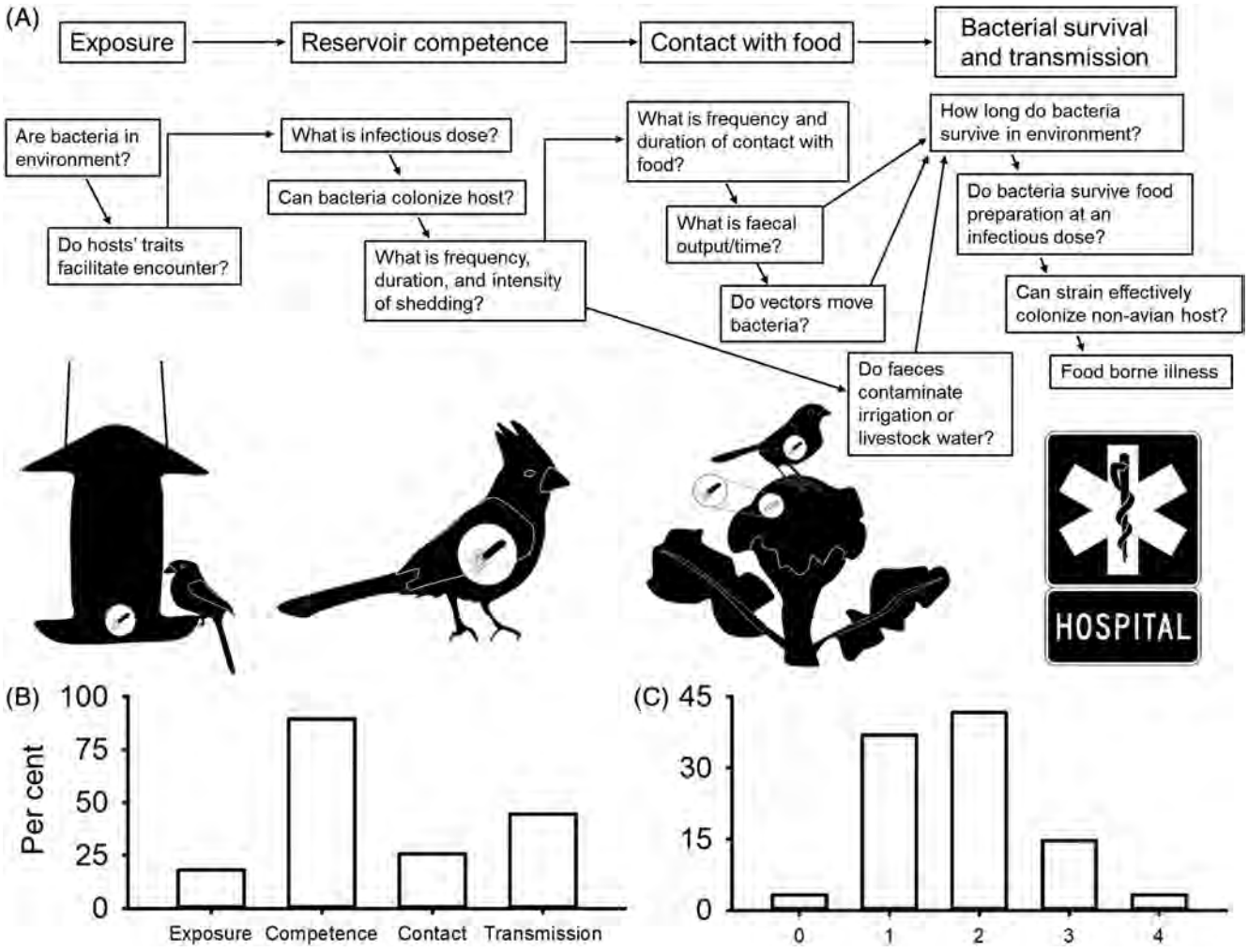

Fig. 1 (A) Conceptual diagram outlining steps from bacterial acquisition to human infection. Icons below flow chart show a bird being exposed to *E. coli* at a bird feeder, *E. coli* replicating within a bird host, a bird defecating *E. coli* on a broccoli plant, and a hospital sign to indicate human enteric illness. (B) Percentage of studies included in meta-analysis that reported data pertaining to exposure, reservoir competence, contact, and bacterial survival and transmission. (C) Percentage of studies that reported data on 0, 1, 2, 3, or 4 of the aspects in the conceptual diagram (exposure, reservoir competence, contact, and bacterial survival and transmission).

are further thought to have high prevalence of enteric pathogens (Skov *et al.*, 2008; Carlson *et al.*, 2011; Callaway *et al.*, 2014; Hald *et al.*, 2016). Birds foraging close to livestock have been demonstrated to have higher prevalence of *Campylobacter* spp. than those foraging further away (Hald *et al.*, 2016). Proximity to urban habitats (Hernandez *et al.*, 2016) and the use of bird feeders (Fichtel, 1978; Daoust *et al.*, 2000; Tizard, 2004) are also thought to increase prevalence. Hernandez *et al.* (2016) found *Salmonella* spp. prevalence was highest in white ibis (*Eudocimus albus*) in urban landscapes and decreased in natural landscapes. Conversely, neither Rouffaer *et al.* (2016), Brobey, Kucknoor & Armacost (2017), nor Hamer, Lehrer & Magle (2012) found variation in *Salmonella* spp. prevalence across an urbanization gradient. However, these studies primarily tested songbirds which have low *Salmonella* spp. prevalence (~4.8%; see below) compared to the Pelecaniformes (egrets, ibis, pelicans; ~17%), limiting ability to make landscape comparisons. It is generally thought that species such as finches and sparrows that gather at high densities at feeders are prone to mass mortality following contamination of a feeding station with *Salmonella* spp., especially in harsh weather conditions when birds are forced to aggregate for food (Fichtel, 1978; Daoust *et al.*, 2000; Tizard, 2004). A review conducted by Brearley *et al.* (2013) found fairly inconsistent results in the impacts of human-modified land usage on pathogen prevalence and suggested one reason may be the need to consider habitat fragmentation in addition to its loss (Allan, Keesing & Ostfeld, 2003; Brearley *et al.*, 2013), which would be a novel approach in assessing how land usage influences enteric pathogen prevalence in wild birds.

Foraging traits may alter enteric pathogen prevalence in wild birds by altering rates of exposure to faecal contamination (Waldenström *et al.*, 2002; Skov *et al.*, 2008; Hald *et al.*, 2016). Waldenström *et al.* (2002) tested *Campylobacter* spp.

prevalence in 1794 migrating birds and found shoreline-foraging birds, opportunistic feeders, and non-granivorous ground-foragers had the highest prevalence. A study conducted in Danish livestock farms that tested 1607 individuals found that birds whose diets consisted primarily of animals or mixed animals and vegetables, those foraging on the ground, and those foraging near livestock stables were more likely to carry *Campylobacter* spp. than aerial foragers and other guilds (Hald *et al.*, 2016). Similarly, Sensale *et al.* (2006) found higher *Campylobacter* spp. prevalence in ground foragers and arboreal/herbaceous insectivores and no *Campylobacter* spp. in granivores or aerial insectivores. Interestingly, Broman *et al.* (2004) found *C. jejuni* strains exhibited high similarities within foraging guilds, suggesting shared sources of transmission. Our literature review yielded no studies that compared *E. coli* or *Salmonella* spp. prevalence by foraging guild. We examine the relationship between diet guild and foraging strata using our meta-data (see Section IV.4).

Daily and seasonal movements may also impact exposure rates and ability to disseminate and maintain pathogens at new locations. For example, European starlings are often cited as a risk for food safety (Carlson *et al.*, 2011, 2015) and have large daily movement patterns from roost to feedlot sites. Further, starlings occasionally will forage between multiple feedlots, disseminating pathogens to new herds (Lejeune *et al.*, 2007; Gaukler *et al.*, 2012; Bray, Larsen & Mott, 2018). Birds often stop and forage along the route, including in agricultural fields, where they could contaminate produce with pathogens acquired from the feedlots. Raptors and crows represent other groups with large daily movement patterns (Rodewald, 2015) that may act as disseminators of pathogens into new environments. Studies on species that have large daily movement abilities are of interest and could help elucidate mechanisms that contribute to the introduction and maintenance of pathogens at important contamination points.

Migratory species are generally thought to have high exposure to pathogens during migration, but others have hypothesized that migration could cause individuals to leave pathogen-rich areas and reduce exposure levels (Altizer *et al.*, 2011). Some evidence exists that migration increases enteric pathogen prevalence, particularly for migratory water birds, likely due to large aggregations of birds from across wide areas with high faecal outputs (Hussong *et al.*, 1979; Waldenström *et al.*, 2002; Hubálek, 2004; Skov *et al.*, 2008; Lu *et al.*, 2013). In addition to exposing birds to a variety of pathogens, migratory behaviours can impact prevalence through changes in host physiology, stress, and immune function and can contribute to dissemination of pathogens across large distances (Hubálek, 2004; Altizer *et al.*, 2011; Callaway *et al.*, 2014). Migratory waterfowl and other water birds have long been known to cause seasonal peaks in enteric pathogens when they aggregate in water bodies during migration and at overwintering sites (e.g. Hussong *et al.*, 1979; Lu *et al.*, 2013). Similarly, Taff *et al.* (2016) found *Campylobacter* spp. prevalence was highest in American crows (*Corvus brachyrhynchos*) during winter when migratory individuals return and crows form large communal roosts. Migratory distance can also impact pathogen prevalence: short-distance migrants have been found to have higher prevalence than long-distance migrants for both *Campylobacter* spp. and *Salmonella* spp. (Waldenström *et al.*, 2002; Sensale *et al.*, 2006; Skov *et al.*, 2008). Skov *et al.* (2008) further compared migratory to resident species and found the lowest *Salmonella* spp. prevalence in resident birds. However, results are certainly not ubiquitous as Hald *et al.* (2016) found no correlation between migratory status and *Campylobacter* spp. prevalence.

(2) Reservoir competence

While habitat usage and species traits may alter exposure levels, once an individual is exposed to a pathogen, susceptibility to infection will vary based on reservoir competence. For instance, individuals could function simply as temporary transporters with a low probability of transmitting bacteria or become infected and shed bacteria for prolonged periods, increasing the risk of transmission to new hosts. Here, we define reservoir competence as the probability that an infected host will transmit an infection to a new host (Barron *et al.*, 2015). Reservoir competence is influenced by factors such as exposure, host immune response, shedding intensity, and shedding duration. Sex, age, body size, body condition, microbiome, coinfection (simultaneous infection of a host by multiple pathogen species), pace of life, variation in innate immunity, daily and seasonal movement, and host density, among other factors, could all impact colonization by bacteria, duration of infection, and intensity of shedding (Mills, Lombardo & Thorpe, 1999; Waldenström *et al.*, 2002; Benskin *et al.*, 2009; Colles *et al.*, 2011; Ostfeld *et al.*, 2014; Owen *et al.*, 2014; Taff *et al.*, 2016; Grond *et al.*, 2018). Although microbiome could affect enteric pathogen prevalence in wild birds (Peachey, Jenkins & Cantacessi, 2017; Grond *et al.*, 2018), we found no studies that tested this idea. Microbiome is influenced by physiology, diet, environment, and phase of the annual cycle (Grond *et al.*, 2018), suggesting that species traits may influence pathogen prevalence if a diverse microbial community were to alter pathogen establishment. Coinfection has been demonstrated to decrease (Johnson & Hoverman, 2012; Johnson *et al.*, 2013) or increase infection success (Wang *et al.*, 2018), although results are inconsistent (Peachey *et al.*, 2017; Wang *et al.*, 2018) and could depend on factors such as host immune response (Peachey *et al.*, 2017) or order of infection (Johnson & Hoverman, 2012; Atterby *et al.*, 2018). Twenty-two papers gathered through our literature review reported coinfection data, although their observational nature makes it difficult to make inferences (Table S6). Experiments examining the impacts of microbiome diversity or coinfection on enteric pathogen colonization, shedding intensity, or shedding duration would be novel contributions to the field.

Sex could influence susceptibility to infection if differential parental investment affects condition, immune investment, or alters habitat usage and subsequent exposure (Monaghan *et al.*, 1985; Martin, Weil & Nelson, 2008;

Girard, Goldberg & Hamer, 2011). We tested the impact of sex on *Salmonella* spp. prevalence in 711 female (11.6% prevalence summed across studies) and 985 male (7.9% prevalence summed across studies) individuals from six studies and found females had 1.45 times (95% CI: 1.08, 1.94) higher *Salmonella* spp. prevalence [estimated average log relative risk ($\hat{\mu}$) = 0.37 ± 0.15 (SE), $\mathcal{Z}$ = 2.46, P = 0.014; Fig. S3; Table S4]. Three studies reported *Campylobacter* spp. prevalence estimates by sex, and all found prevalence did not differ by sex but did not report sample sizes needed to conduct analyses across studies. Similarly, studies reporting generic *E. coli* estimates by sex did not report sample sizes needed to conduct analyses but stated results were not significant. One study reported sample sizes of male and female birds tested for pathogenic *E. coli* and found that prevalence was higher in female (14.3%, N = 7) versus male (7.1%, N = 14) birds, but no statistical analysis was conducted.

Age may impact bacterial colonization, intensity, and shedding through differences in acquired immunity (Levesque *et al.*, 2000; Colles *et al.*, 2009, 2011; Taff *et al.*, 2016). We tested the impact of age on *Campylobacter* spp. prevalence from 10 estimates including 1138 juvenile (19.0% prevalence summed across studies) and 1273 adult (35.5% prevalence summed across studies) individuals from seven studies and found juveniles had 1.04 times higher prevalence (95% CI: 0.60, 1.79), but the difference was not significant [$\hat{\mu}$ = 0.036 ± 0.28 (SE), $\mathcal{Z}$ = 0.13, P = 0.90; Fig. S4; Table S5]. We tested the impact of age on pathogenic *E. coli* prevalence from two estimates from two studies including 171 juvenile (18.1% prevalence summed across studies) and 493 adult (8.3% prevalence summed across studies) individuals and found adults had 1.57 times higher prevalence (95% CI: 0.37, 6.68), but the difference was not significant [$\hat{\mu}$ = 0.45 ± 0.74 (SE), $\mathcal{Z}$ = 0.61, P = 0.54; Fig. S5; Table S5]. Two studies reported differences in generic *E. coli* by age but did not report the number of individuals sampled (Table S5). Finally, we tested the impact of age on *Salmonella* spp. prevalence from 13 estimates from 12 studies including 1104 juvenile (13.9% prevalence) and 2946 adult (5.0% prevalence) individuals and found juveniles had 1.91 times higher prevalence [95% CI: 1.17, 3.12; $\hat{\mu}$ = 0.65 ± 0.25 (SE), $\mathcal{Z}$ = 2.57, P = 0.010; Fig. S6; Table S5]. Our finding that juvenile birds have higher prevalence of *Salmonella* spp. supports the hypothesis that acquired immunity may decrease prevalence in wild birds (Levesque *et al.*, 2000; Colles *et al.*, 2009, 2011; Taff *et al.*, 2016). *Salmonella* Typhimurium is known to cause mass mortalities in wild birds (Tizard, 2004; Connolly *et al.*, 2006; Hall & Saito, 2008), but *Campylobacter* spp. are generally thought to be a natural commensal (Wassenaar, 2011; Griekspoor *et al.*, 2013), potentially explaining our finding that age does not appear to influence *Campylobacter* spp. prevalence but it does appear to influence *Salmonella* spp. prevalence.

Poor body condition may increase susceptibility to infection, though current evidence is mixed (Espinosa-Arguelles *et al.*, 2010; Fukui *et al.*, 2014; Sánchez *et al.*, 2018). We found 36 estimates from 15 studies that attempted to relate condition metrics to enteric pathogen prevalence (Table S7). Twenty-one estimates were reported for *Campylobacter* spp. prevalence. Seven of these estimates were reported as significant: four reported a negative effect (combined condition index, mass, and wing cord) while three reported a positive effect (body score, tarsus length, and mass). The remaining 14 were non-significant (combined condition indices, skeletal size, metatarsus length, tarsus asymmetry and length, left wing length, mass, fat score, packed cell volume, wing ectoparasites). One study reported estimates for pathogenic *E. coli* (STEC) and found mass was negatively correlated with prevalence in juvenile/subadult rock pigeons but was not correlated with prevalence in adults. One study reported generic *E. coli* prevalence in relation to European starling mass and found no effect. Five studies related *Salmonella* spp. prevalence to mass, three of which concerned Salmonellosis cases. One of the two studies conducted on free-living populations found a significant negative relationship while the other found no effect. All three studies concerning Salmonellosis cases found negative relationships between infection and mass, although only one conducted a statistical analysis. Standardization of methods across the literature would facilitate comparison, and experimental work may help disentangle cause and effect (Waldenström *et al.*, 2010; Taff & Townsend, 2017; Sánchez *et al.*, 2018). A recent meta-analysis by Sánchez *et al.* (2018) found that the method used to evaluate body condition was one of the strongest predictors of positive, negative, or null condition–infection relationships, suggesting the importance of choosing appropriate condition metrics. Sánchez *et al.* (2018) recommend utilizing multiple condition metrics appropriate for the host–parasite biology being studied and pairing these with experimental methods when possible. For example, it is likely commensal *Campylobacter* spp. and avian pathogenic serovars of *Salmonella* Typhimurium have vastly differing impacts on host condition, but more work is needed to demonstrate this.

Innate immunity can vary among individuals both within and among species. Differences in innate immunity leading to differences in bacteria-killing ability and bacterial colonization within a species have been observed in tree swallows (*Tachycineta bicolor*) (Mills *et al.*, 1999; Schmitt & Bélisle, 2017). A cross-fostering experiment of tree swallows conducted by Morrison, Ardia & Clotfelter (2009) suggested heritable immunity was more important in bacteria-killing ability against *E. coli* than body condition or brood size of the foster nest. Girard *et al.* (2011) found variation in bacteria-killing ability against *E. coli* between American robins, house sparrows, and gray catbirds (*Dumetella carolinensis*), suggesting that species could vary in susceptibility to infection due to innate differences in ability to defend against infection, leading to differences in risk of causing enteric pathogen outbreaks. Further, physiological differences among species such as full *versus* rudimentary caeca may affect susceptibility to pathogen colonization (Albuquerque *et al.*, 2013). The ecoimmunological 'pace of life' hypothesis predicts that bird species with early maturity, rapid breeding, short longevity, etc., face a trade-off between resources devoted to these life-history characteristics and those allotted to anti-pathogen defences

(Ostfeld *et al.*, 2014). Additionally, immunological trade-offs occur throughout the annual cycle during energetically costly events including breeding, migration, and moult periods (Martin, 2005; Martin *et al.*, 2008; Altizer *et al.*, 2011). Thus, reservoir competence is likely to vary both within and among species due to differences in innate immunity, season, life-history events, and anatomical factors.

Infectious dose (the minimum number of microorganisms sufficient to establish an infection) may also vary among individuals and species. However, few studies have experimentally infected birds to determine what constitutes an infectious dose (Table S8). Rock pigeons orally inoculated with 9.5×10^7 colony forming units/ml (CFU/ml) of *Salmonella* Enteritidis began to shed *Salmonella* spp. 3 days post inoculation, but lower doses were not tested, leaving uncertainty around the infectious dose. Rock pigeons shed between 1.5×10^4 and 2×10^9 CFU/ml up to 14 days post inoculation during a 35-day trial (Albuquerque *et al.*, 2013). In a 10-day trial, house sparrows orally inoculated with 10^2 CFU of a songbird outbreak strain of *Salmonella* Typhimurium shed on days 1 and 5, birds given 10^3 CFU shed on days 1–2 and 6–10, birds given 10^5 CFU shed most days (2/6 died on days 8 and 10), and birds given 10^8 CFU shed every day until their death (6/6 birds died on days 3–8) (Connolly *et al.*, 2006). In another study, herring gulls (*Larus argentatus*) already infected with *Salmonella* spp. were captured and maintained in captivity for 3 weeks and shed 170 most probable numbers/gram (MPN/g) for up to 4 days (Girdwood *et al.*, 1985). Infectious dose is important because bird species and individuals within species that are susceptible to lower infectious doses are more likely to become infected, but few data are available to characterize these differences or evaluate their impacts on disease risk in the field.

Further, many strains appear to be host adapted which can alter infectious dose and duration/intensity of shedding (Atterby *et al.*, 2018). Mallards inoculated with 5×10^4 CFU of *C. jejuni* of mallard, chicken, or song thrush (*Turdus philomelos*) origin per ml of water showed interesting and differing responses to the strains. Birds exposed to the mallard strain excreted around 10^4–10^6 CFU/ml throughout the 18-day experiment. Birds exposed to the chicken strain excreted an average peak level of 10^4 CFU/ml, and at the end of the experiment, only two of six birds continued to shed the bacteria. Mallards exposed to the song thrush strain shed 10^3–10^4 CFU/ml for the first few days after exposure then shedding declined rapidly (Atterby *et al.*, 2018). In another experiment, European robins (*Erithacus rubecula*) were exposed to either a song thrush *C. jejuni* strain or a *C. jejuni* strain of human origin and monitored for 25 days. Robins inoculated with the song thrush strain shed bacteria for 6.8 days on average, whereas those given the human isolate were not colonized, and bacteria were only detected in three of eight birds in the human isolate treatment group for up to 3 days post inoculation (Waldenström *et al.*, 2010). This suggests that songbirds may not be competent hosts of human-adapted strains. European starlings inoculated with doses ranging from $1 \times 10^{0.6}$ to 5×10^6 CFU of *E. coli* O157:H7 had an ID_{50} (number of microbes necessary to infect a host in 50% of the exposed population) of $\log_{10}$ 4.5 CFU for one strain and $\log_{10}$ 5.5 CFU for another strain. As dose increased, duration of shedding increased; shedding intensity ranged from around 10^1 to 10^6 CFU/g. High exposure levels resulted in shedding up to the final day of the 14-day trial (Kauffman & Lejeune, 2011). Altogether, this body of studies suggest infectious dose, shedding intensity, and shedding duration may vary by bacterial strain.

Few data exist on naturally occurring bacterial shedding intensity, and current data suggest wide variation in shedding intensity by bacterial species and avian taxa. For example, naturally occurring wild bird faeces have been found to have concentrations of *Campylobacter* spp. ranging from 340 cell equivalents per gram (CE/g) [California gull (*Larus californicus*)] to 1×10^8 CFU/g [ring-necked pheasants (*Phasianus colchicus*)], averaging between 4.8×10^3 CFU/g (Canada goose) to 6.7×10^6 CE/g (California gull; Table S8). Naturally occurring wild bird faeces have been found to have concentrations of generic *E. coli* ranging from 1.9×10^2 CFU/g to 2.5×10^9 CFU/g (herring gull), averaging between 2.8×10^4 MPN/g (sandhill crane) and 4.9×10^8 CFU/g (*Larus* spp.). Naturally occurring wild bird faeces have been found to have concentrations of *Salmonella* spp. averaging from 22 MPN/g to 2.4×10^9 CFU/g (herring gull). We found no reports of naturally occurring pathogenic *E. coli* concentrations. We only found reports of naturally occurring enteric bacteria concentrations for herring gull, California gull, sandhill crane, Canada goose, mallard, ring-necked pheasant, cattle egret (*Bubulcus ibis*), and unspecified *Larus* spp., with no reports for Passeriformes (songbirds, flycatchers). Colles *et al.* (2009) reported carriage among recaptured European starlings and found 18.2% were shedding *Campylobacter* spp. on each capture occasion (1 and 588 days apart), 40.1% were negative on each occasion (1 to 364 days apart), and 41.7% changed status (1–392 days). The majority (83.8%) of *C. jejuni* isolates from starlings shedding on more than one occasion were of a different genotype between surveys, suggesting rapid turnover and re-colonization.

The virulence of bacteria to bird species and individuals within a species is variable but can affect the duration and intensity of shedding and subsequent likelihood of infecting new hosts. For example, oral inoculations of *Salmonella pullorum* [a bacterium common in Galliformes (pheasants/quail)] at 1×10^4 CFU/ml was sufficient to cause mortality of northern bobwhite quail (*Colinus virginianus*) whereas mallards given up to 1×10^{10} CFU/ml showed no signs of disease, although bacteria were isolated from mallard tissues, indicating successful colonization (Buchholz & Fairbrother, 1992). Species that are asymptomatic carriers of bacteria may transmit bacteria for a longer duration over a wider area than species that are killed rapidly by the pathogen (Tizard, 2004). *Salmonella* Typhimurium tends to cause mass mortalities among finches, sparrows, and cowbirds (Faddoul, Fellows & Baird, 1966; Daoust *et al.*, 2000; Hall & Saito, 2008), whereas other bird species such as raptors and pigeons tend to survive infection, although mortality can occur (Tizard, 2004; Albuquerque

et al., 2013). Thus, species or individuals that survive infection may pose a greater risk than individuals killed rapidly. Wild birds are generally considered asymptomatic carriers of *Campylobacter* spp. [but see Waldenström *et al.*, 2010 and Taff & Townsend, 2017], and although avian pathogenic *E. coli* (APEC) can cause avian disease, it is generally associated with environmental and predisposing factors (Dho-Moulin & Fairbrother, 1999); thus, the severity of infection with *Campylobacter* spp. and *E. coli* is unlikely to compare to avian Salmonellosis. Wild birds may be more competent hosts of *Campylobacter* spp. if they remain largely unaffected, while species highly vulnerable to die-offs due to *Salmonella* Typhimurium may be poor hosts due to rapid death and less time for dissemination.

(3) Contact

For enteric pathogens to spill over from wild birds to humans, humans must either come into direct contact with the pathogens (i.e. direct hand to mouth contact) or consume food items or water contaminated with faeces. Direct contact with faeces may be the greatest source of enteric pathogen spillover from wild birds to humans (Strachan *et al.*, 2013; Cody *et al.*, 2015). Direct faecal contact often occurs when children in playgrounds, parks, or beaches touch bird faeces and then place their hands in their mouths (Strachan *et al.*, 2013; Abdollahpour *et al.*, 2015; Cody *et al.*, 2015). Handling and consuming undercooked game meat is another direct source of enteric pathogens (Navarro-Gonzalez *et al.*, 2016). Indirect sources include contaminated produce (Gardner *et al.*, 2011), infected livestock (Carlson *et al.*, 2011; Hald *et al.*, 2016), contaminated water (Lu *et al.*, 2013; Strawn *et al.*, 2013; Clark, 2014; Marine *et al.*, 2015), and domesticated household cats infected as a result of consuming birds (Fichtel, 1978; Tizard, 2004), among other sources (e.g. Penfold, Amery & Morley Peet, 1979; Neal & Slack, 1997; Ejidokun *et al.*, 2006).

Avian faecal contact with produce or other crops can occur in production fields, food wash and packing structures, storage facilities, or during preparation (Penfold *et al.*, 1979; Gardner *et al.*, 2011). Wild birds can directly contaminate produce by defecating while in fields (resting, foraging, etc.) or while flying over. Few data are available comparing faecal outputs per unit time during different activities for wild bird species, but activities that generate greater rates of faecal output could increase the risk of crop contamination (Feare *et al.*, 1999). Canada geese are known to have higher rates of faecal output while feeding compared to loafing (Feare *et al.*, 1999), suggesting that birds actively foraging in fields for insects or crops may have higher defecation rates that could increase contamination risk. Birds actively foraging in patches likely have higher faecal output rates than birds in flight (Guillemette, 1994), and more sustained contact times could lead to greater accumulation of faeces in production areas on or near crops (Feare *et al.*, 1999). Further, gut retention time can vary by diet items, which could increase or decrease faecal output rates for particular diet guilds (Levey & Karasov, 1994).

Intraspecific differences in habitat associations could alter bird contact with crops that differ in susceptibilities to facilitating food-borne illness (Brandl, 2006; Berger *et al.*, 2010). For example, arboreal bird species likely spend more time foraging in tree fruit than row crops, whereas ground- and understorey foraging species may be more likely to forage in crops such as lettuces, brassicas, and cucurbits. Understorey foragers that forage on leaves or stems may be more likely to deposit droppings on edible parts of plants whereas ground foragers may primarily contaminate soil. Grassland species may be more likely to inhabit livestock pastures, and species that nest on structures may have greater contact with livestock near livestock shelters, increasing likelihood of transmission to livestock. Species nesting within food wash/packing structures may also contaminate produce before it leaves the farm. Species with low pathogen prevalence but high contact rates with sensitive areas of food production may pose greater risk than species with high prevalence but low contact rates, and testing this idea would be an interesting avenue for future research.

In addition to direct faecal contact with crops and livestock, birds may cause indirect contamination. For example, European starlings have been shown to mechanically vector *Salmonella enterica* within feedlots when cattle excrement adheres to feathers and feet (Carlson *et al.*, 2015), although data are limited on the ability of other species to mechanically vector enteric pathogens. Flies could also mechanically vector pathogens from wild bird faeces to sensitive crop areas or to chickens that consume the contaminated arthropods (Skov *et al.*, 2008). Further, wild birds such as geese and ducks could contaminate produce by defecating in irrigation water (Lu *et al.*, 2013; Strawn *et al.*, 2013; Clark, 2014; Marine *et al.*, 2015).

The increase in enteric pathogen prevalence during migration (see Section III.1) is a concern because farmland habitat is recognized as important stopover habitat for a variety of birds, which may increase their direct and indirect contact rates with produce concurrent with a spike in prevalence (Hussong *et al.*, 1979; Dänhardt *et al.*, 2010; Lu *et al.*, 2013; Callaway *et al.*, 2014; Taff *et al.*, 2016). Indeed, the only food-borne illness outbreak directly linked to wild birds occurred during sandhill crane migration when cranes were using a produce farm as a stopover site (Gardner *et al.*, 2011). Although Marine *et al.* (2015) found autumn had the highest prevalence of *Salmonella* spp. on leafy greens, irrigation water, compost, field soil, and pond sediment samples, neither Strawn *et al.* (2013) nor Benjamin *et al.* (2013) found higher prevalence of *Salmonella* spp. or *E. coli* in on-farm water or soil samples in autumn. More research could help elucidate whether increased prevalence of pathogens in wild birds and increased crop contact rates during migration translate to higher on-farm pathogen contamination.

The seasonality of bird contact with people or food production *versus* the timing of pathogen acquisition by birds could mediate spillover probability if pathogen shedding is short in duration (Table S9). For example, finches, blackbirds, and sparrows are often infected with *Salmonella* spp. when they aggregate at feeders during cold weather (Fichtel, 1978;

Daoust *et al.*, 2000; Tizard, 2004). However, people are less likely to come into direct contact with bird faeces during winter (Strachan *et al.*, 2013; Cody *et al.*, 2015), and crop production is generally reduced or absent. Nevertheless, if birds infected during the winter can shed pathogens during warmer months when humans have greater direct contact with wild bird faeces and when birds forage in agricultural production areas, birds could remain a year-round risk. In addition to seasonal behavioural changes such as using bird feeders more often, many resident bird species alter habitat usage throughout the annual cycle, which may alter exposure rates (Zuckerberg *et al.*, 2016) and cause fluctuations in risk levels. For example, European starlings have the highest abundances in feedlots in winter (Fischl & Caccamise, 1985) where the birds can acquire enteric pathogens and spread bacteria between herds (Carlson *et al.*, 2011; Kauffman & Lejeune, 2011), but the likelihood of harbouring the pathogens over the winter and shedding pathogens on produce in the crop growing season is unknown. Glunder, Neumann & Braune (1992) observed *Campylobacter* spp. shedding in 27 herring gull chicks in captivity for 58 weeks and failed to detect *Campylobacter* spp. after 4 weeks. Albuquerque *et al.* (2013) inoculated rock pigeons with *Salmonella* Enteritidis and observed shedding up to day 14 of the 35 day trial, but we know of no other studies examining how long birds can shed enteric pathogens, leaving uncertainty if species infected during the winter are likely to shed pathogens during the growing season.

In our literature review, we found 58 estimates from 45 studies on seasonal variation in prevalence (Table S9). It is generally thought that outbreaks of Salmonellosis in wild birds occur in winter when birds aggregate at feeders during harsh weather (Daoust *et al.*, 2000; Hall & Saito, 2008). Eleven of twelve studies investigating mortality from suspected Salmonellosis outbreaks reported the highest incidence in the winter months. Twelve studies conducted statistical analyses testing for seasonal variation in *Salmonella* spp. prevalence in populations of live birds: seven found no difference, three found higher prevalence in summer, one found higher prevalence in autumn, and one found higher prevalence in winter. Seven studies conducted statistical analyses testing for seasonal variation in *Campylobacter* spp.: one found no difference, three found higher prevalence in summer, one found higher prevalence in winter, one found higher prevalence in winter and spring, and one found higher prevalence in spring. The seven studies testing for seasonal variation in pathogenic *E. coli* had the most consistent trends: five studies found the lowest prevalence in winter, three found the highest prevalence in summer, two found the highest prevalence in autumn, one reported the highest prevalence in a combined summer/autumn period, and one found no significant difference.

(4) Bacterial survival and transmission

In order for pathogens in bird droppings to spill over into humans, the pathogens must survive in faeces, water, or food until faecal–oral contact occurs. If contact occurs with food, the bacteria must successfully colonize livestock or survive in crop fields on produce, survive through washing, shipment, food processing at plants, preparation, and finally enter and establish within a human host. Data on pathogen survival in wild bird faeces are limited. The most information about pathogen survival in wild bird faeces has come from studies on Canada geese. A 28-day trial conducted in parks in London, England that inoculated Canada goose droppings with 10^4–10^5 CFU/g of *Salmonella* Newport found that the bacteria were able to survive through the full 28-day trial, despite heavy rain (Fontaine *et al.*, 1980). A 77-day trial in New Zealand that inoculated Canada goose faeces with 10^8 CFU/g *C. jejuni*, placed faeces in pasture, and measured survival in both summer and winter found *C. jejuni* fell below the limit of detection by day 2 in summer. In winter, *C. jejuni* were reduced to below 1% of the original concentration by day 4 and were last detected on day 9. Naturally occurring generic *E. coli* averaging around 3.5×10^6 CFU/g in summer and 4.9×10^4 CFU/g in winter were also monitored in the faeces. In summer, concentrations doubled on day 2 then steadily decreased until day 42 when concentrations were 1% of original levels and dropped to <0.005% on the final day (day 77) of the trial. In winter, *E. coli* concentrations declined 10-fold by day 2, fell below the detection limit on day 14, and remained below detection until days 56 and 63 when *E. coli* were again detected (Moriarty *et al.*, 2012). Bacterial survival in Canada goose faeces may be greater than survival in songbird faeces, which tend to be smaller with greater relative surface area, leaving faeces subject to greater desiccation, but data are lacking to support this assumption. *E. coli* O157:H7 have remained viable in laboratory-kept pooled and homogenized European starling faeces stored in the dark at 22°C for up to 76 days (Kauffman & Lejeune, 2011), but outdoor conditions could vastly alter survival. We know of no other published estimates on *Campylobacter* spp., *E. coli*, or *Salmonella* spp. in songbird faeces. Research examining bacterial survival in a diverse range of wild bird faeces is a high priority. Shorter *versus* longer survival times in faeces of various species of wild birds could cause vastly differing likelihoods of viable bacteria reaching a human consumer at an infectious dose.

Once deposited, bacteria could leave faeces and enter soil or water where survival may differ based on both biotic and abiotic factors. Soil or water with diverse microorganisms could suppress pathogens through predation or competition (Flint, 1987; Jiang, Morgan & Doyle, 2002; Liang *et al.*, 2011; Jacobsen & Bech, 2012). However, the relationship is not always straightforward: biofilms could also protect pathogens from ultraviolet exposure and increase bacterial survival (Brandl *et al.*, 2005). Salinity, pH, nutrient sources, soil type, temperature, and moisture can all also influence bacterial survival (Van Donsel, Geldreich & Clarke, 1967; Reddy, Khalell & Overcash, 1981; Ogden *et al.*, 2001; Natvig *et al.*, 2002; Ma *et al.*, 2011, 2012). Thus, the relationships between biotic and abiotic factors and bacterial survival are complex and still not well understood.

Salmonella spp. generally have the highest survival in soil, followed by *E. coli*, then *Campylobacter* spp. (Guan & Holley,

2003; Winfield & Groisman, 2003). *Salmonella* Typhimurium, the most commonly reported serovar in wild birds, can survive up to 231 days in naturally occurring soil (Islam *et al.*, 2004a), and *Salmonella* Newport can survive up to 405 days in manure-amended autoclaved soil if microbial competition has been eliminated (You *et al.*, 2006). *E. coli* can survive in soil for up to 217 days (Islam *et al.*, 2004c). Further, *E. coli* have been found to survive in sheep manure piles for up to 21 months (Kudva, Blanch & Hovde, 1998). *Campylobacter* spp. can survive in manure-amended soils for up to 120 days (Hutchison *et al.*, 2004). Thus, all three bacteria can have long survival times in soils where they could remain viable until encountered.

Most studies examining bacterial survival in water are limited by short experimental durations (<2 weeks), but some papers have examined survival over periods of several months. *E. coli* can survive up to 260 days in autoclaved and filtered water (Flint, 1987). *Campylobacter* spp. can survive up to 4 months (~122 days) in stream water (Rollins & Colwell, 1986). *Salmonella* Typhimurium can survive up to 14 weeks (~98 days) in water heavily contaminated with sheep faeces (Tannock & Smith, 1972). Survival of all bacteria has been found to be highest at the lowest temperatures, with decreasing survival as temperatures increase (Rollins & Colwell, 1986; Flint, 1987; Mezrioui, Baleux & Troussellier, 1995). Studies of longer durations examining both biotic and abiotic mediators would help improve our understanding of the long-term viability of enteric pathogens in the environment prior to encounter by animal hosts.

Many factors can influence pathogen survival on crop surfaces including the physicochemical nature of plant surfaces, biofilm formation, microbe–microbe interactions, and plant–microbe interactions (Brandl, 2006; Berger *et al.*, 2010). *Salmonella* Typhimurium can survive on lettuce for up to 63 days, on parsley for up to 231 days, on radishes for up to 84 days, and on carrots for up to 203 days (Islam *et al.*, 2004a,*b*). *E. coli* can survive on lettuce for up to 77 days, on parsley for up to 177 days, on onions for up to 74 days, and on carrots for up to 168 days (Islam *et al.*, 2004c, 2005). *Campylobacter* spp. can survive on spinach up to 9 days, on lettuce for up to 1 day, and in radish roots up to 23 days (Brandl *et al.*, 2004). Crop–bacteria combinations that have greater survival durations are more likely to reach human consumers, although data are still limited to a few crop–bacteria examples. Further, if the outer skin or shells are removed prior to consumption, pathogens may be less likely to infect a human consumer.

Once contaminated produce or livestock leaves the farm environment, the risk of causing foodborne illness could differ depending on transport, slaughter practices, food preparation, and proper cooking (Cesare *et al.*, 2003; Wassenaar, 2011; Batz *et al.*, 2012). Batz *et al.* (2012) found *Campylobacter*–poultry had the highest annual disease burden of pathogen–food combinations examined, with *S. enterica*–poultry in fourth place, *S. enterica*–complex foods in seventh, *S. enterica*–produce in eighth, and *S. enterica*–eggs in tenth. *E. coli* O157:H7–beef was the highest *E. coli*–food combination in 21st place. *Salmonella* spp. can have an infectious dose as low as 1.7×10^1 CFU in humans (Blaser & Newman, 1982). *Campylobacter* spp. have an infectious dose as low as 8×10^2 CFU in humans (Black *et al.*, 1988). *E. coli* also have an extremely low infectious dose with 4–45 bacteria being sufficient to cause enteric illness in humans (Tilden *et al.*, 1996). As summarized above for birds, the infectious dose for humans could vary among strains and individual people due to variation in genetic factors, age, immunological status, physiological state, and health (Blaser & Newman, 1982; Feare *et al.*, 1999; Plowright *et al.*, 2017). Further, different types of foods can differ in risk not only due to differences in hospitableness to bacteria and preparation but also due to differences in fat and protein content, which can affect bacterial survival inside a human stomach (Fontaine *et al.*, 1980). While there may be wide variation in the likelihoods of food–pathogen combinations resulting in illness (Fontaine *et al.*, 1980; Batz *et al.*, 2012), the infectious doses could be extremely low for some individuals (Blaser & Newman, 1982; Black *et al.*, 1988; Tilden *et al.*, 1996).

A growing body of research examining genetic relatedness between bacteria of wild birds, livestock, and human origin suggests that crossover is rare and most strains are host adapted (Table S10) (Waldenström *et al.*, 2002; Colles *et al.*, 2009; Kauffman & Lejeune, 2011; Weis *et al.*, 2016; Atterby *et al.*, 2018). Current literature suggests isolates from wild birds often exhibit sub-types with higher levels of similarity to isolates from birds of the same species or guild than to isolates from other groups of wild birds, livestock, or humans (Broman *et al.*, 2004; Griekspoor *et al.*, 2013). Both laboratory and field studies have demonstrated that wild birds can harbour strains that infect livestock and humans (Skov *et al.*, 2008; Kauffman & Lejeune, 2011). However, host adaptation of bacterial strains may cause wild birds to have low reservoir competence for isolates from livestock and humans, and *vice versa* (Waldenström *et al.*, 2010; Atterby *et al.*, 2018). Thus, spillover of enteric pathogens from birds may be less likely than suggested by high prevalence values for a given bacterial genus or species (Fig. 2). There is generally a low prevalence of enteric bacteria in livestock or humans that have genetic similarity to bird isolates, suggesting that spillovers are rare [Table S10; e.g. no similar strains in starling isolates in Colles *et al.*, 2009, 0.9% of mallard isolates in Colles *et al.*, 2011, and 2.2% of migrating bird isolates in Broman *et al.*, 2004]. For example, although mallards have a high prevalence of *Campylobacter* spp. (~26%; see Section IV.1*a*), if only 0.9% isolates are known to cause human disease (Colles *et al.*, 2011), then only about 2.3 in 1000 mallards would carry a human disease isolate, much lower than the genus-level estimate suggests (Fig. 2A). Moreover, the rare mallards carrying isolates that can cause human disease must also contact humans or food for spillover to occur. Using the Smith *et al.* (2019) farm bird database, we estimated the number of farm survey points (100 m radius point count survey) with one or more mallards observed over the two-year study. Mallards were only observed in or flying over 18/217 (8.3%) survey points during one or more survey occasion. Thus, only

about 2 in 10000 survey points were likely to harbour a mallard with a disease isolate (Fig. 2B). Further, extrapolating *Campylobacter* spp. survival in Canada goose faeces to mallards from Moriarty *et al.* (2012), bacteria are unlikely to survive for more than 2 days in summer (Fig. 2C). Thus, what appears to be a high likelihood of spillover based on prevalence data alone decreases when multiple transmission parameters are considered together (Figs 1 and 2D).

Another approach that has been used to estimate spillover is to analyse the number of livestock isolates or human disease isolates of wild bird origin. Pennycott, Park & Mather (2006) found *Salmonella* spp. isolates from livestock of wild duck/goose origin accounted for 3% of isolates, while all other wild bird isolates accounted for <1%. Similarly, strains isolated from human disease cases tend to show high similarity to livestock strains not of wild bird origin and low similarity to wild bird isolates (Griekspoor *et al.*, 2013). Seguino *et al.* (2018) found that wild bird isolates accounted for 0.23% of human *C. jejuni* and *C. coli* infections while livestock sources accounted for 16%. Cody *et al.* (2015) found that wild bird

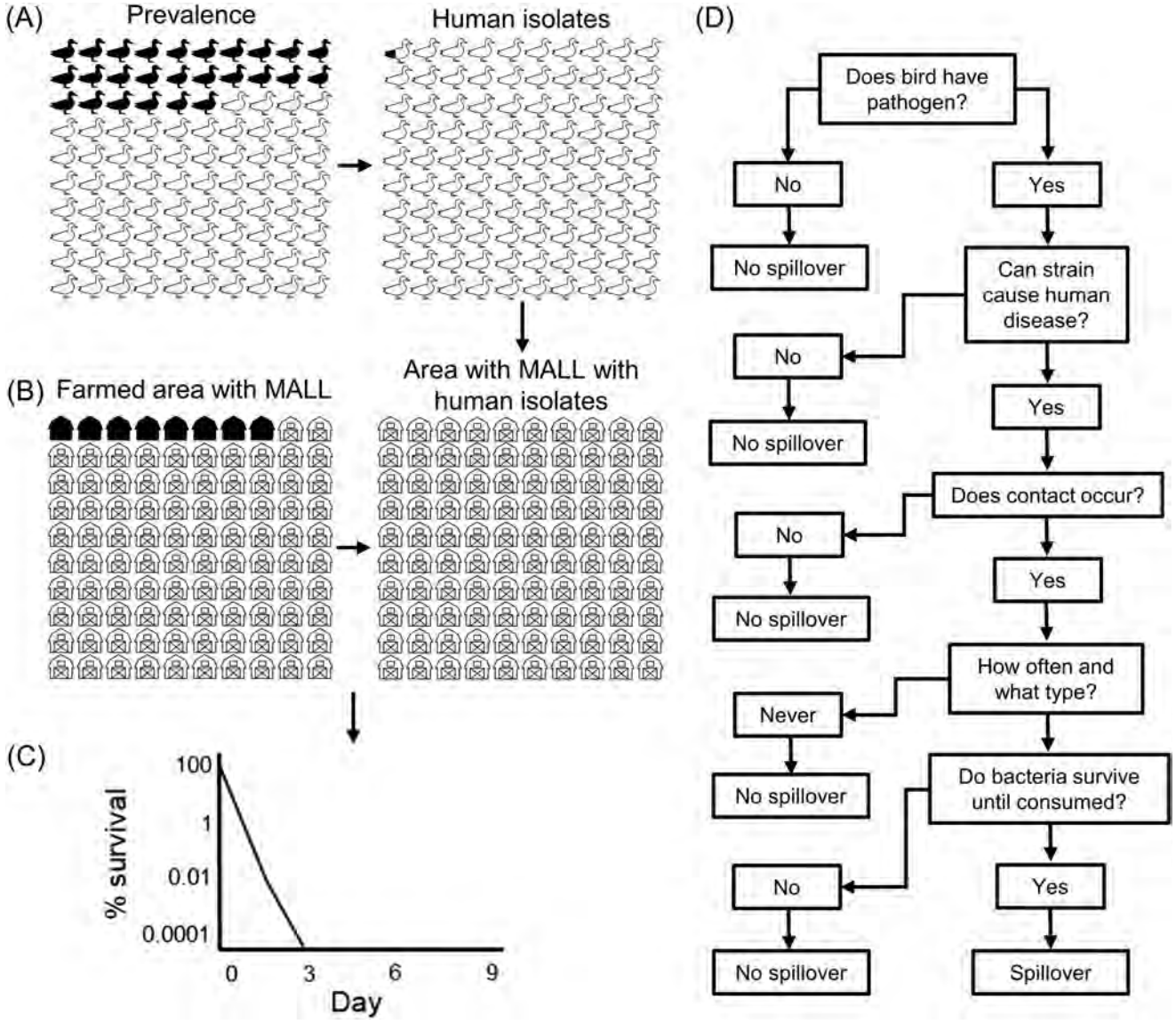

Fig. 2 Conceptual diagram of likelihood of *Campylobacter* spp. spillover from mallards (MALL) to humans. (A) *Campylobacter* spp. prevalence in mallards estimated from meta-analysis (26%) on left and prevalence of *Campylobacter* isolates matched with human disease cases estimated from Colles *et al.* (2011) (26% prevalence of which 0.9% of isolates are known to cause human disease) on right. (B) Estimated prevalence of mallards in farmland from Smith *et al.* (2019) farm bird database (8.3% of points) on left and area likely to have mallards with human isolates on left (about 2 in 10000). (C) Estimated survival time of *Campylobacter* spp. in mallard faeces modified from Canada goose faeces study in Moriarty *et al.* (2012). (D) Flow chart to determine whether spillover will occur.

C. jejuni isolates accounted for between 2.1 and 3.5% of cases in the UK annually, with the largest proportion occurring in the summer months when humans frequent parks and beaches. Similarly, Strachan *et al.* (2013) found the highest proportion of *Campylobacter* spp. human clinical isolates of wild bird origin in the summer from young children (9% of cases were wild bird isolates). However, studies that have quantified relatedness using more than one technique often find contradictory results, leading to uncertainty in robustness of conclusions. For example, Sanad *et al.* (2013) used both pulse-field gel electrophoresis and multi-locus sequence typing to compare European starling and cattle isolates and obtained highly variable phylogenies using the two techniques. This points to the need to determine which methods yield the most robust results for future comparisons. Research quantifying the risk of wild birds transmitting enteric pathogens should compare wild bird isolates to human or livestock disease isolates robustly, given that shared strains are likely a minority of cases (Broman *et al.*, 2004; Colles *et al.*, 2011; Seguino *et al.*, 2018).

(5) Integrating from exposure to transmission

We collected data on 30 binary variables from each study gathered through our literature review that met inclusion criteria 1–6, which we classified as related to exposure ($N = 6$ variables), reservoir competence ($N = 14$ variables), contact ($N = 4$ variables), or bacterial survival and transmission ($N = 6$ variables) (Data S1 and S2; Tables S1 and S2). We then quantified the number of studies that presented data on one or more aspects within each category and the number of categories each study covered. Thirty-eight (18.0%) studies reported data on one or more aspects of exposure, 189 (89.6%) studies reported data on one or more aspects of reservoir competence, 54 (25.6%) studies reported data on one or more aspects of contact, and 94 (44.5%) studies reported data on one or more aspects of bacterial survival and transmission (Fig. 1B). Seven studies (3.3%) did not report data beyond simple prevalence estimates, 78 (37.0%) reported data on one of the four categories, 88 (41.7%) reported data on two of the four categories, 31 (14.7%) reported data on three of the four categories, and 7 (3.3%) reported data on all four categories (Fig. 1C). Studies which integrate all stages of our framework described above will be most effective at generating useful data to develop risk models and to develop policy to reduce pathogen spillover from wild birds into humans. Indeed, this appears to be a problem across the disease literature: a systematic review of 442 modelling studies covering 85 zoonotic pathogens conducted by Lloyd-Smith *et al.* (2009) found the disease ecology literature often fails to account for the full ecology of pathogens, with only six of the 442 studies examined including a mechanistic model of zoonotic spillover.

Models integrating data across the entire process are no trivial undertaking (Plowright *et al.*, 2017; Childs *et al.*, 2019; Cross *et al.*, 2019; Washburne *et al.*, 2019). Cross *et al.* (2019) present case studies to highlight challenges and potential solutions to estimating spatiotemporal variation in spillover risk. For example, data sets on multiple host species collected in similar locations, seasons, and at similar resolutions along with data sets collected at all levels of the spillover process are rare. Cross *et al.* (2019) describe mechanistic approaches where researchers use data on host density, pathogen prevalence, transmission, and shedding to make predictions about spillover events. Conversely, phenomenological models use spillover events to estimate risk covariates that are correlated with host and pathogen distributions which are useful when data on host density and pathogen shedding are lacking. Washburne *et al.* (2019) present percolation models as a tool to model spillover whereby pathways to spillover are represented as directed graphs as pathogens move from reservoirs to people. Pathogens shed by reservoirs progress through the stages of transmission where pathogens diminish at each stage along the pathway due to failure to persist in the environment, failure to contact humans, failure to infect humans given contact, and failure to be detected by researchers. Each stage is represented as a series of probabilistic models. However, these models may fail if there are environmental feedbacks between environmental factors and wildlife reservoirs, which may be the case in enteric pathogen systems. Finally, Childs *et al.* (2019) present an environmental risk model to examine spillover probability of yellow fever virus from non-human primates to humans that may be successfully modified to model enteric pathogen spillover. However, the models presented by Childs *et al.* (2019) are fairly data intensive.

IV. META-ANALYSIS OF ENTERIC PATHOGEN PREVALENCE IN WILD BIRDS

Next, we conducted a meta-analysis on enteric pathogen prevalence in North American breeding bird species with several primary objectives. We sought to (*i*) generate more robust prevalence estimates than available in individual studies and compile estimates into an easily accessible database for wild bird community enteric pathogen transmission models (Data S2), (*ii*) identify which species and guilds should be the focus of future research, while pointing to understudied groups, and (*iii*) use our meta-data to test for differences in enteric pathogen prevalence by taxa and foraging guilds. We use prevalence as a proxy for transmission due to the much greater availability of prevalence data than other types of data, but we caution the reader in extrapolations from prevalence to risk of spillover (Fig. 2).

(1) Prevalence estimates

(*a*) *Campylobacter* spp.

14.8% (64/431) of North American breeding birds had *Campylobacter* spp. prevalence data (1+ observations) meeting our inclusion criteria 1–9 (Data S2). The species with the most observations meeting our inclusion criteria 1–9 were rock

pigeon [N = 3659 from 15 studies, range 6–1800 individuals tested, 0.1–70% reported prevalence, estimated prevalence 16 ± 5.3% (SE)], European starling [N = 2094 from 12 studies, range 1–957 individuals tested, 0–75% reported prevalence, estimated prevalence 28 ± 6.0% (SE)], mallard [N = 1941 from 11 studies, range 5–716 individuals tested, 0–79% reported prevalence, estimated prevalence 26 ± 7.0% (SE)], Canada goose [N = 1322 from 8 studies, range 44–357 individuals tested, 0–52% reported prevalence, estimated prevalence 16 ± 6.7% (SE)], and ring-necked pheasant [N = 932 from 8 studies, range 1–287 individuals tested, 0–37% reported prevalence, estimated prevalence 18 ± 5.6% (SE)]. Estimated *Campylobacter* spp. prevalence across all birds (N = 13606 individuals of 64 species) and studies (N = 56) was 27 ± 3.5% (SE). To determine prevalence with 5% precision with 27% average prevalence, we estimate that a study would have to test at least 303 individuals, but only 1.6% (7/431) of species examined met this threshold. The estimated prevalence for these seven species was 24 ± 4.4% (SE), with American crow, European starling, and mallard having the highest prevalence [52 ± 12% (SE), 28 ± 6.0% (SE), 26 ± 7.0% (SE), respectively; Fig. S7]. Antibiotic-resistant *Campylobacter* spp. were reported from five species (European starling, mallard, Canada goose, ring-necked pheasant, and house sparrow), all of which had >600 observations. 76% of reported campylobacters were identified as *C. jejuni*, 7.1% were identified as *C. coli*, 1.9% were identified as *C. lari*, and 0.08% were identified as *C. canadensis*. *C. peloridis* was reported once in herring gulls (*Larus argentatus*).

(*b*) *E. coli*

5.3% (23/431) of North American breeding birds had pathogenic *E. coli* prevalence data (1+ observations) meeting our inclusion criteria 1–9. The species with the most observations of data meeting inclusion criteria 1–9 on pathogenic *E. coli* were rock pigeon [N = 4954 from 17 studies, range 14–1800 individuals tested, 0–71% reported prevalence, estimated prevalence 8.5 ± 1.5% (SE)], European starling [N = 1081 from 5 studies, range 7–434 individuals tested, 0–14% prevalence, estimated prevalence 2.1 ± 0.6% (SE)], Canada goose [N = 1076 from 8 studies, range 1–600 individuals tested, 0–93% prevalence, estimated prevalence 33 ± 11% (SE)], house sparrow [N = 556 from 4 studies, range 40–237 individuals tested, 0–27% prevalence, estimated prevalence 8.4 ± 5.9% (SE)], and brown-headed cowbird (*Molothrus ater*, N = 309 from 1 study, 3.6% prevalence across the 309 individuals tested). Estimated pathogenic *E. coli* prevalence across birds (N = 9185) and studies (N = 36) was 20 ± 6.3% (SE). To determine prevalence of pathogenic *E. coli* with 5% precision and 20% average prevalence for a given species or community, we estimate that a study would have to test at least 246 individuals; yet, only 1.6% (7/431) of species examined met this threshold. We estimated the prevalence for these seven was 14 ± 6.1% (SE). Of those species, mallard, Canada goose, and Eurasian tree sparrow (*Passer montanus*) had the highest prevalence [41 ± 18% (SE), 33 ± 11% (SE), 12.9 ± 5.6% (SE), respectively; Fig. S7]. Including other studies that did not report data from which prevalence could be estimated, including necropsy studies, 7.4% (32/431) of species were tested for pathogenic *E. coli*. 8.1% (35/431) of North American breeding birds had any data allowing calculation of prevalence of generic *E. coli*, which we estimated to have an overall prevalence of 54 ± 5.9% (SE). Antibiotic resistance of any *E. coli* was reported in 20/431 (4.6%) species.

(*c*) *Salmonella* spp.

Salmonella spp. were the most studied bacteria with 33% (141/431) of North American breeding birds having prevalence data (1+ observations) meeting our inclusion criteria 1–9. The species with the most observations of data meeting inclusion criteria 1–9 were herring gull [N = 12470 from 10 studies, range 1–5324 individuals tested, 0–22% prevalence, estimated prevalence 8.2 ± 2.2% (SE)], house sparrow [N = 5581 from 19 studies, range 2–1124 individuals tested, 0–21% prevalence, estimated prevalence 2.5 ± 0.7% (SE)], rock pigeon [N = 5458 from 30 studies, range 4–1800 individuals tested, 0–100% prevalence, estimated prevalence 4.0 ± 0.9% (SE)], wild turkey [*Meleagris gallopavo*, N = 2401 from 4 studies, range 70–1164 individuals, 0–22.5% prevalence, estimated prevalence 5.5 ± 4.7% (SE)], and European starling [N = 2288 from 20 studies, range 4–1800 individuals tested, 0–100% prevalence, estimated prevalence 2.7 ± 1.0% (SE)]. Estimated *Salmonella* spp. prevalence across all birds (N = 40295) and studies (N = 102) was 6.4 ± 0.9% (SE). To determine prevalence with 5% precision with 6.4% average prevalence, a study would have to test at least 93 individuals, but only 7.4% (32/431) of species examined met this minimum threshold. Of these 32 species, we estimated *Salmonella* spp. prevalence was 6.3 ± 1.0% (SE). Of these species, Franklin's gull (*Leucophaeus pipixcan*), white-winged dove (*Zenaida asiatica*), and cattle egret had the highest prevalence [41 ± 34% (SE), 26 ± 3.3% (SE), 22 ± 15% (SE), respectively; Fig. S7]. Including necropsy studies or other studies that did not report data from which prevalence could be estimated, 36% (157/431) were tested for *Salmonella* spp. Antibiotic-resistant bacteria were reported from 17 bird species. Studies reported 105 serovars, the most common of which was Typhimurium (738/2298 individual birds from studies that reported serovar), followed by Virchow (410/2298), and Bredeney (87/2298).

(*d*) *Prevalence by taxonomic order*

We summarized pathogen prevalence by taxonomic order for the three pathogens (Fig. S8; Tables S11–S16), primarily to provide a basis for comparison of bird groups with high prevalence and to point to under-studied avenues for future research. The Gruiformes (cranes, rails) had the highest *Campylobacter* spp. prevalence [N = 217; estimated prevalence = 76.6 ± 10.6% (SE); Tables S11 and S12]. Other

orders did not differ significantly in *Campylobacter* spp. prevalence (Tukey HSD, $P > 0.05$; Table S12). There were few individuals tested for pathogenic *E. coli*, limiting comparisons (Tables S13 and S14). Galliformes had the highest prevalence of pathogenic *E. coli* [$N = 70$; estimated prevalence = 55.2 ± 16.6% (SE); Tables S13 and S14], making this group a high priority for continued research. The prevalence of pathogenic *E. coli* was significantly higher (Tukey HSD, $P < 0.05$; Table S14) in the Galliformes compared to the Charadriiformes [gulls; $N = 93$; estimated prevalence = 0 ± 20.9% (SE)], Pelecaniformes [$N = 66$; estimated prevalence = 10.3 ± 13.4% (SE)], and Anseriformes [ducks, geese; $N = 1563$; estimated prevalence = 22.1 ± 10.3% (SE)], but other pairwise comparisons were not significant. The Pelecaniformes had the highest estimated *Salmonella* spp. prevalence [Tables S15 and S16; $N = 566$, 16.8 ± 3.7% (SE)], which was significantly higher (Tukey HSD, $P < 0.05$; Table S16) than *Salmonella* spp. prevalence in the Passeriformes [$N = 13997$; estimated prevalence = 4.8 ± 1.0% (SE)], Anseriformes [$N = 3136$; estimated prevalence = 4.7 ± 1.2% (SE)], Columbiformes [doves, pigeons; $N = 5724$; estimated prevalence = 5.2 ± 1.3% (SE)], Charadriiformes [$N = 13395$; estimated prevalence = 8.4 ± 1.5% (SE)], Strigiformes [owls; $N = 151$; estimated prevalence = 7.4 ± 3.1% (SE)], and Galliformes [$N = 2688$; estimated prevalence = 8.0 ± 2.6% (SE)]. *Salmonella* spp. prevalence was higher in the Charadriiformes than the Columbiformes, Passeriformes, and Anseriformes.

(2) Taxonomic bias in research

To assess taxonomic bias in study species selection and representativeness of the current literature of birds that contact humans and food production, we used a two-part comparison. First, we compared observations by taxon to both the percentage of North American breeding birds each order comprises (species richness comparison) and the percentage of eBird observations by order (abundance comparison). Second, we compared observations by taxon to a database on farm bird abundances from Smith *et al.* (2019) to assess representativeness of currently available literature with respect to wild birds present within a vulnerable farming system as a case study of one of several important transmission points of enteric pathogens. The analyses revealed similar trends, so we only present results from the first comparison below and refer the reader to Tables S17–S19 and Figs S9 and S10 for farm bird summaries.

Compared to the percentage of species of North American breeding birds each taxon represents, there was significant bias in the number of individuals tested by taxonomic order for *Campylobacter* spp. ($\chi^2_{17} = 70004$, $P < 0.0001$), pathogenic *E. coli* ($\chi^2_{17} = 154{,}655$, $P < 0.0001$), generic *E. coli* ($\chi^2_{17} = 39333$, $P < 0.0001$), and *Salmonella* spp. ($\chi^2_{17} = 90432$, $P < 0.0001$) (Fig. S11; Table S20). Compared to the percentage of eBird sightings reported for North American breeding birds within each taxon, there was significant bias in the number of individuals tested by taxonomic order for *Campylobacter* spp. ($\chi^2_{17} = 36553$, $P < 0.0001$), pathogenic *E. coli* ($\chi^2_{17} = 57753$, $P < 0.0001$), generic *E. coli* ($\chi^2_{17} = 21314$, $P < 0.0001$), and *Salmonella* spp. ($\chi^2_{17} = 98876$, $P < 0.0001$) (Fig. 3; Table S21). Anseriformes, Charadriiformes, and Columbiformes tended to be overrepresented, while Passeriformes were under-represented in all categories. No data meeting inclusion criteria 1–6 or 8–9 existed for one or more of the bacteria for Accipitriformes (hawks, eagles, new world vultures), Caprimulgiformes (nighthawks, swifts, hummingbirds), Falconiformes (falcons), Gruiformes, Pelecaniformes, Piciformes (woodpeckers), Strigiformes, or Suliformes (cormorants). No data meeting inclusion criteria 1–6 or 8–9 existed for any bacteria for Ciconiiformes (storks), Coraciiformes (kingfishers), Cuculiformes (cuckoos), Gaviiformes (loons), or Podicipediformes (grebes). Passeriformes were the most diverse and abundant taxon within all data sets examined, suggesting that further research on this group should be a high priority. Accipitriformes and Piciformes were also common species in both data sets, but few studies have tested them for pathogens, again suggesting high priority for testing these taxa.

Next, we examined what percentage of species and what proportion of sightings represented individuals with enough data to estimate prevalence with 5% precision for no, one, two, or three pathogens (Fig. 4; Tables S19 and S22). Of all North American breeding bird species, 5/431 (1.2%) had sufficient data to calculate prevalence for three pathogens, 4/431 (0.9%) had sufficient data to calculate prevalence for two pathogens, 23/431 (5.3%) had sufficient data to calculate prevalence for one pathogen, 119/431 (28%) had some data but insufficient observations to determine prevalence, and 280/431 (65%) had no data (Fig. 4A). Our second analysis considered the percentage of birds in North America that had enough data to estimate prevalence with 5% precision for no, one, two, or three pathogens. Thus, we weighted each species by the number of observations in eBird and divided each by the total number of individuals reported across species in eBird. 7.6% of all sightings were comprised of species that had sufficient data to calculate prevalence for three pathogens, 3.1% of sightings were comprised of species with enough data to calculate prevalence for two pathogens, 12% of sightings were comprised of species with enough data to calculate prevalence for one pathogen, 51% of sightings were comprised of species that had some data but insufficient observations to determine prevalence, and 26% of sightings were comprised of species that had no data (Fig. 4B). These trends point to large inadequacies of the current literature for the majority of species and individuals that may contact humans. Future work should focus on species that are currently under-studied to understand whole-community risk better (Fig. 5). Although prior work has focused on key globally abundant species, large portions of highly abundant species have few to no data (e.g. American robin), which hinders the ability of robust risk modelling efforts to control enteric pathogen transmission to humans.

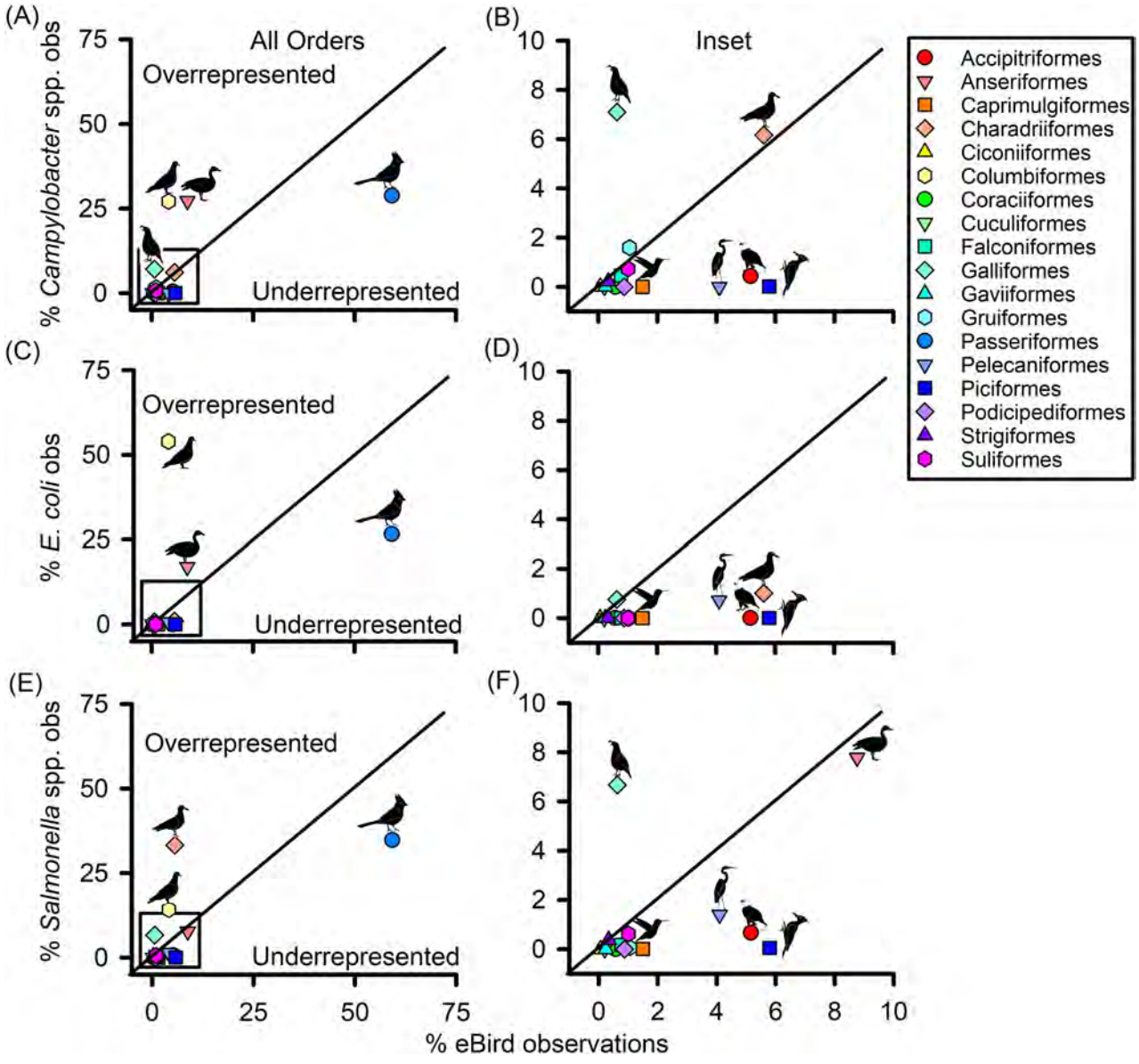

Fig. 3 Scatterplot showing the percentage of pathogen observations (obs) belonging to each taxonomic order *versus* the % of eBird observations (eBird.org) each taxon comprises (Sullivan *et al.*, 2009). (A), (C) and (E) show all orders. (B), (D) and (F) show orders that comprise less than 10% of pathogen observations and less than 10% of eBird observations (boxed regions in A, C and E, respectively).

(3) Prevalence by testing method

Next, we compared the prevalence of *Salmonella* spp. for three of the most tested species (European starling, house sparrow, and rock pigeon) by substance tested for bacteria [cloacal swabs, faeces, blood, and dissected internal organs ('necropsy')]. We hypothesized that studies that tested internal organs would find higher prevalence of pathogens because a bird would not have to be shedding bacteria in order to obtain a positive result (Girdwood *et al.*, 1985). If this were the case, species that are commonly tested by internal organ cultures after culling (invasive birds, hunted waterfowl, gulls, hunted upland game birds such as pheasants) may appear to have higher prevalence than species that require special permits to kill (native songbirds and other protected migratory birds that are not hunted for recreation). We did not make comparisons by substance tested for gamebirds or native species due to limited data.

Studies that tested house sparrow internal organs (model estimate 4.0 ± 0.6%) had higher prevalence than those that tested cloacal swabs (model estimate 0.7 ± 0.2%) or faeces (0.2 ± 0.2%) ($\chi = 5.33$, $P < 0.0001$ and $\chi = 6.06$, $P < 0.0001$, respectively) (Fig. 6; Table S23). Cloacal swabs

Fig. 4 Pie charts showing (A, C) the proportion of species or (B, D) relative abundances for which enough observations exist to estimate pathogen prevalence for *Campylobacter* spp., pathogenic *E. coli* and *Salmonella* spp. (purple), two of the three pathogens (dark blue), one of these three pathogens (blue), species with some data but insufficient numbers to determine prevalence (green), and no observations (yellow). (A) North American (NA) breeding bird species found in the North American Breeding Bird Survey (Sauer *et al.*, 2017), (B) eBird (ebird.org) relative abundances (Sullivan *et al.*, 2009), (C) West Coast farm bird species observed by Smith *et al.* (2019), and (D) farm bird species relative abundances from the database in Smith *et al.* (2019). Path = pathogens.

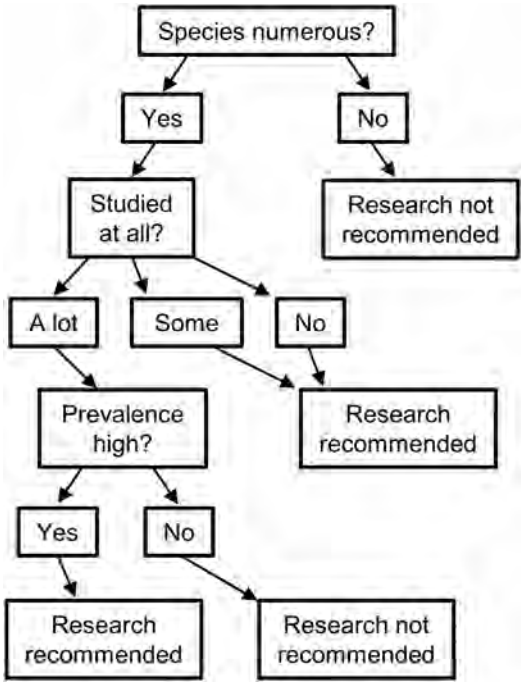

Fig. 5 Flow chart suggesting directions for future research.

had higher prevalence than faeces ($Z = -2.1$, $P = 0.035$). However, prevalence did not differ by substance tested for European starlings or rock pigeons ($P > 0.05$; Fig. 6; Tables S24 and S25). In line with our findings for house sparrows, Girdwood *et al.* (1985) found that 15.6% ($N = 746$) of gulls tested were positive for *Salmonella* spp. when the entire gut was tested whereas only 9.6% of the same gulls were positive when cloacal swabs were tested. Therefore, we suggest some caution when comparing prevalence between species that are often killed and dissected (geese, invasive species, gulls) compared to protected native species (finches, thrushes, etc.), but it does not appear that necropsies always yield higher prevalence estimates. We suggest that future work uses consistent methodology to facilitate comparisons among species.

(4) Prevalence by ecological guild

Next, we used our meta-data to assess the idea that foraging traits may alter enteric pathogen prevalence in wild birds by altering exposure rates to faecal contamination (Waldenström *et al.*, 2002; Skov *et al.*, 2008; Hald *et al.*, 2016). Across 85 Tukey HSD pairwise comparisons of diet guild and foraging strata for the three enteric pathogens (Fig. 7; Fig. S12; Tables S26–S37), only two were significant:

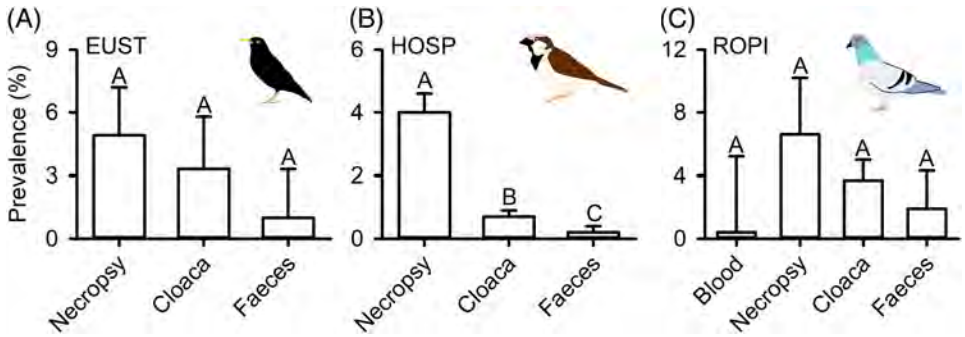

Fig. 6 Comparison of estimated *Salmonella* spp. prevalence (+SE) by substance tested for the three species with data from the most individual studies. (A) European starling, (B) house sparrow, and (C) rock pigeon. Different letters indicate significant differences using pairwise Tukey HSD tests.

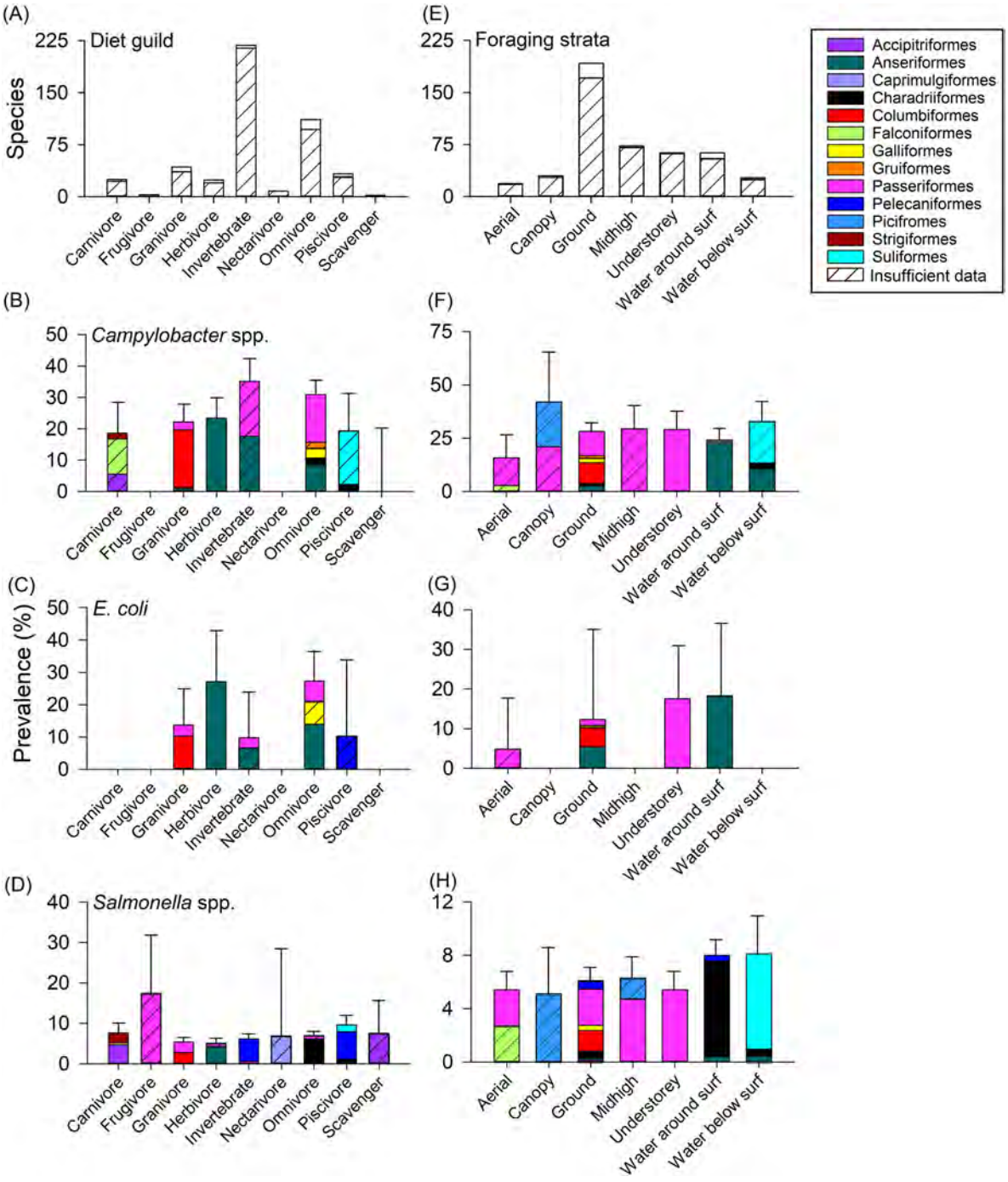

Fig. 7 Estimated prevalence (+SE) of enteric pathogens by (B–D) diet guild and (F–H) foraging strata, and number of species within each diet guild (A) and foraging strata (E). In A and E, pattern indicates insufficient observations to determine prevalence for any of the three pathogens while white indicates species for which enough observations were available to determine prevalence for one or more pathogens. (B, F) *Campylobacter* spp., (C, G) pathogenic *E. coli*, and (D, H) *Salmonella* spp. for each diet guild or each foraging strata. Colour indicates proportion of positive individuals for each estimate from each taxonomic order. Pattern indicates insufficient observations to determine prevalence within an order. Spaces with no error bars indicate no observations in the literature.

omnivores had higher *Salmonella* spp. prevalence [$N = 20577$; $6.9 \pm 1.0\%$ (SE)] than granivores [$N = 13252$; $5.4 \pm 1.0\%$ (SE); $Z = 2.33$; $P = 0.020$], and omnivores had higher *Salmonella* spp. prevalence than herbivores [$N = 2642$; $4.9 \pm 1.3\%$ (SE); $Z = 1.99$; $P = 0.047$]. We note the limitations in our data due to heavy representation of a few species that represent only a fraction of common urban and farm inhabitants and note the low number of observations for most guilds.

Thus, we suggest extreme caution in extrapolating any ecological trends from our analyses. Our results may also be biased due to confounding factors such as species traits (e.g. habitat association), sampling biases resulting from lack of data for most species, or both. For example, as summarized in our literature review above, primary studies have generally found lower enteric pathogen prevalence in granivorous species and higher enteric pathogen prevalence in ground-foraging species which may have lower and higher enteric pathogen exposure, respectively (Waldenström *et al.*, 2002; Sensale *et al.*, 2006; Skov *et al.*, 2008; Hald *et al.*, 2016). By contrast, we found only two differences across many comparisons. An alternative explanation is that few species forage exclusively within a single diet guild or strata and most exhibit some level of diet plasticity (Wilman *et al.*, 2014). Thus, grouping species into discrete classifications may be of little use in quantifying an association between diet or foraging strata and enteric pathogen prevalence.

V. FUTURE RESEARCH

In Section III, we developed a conceptual framework for developing risk models, expanding upon those proposed by Lloyd-Smith *et al.* (2009) and Plowright *et al.* (2017) (Fig. 1), and suggested avenues for future research. Based on a literature review, we identified critical gaps in knowledge throughout the pathogen life cycle and recommend that future studies use an approach that integrates across all aspects of pathogen transmission, from environmental exposure to successful colonization of human hosts (Fig. 1) (e.g. Hernandez *et al.*, 2016). In Section IV, we used a meta-analytic approach to summarize pathogen prevalence by species and orders, and we assessed what species, orders, and ecological guilds should be priorities for future research. Here, we synthesize what we see as the highest priority areas for future research.

We recommend that research efforts quantify prevalence in common but understudied species as a top priority (Fig. 5). We found few to no estimates of prevalence for Passeriformes, Accipitriformes, Caprimulgiformes, Falconiformes, Gruiformes, Piciformes, and Strigiformes, despite being common in agricultural and anthropogenic settings and representing 74% of all North American breeding birds. By contrast, we found disproportionately high amounts of data focused on Anseriformes, Charadriiformes, and Columbiformes (Fig. 3; Figs S9–S11; Tables S17, S18, S20 and S21). A large proportion of our meta-data included studies of a few highly abundant, globally distributed species, including rock pigeons, European starlings, Canada geese, mallards, herring gulls, and house sparrows (54.5% of studies were conducted outside of North America). We found few estimates on many common North American breeding birds (Fig. 4): 65% of North American breeding bird species had no pathogen data, and only 1.2% of species had enough data to determine prevalence for all three pathogens. Although data on a few key globally distributed species have the largest reach, some of the most abundant species in North America had few to no data, although necropsy studies indicate many of these species may be important reservoirs (e.g. American robin). This highlights a critical need for studies to focus on common, yet underrepresented species that could have disproportionately high influence on pathogen transmission within human-dominated landscapes (Fig. 5).

Although we cannot eliminate the possibility that our taxonomic and foraging guild analyses simply reflect a bias in the research, we suggest they may, to some degree, reflect unique pathogen–host–environment relationships that create some highly competent systems, driven by the unique lifestyle and survivability of each enteric pathogen (Jones *et al.*, 2013). *Campylobacter* spp. occurred at high prevalence (27%) throughout the groups tested compared to pathogenic *E. coli* (20%) and *Salmonella* spp. (6.4%) (Figs S7–S18; Tables S11, S13, S15). *Campylobacter* spp. are generally thought to be a natural commensal of wild birds with a more recent crossover into humans and livestock (Wassenaar, 2011; Griekspoor *et al.*, 2013). As a commensal, there would be little immune investment to kill the bacteria, allowing them to remain at high prevalence. This might suggest that prevalence is dictated more by survival in the non-host environment, which is thought to be low in comparison to *Salmonella* spp. and *E. coli* (see Section III.4). Because *Campylobacter* spp. generally have low survival on plant surfaces (Brandl *et al.*, 2004) but high prevalence in bird hosts, human disease from *Campylobacter* spp. may be more likely from consumption of poultry, game, or direct contact with faeces. Indeed, *Campylobacter* infection in humans most commonly arises from consumption of poultry meat rather than from produce, although exceptions do occur (Batz *et al.*, 2012). However, most cases of human Campylobacteriosis of avian origin are thought to occur from direct faecal contact (Strachan *et al.*, 2013; Cody *et al.*, 2015).

In contrast to *Campylobacter* spp., *Salmonella* spp. can cause mass mortalities in wild birds, with the highest documented mortalities in songbirds caused by *Salmonella* Typhimurium (Tizard, 2004; Connolly *et al.*, 2006; Hall & Saito, 2008). It is, therefore, hypothesized that *Salmonella* spp. should exist at low prevalence in wild birds (Brittingham *et al.*, 1988; Tizard, 2004; Hall & Saito, 2008), which our data supported (6.4% prevalence). *Salmonella* spp. are also thought to have high survival in the environment compared to the other pathogens (Winfield & Groisman, 2003). *Salmonella* spp. also have a large host range, including mammals, reptiles, birds, and amphibians (Navarro-Gonzalez *et al.*, 2016). It has been suggested that raptors may have high exposure to *Salmonella* spp. through consumption of contaminated rodents or infected birds (Tizard, 2004), which our data suggest may be the case (7.5% in Accipitriformes, 9.5% in Falconiformes, and 7.4% in Strigiformes; Table S15) and point to avenues of future research on this agriculturally important group (Shave *et al.*, 2018). Thus, *Salmonella* spp. likely exist at lower prevalence in wild birds than *Campylobacter* spp. and *E. coli* due to negative impacts on host health (Tizard, 2004; Hall & Saito, 2008), but high survival in the environment and possibly lower impacts on large birds such as the Pelecaniformes (17% prevalence) and Charadriiformes (8.4% prevalence) may lead to higher

prevalence in large, water-associated birds (Fig. S8; Table S15) (Winfield & Groisman, 2003; Tizard, 2004).

We found the least data on pathogenic *E. coli* of the three pathogens examined. The Galliformes (55% prevalence;) appear to be especially competent hosts (Figs S7 and S8; Tables S13 and S14), although it is unclear what role other species play due to low sample sizes. Pheasants may have had high prevalence due to captive rearing and release for hunting (Nebola *et al.*, 2007). Despite the associations between several bird taxa and particular pathogens described above, we find it likely that our results are a combination of research bias and competent pathogen–host–environment systems. Future research efforts will need to continue to determine what constitutes a highly competent pathogen–host–environment system by integrating all components of the host–pathogen cycle, from exposure to reservoir competence, contact, bacterial environmental survival, and colonization of a human host (Fig. 1).

We end with several conclusions from our review of the literature. First, the data are too limited and biased currently to make any data-driven recommendations for managing wild birds to reduce enteric pathogen spillover to people or livestock. Beyond collecting data on understudied species, we suggest a few key pieces of data will be most crucial to advancing policy to reduce enteric pathogen transmission between humans and wild birds. First, robust studies demonstrating that wild birds and humans/livestock share the same strains are needed (Table S10). Experiments that inoculate wild birds with bacterial strains of human, livestock, and wild bird origin will provide the strongest evidence (e.g. Atterby *et al.*, 2018). Current evidence suggests wild birds are often poor reservoir hosts of human strains (Waldenström *et al.*, 2010; Atterby *et al.*, 2018). Second, experiments determining the long-term shedding potential of enteric pathogens by wild birds are crucial. Understanding long-term shedding potential will help to determine if species commonly infected in winter such as birds at feeders (Tizard, 2004) can shed enteric bacteria in summer when children commonly contact faeces outdoors and most crops are grown. Third, studies must quantify contact rates, direct and indirect, in developing risk assessments. Finally, determining the shedding intensity and subsequent survival of bacteria in wild bird faeces, particularly for songbirds, is a key piece of missing information from the literature. Bacteria in bird faeces must survive long enough at an infectious dose until consumed by a human to potentially infect a human host, so shedding intensity and subsequent survival data are crucial for risk models. Without these four key pieces of information, studies could over-estimate risk if only considering the most basic estimate of prevalence. It is likely that the current focus on pathogen prevalence has over-inflated the perceived risk of wild birds to human health.

VI. CONCLUSIONS

(1) Wild birds are often thought to pose a risk to human health through transmission of enteric pathogens, yet few data are available to assess this idea. Here, we developed a comprehensive framework for understanding spillover of enteric pathogens from wild birds to humans and identified where important knowledge gaps remain.

(2) Only 3% of studies included in our meta-analysis provided data from all phases in the pathogen transmission process included in our conceptual framework. Most studies provided data limited to pathogen prevalence, but even prevalence data were limited to a small number of common species (e.g. rock pigeons, European starlings, mallards, Canada geese, house sparrows, herring gulls). No pathogen prevalence data were available for 65% of North American breeding bird species, including many commonly in contact with humans and agricultural production (e.g. many Passeriformes and raptors).

(3) We found an overall *Campylobacter* spp. prevalence of 27%, pathogenic *E. coli* prevalence of 20%, and *Salmonella* spp. prevalence of 6.4%. These estimates were derived from data from only 14.8% of North American breeding bird species for *Campylobacter* spp., 5.3% of bird species for pathogenic *E. coli*, and 33% of bird species for *Salmonella* spp. Given that most bird species in North America are understudied or entirely untested as reservoirs of enteric bacteria, it remains unknown how important a role most birds play in the risk of enteric pathogen transmission to humans.

(4) The primary focus in the literature on pathogen prevalence data likely overestimates the probability of enteric pathogen spillover from wild birds to humans because a pathogen from bird faeces must survive at an infectious dose until consumed by humans and be a strain able to cause disease in humans. For example, although we estimated a *Campylobacter* spp. prevalence of 26% in mallards (apparently high risk), Colles *et al.* (2011) estimated that only 0.9% of mallard *Campylobacter* spp. isolates cause human disease, suggesting the prevalence of human pathogenic *Campylobacter* spp. strains is about 2.3 in 1000 mallards, much lower than the *Campylobacter* genus prevalence alone suggests.

(5) We conclude that the current research is not sufficient for estimating the risk of enteric pathogen spillover from wild birds to humans. Future research should focus on the large number of under-studied species commonly in contact with people and food production, identify if bacteria are human-pathogenic strains, and model the entire transmission process. Fully assessing the probability of enteric pathogen spillover from wild birds to humans is complex and data intensive. Yet, the only way to develop data-driven and effective management strategies for reducing pathogen transmission is to understand the entire transmission process fully. Only upon doing this can effective policy be implemented to reduce spillover.

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IX. Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

S1. Supplementary tables and figures.

Data S1. List of studies and their methods included in meta-analysis.

Data S2. Meta-data on enteric pathogen prevalence in North American breeding bird species.

Data S3. Meta-data of enteric pathogen prevalence for each study included in meta-regressions.

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