Retrospective Study of *Campylobacter* Infection in a Zoological Collection

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Little is known about the epidemiology of *Campylobacter* spp. in wild animal populations. However, zoological collections can provide valuable insights. Using records from the Zoological Society of London Whipsnade Zoo compiled between 1990 and 2003, the roles of a range of biotic and abiotic factors associated with the occurrence of campylobacteriosis were investigated. The occurrence of campylobacteriosis varied widely across host taxonomic orders. Furthermore, in mammals, a combination of changes in both rainfall and temperature in the week preceding the onset of gastroenteritis were associated with isolation of *Campylobacter* from feces. In birds, there was a weak negative correlation between mean weekly rainfall and isolation of *Campylobacter* from feces. Importantly, in birds we found that the mean weekly rainfall 3 to 4 weeks before symptoms of gastroenteritis appeared was the best predictor of *Campylobacter* infection. *Campylobacter*-related gastroenteritis cases with mixed concurrent infections were positively associated with the presence of parasites (helminths and protozoans) in mammals, while in birds *Campylobacter* was associated with other concurrent bacterial infections rather than with the presence of helminths and protozoans. This study suggests that climatic elements are important factors associated with *Campylobacter*-related gastroenteritis. Further investigations are required to improve our understanding of *Campylobacter* epidemiology in captive wild animal populations.

Investigation of and understanding wildlife diseases are vital aspects of natural resource management programs. The need to conserve endangered species and the development of semi-free-range wild animal parks have attracted attention to the potential transmission of infectious microorganisms, their impact on the health of individuals and groups of wild animals, and their effect on conservation programs (47).

Animals kept in captivity or bred in semi-free-range areas, such as zoological gardens, may become infected with enteropathogens in their enclosures (1). Thermophilic *Campylobacter* spp. have been isolated from the intestinal tracts of a wide variety of healthy and diseased warm-blooded animals, including domestic species and captive and free-living wild animals (33, 34, 46, 48, 49, 50, 53). Furthermore, many species can be asymptomatic carriers (28). Wild and domestic birds act as major vertebrate reservoirs of *Campylobacter* spp. (25, 29, 46, 48) and play a role in the epidemiology of the disease (6, 56). In addition, adult house flies (*Musca domestica*) (45) and the tenebrionid beetle *Alphitobius diaperinus* (4) have been recorded as a potential nonvertebrate reservoirs for *Campylobacter* spp.

Although captive and free-living wild animals can be healthy asymptomatic carriers of *Campylobacter* spp., previous research reports showed that *Campylobacter* spp. were linked to many disease outbreaks in semiwild and wild animals, with negative effects on the health, productivity, and welfare of a variety of species. *Campylobacter jejuni* was implicated in a serious episode of abortions in mink (*Mustela vison*) on a ranch in central Ontario, Canada (23). Moreover, *C. jejuni* and *Campylobacter coli* were thought to be the causative agents of an outbreak of colitis with severe diarrhea and death in weanling mink kits (24). *C. jejuni* was associated with diarrhea and intestinal intussusceptions in a raccoon (*Procyon lotor*) (19). *Campylobacter fetus* had a clear impact on the productivity of wild free-living Dall’s sheep (*Ovis dalli dalli*) in Alaska (15). Furthermore, *C. fetus* was considered the cause of placental lesions and fetal death of a rhesus monkey (*Macaca mulatta*) (5). *Campylobacter* species, especially *C. jejuni*, are some of the pathogens that are isolated most frequently from diarrheic and healthy captive and free-ranging nonhuman primates (36, 38, 42, 47, 54, 2). *C. fetus* subsp. *fetus* was reported to cause retarded molting and high mortality in tropical finches, especially among fledglings (14). *C. jejuni* was associated with enteritis and hepatitis in captive ostrich chicks (*Struthio camelus*) (22). Recently, *C. jejuni*, *Campylobacter lari*, and *Campylobacter insulaeignae* were isolated from stranded and rehabilitated marine mammals, but their roles in causing disease in these species were not determined (16, 51, 52).

Contaminated water and food and direct contact with infected animals are the main sources of sporadic infection in both humans and other mammals (49). Contamination of the environment by domestic and wild animal feces is an alternative exposure pathway (7, 17). Although there have been a few reports of potential transmission of *Campylobacter* spp. from breeder hens to their offspring (10, 12, 13), these findings were not well supported and vertical transmission was not considered a significant source of contamination of chicken flocks with *Campylobacter* spp. (9).
Whipsnade Zoo (WZ) is a 243-ha zoological collection owned by the Zoological Society of London (ZSL) in Bedfordshire, United Kingdom. WZ has over 2,500 mammals, birds, reptiles, and amphibians in its collection, and these animals belong to more than 100 species and include free-ranging populations of Chinese water deer (Hydropotes inermis), Bennett’s wallabies (Macropus rufogriseus frutica), mara (Dolichotis patagonum), Reeve’s muntjac (Muntiacus reevesi), common pheasant (Phasianus colchicus), and jungle fowl (Gallus gallus).

Between 1990 and 2003 Campylobacter spp. were isolated from a variety of diseased and healthy species at WZ. Changes in captive animal management plans and environmental stressors, such as shipment, overcrowding, diet, temperature, ventilation, humidity, and the frequent presence of humans, contribute to shedding of Campylobacter spp. and increase the risk of horizontal transmission. These conditions provide the opportunity for intra- and interspecific transmission of infectious agents through the oro-fecal route. Differences in living conditions may influence the frequency of Campylobacter isolation (32). Previous studies of Campylobacter spp. at WZ showed that the prevalence of Campylobacter spp. in the Asian ungulates may have been affected by their stocking density (53). In contrast, there was no statistical difference between the prevalence of Campylobacter spp. in intensively stocked mara populations and the prevalence of Campylobacter spp. in extensively stocked mara populations (35). However, previous exposure, the virulence of ingested organisms, the infective dose, the rate of exposure, and the development of immune defense play roles in Campylobacter-associated disease manifestations (18), as does the animal species affected (31).

One of the motivations for the current analysis was that in 2001 Campylobacter spp. were isolated from three neonatal axis axis with enteritis and were determined to be one factor in their death. Since colonization of the gastrointestinal tract by Campylobacter spp. is generally not associated with illness and some species can be asymptomatic carriers, an important and unanswered question remains: what factors cause infection to reach levels which result in clinical symptoms of gastroenteritis? Therefore, the aim of this study was to determine the effects of potential biotic variables (parasites, including helminths, protozoans, and other pathogenic bacteria) and abiotic covariates (climatic factors, including temperature, ventilation, humidity, and the frequent presence of humans, contribute to shedding of Campylobacter spp. and increase the risk of horizontal transmission. These conditions provide the opportunity for intra- and interspecific transmission of infectious agents through the oro-fecal route. Differences in living conditions may influence the frequency of Campylobacter isolation (32). Previous studies of Campylobacter spp. at WZ showed that the prevalence of Campylobacter spp. in the Asian ungulates may have been affected by their stocking density (53). In contrast, there was no statistical difference between the prevalence of Campylobacter spp. in intensively stocked mara populations and the prevalence of Campylobacter spp. in extensively stocked mara populations (35). However, previous exposure, the virulence of ingested organisms, the infective dose, the rate of exposure, and the development of immune defense play roles in Campylobacter-associated disease manifestations (18), as does the animal species affected (31).

MATERIALS AND METHODS

Data collection. ZSL holds microbiological records collected from gastrointestinal cases examined for Campylobacter spp. for the zoological collection at WZ. The present study examined a database of these records from 1990 to 2003. In accordance with the microbiology protocol of the ZSL Veterinary Department, samples from mammals, birds, and reptiles which exhibited diarrhea and other gastrointestinal diseases were tested for Campylobacter spp. within 24 h of collection of the samples. In addition, these samples were screened for other pathogenic microorganisms. The data included the date that each sample was screened for Campylobacter spp., the taxonomic level (class, order, species), the result of the screening (positive or negative for Campylobacter spp.), and the presence of other pathogenic bacteria or endoparasites (helminths and protozoans) along with Campylobacter-positive isolates. Of the 1,747 samples examined for Campylobacter spp., 546 were excluded. Samples were excluded for one of three reasons: there was no evidence of gastroenteritis (samples obtained from either animals during routine health checks or newly imported animals); there was pseudoreplication (postmortem records for samples taken from different tissues of the same individual); or samples were obtained from water rather than individual animals (n = 8). Furthermore, samples from the reptile orders (n = 28) and domestic species (n = 41) tested were not subjected to investigation and were excluded due to small sample sizes. This resulted in a total of 1,201 samples from mammals (n = 683) and birds (n = 518) that were used for analysis.

All samples in this study were directly plated onto modified charcoal cefaperazone deoxycholate agar (Greenpeace, London, United Kingdom) without any primary enrichment. The samples were incubated at 37°C in a microaerophilic atmosphere (5% O2, 10% CO2, 85% N2) for 48 h. Positive isolates were identified as Campylobacter spp. on the basis of colony morphology on charcoal cefaperazone deoxycholate agar, on the basis of morphology determined by microscopic examination of a Gram-stained smear, and by using the oxidase test (11, 44).

Climate data for ZSL WZ between 1990 and 2003 were collected from the monthly reports provided by the Rothamsted Meteorological Station (a branch of the National Meteorological Office, United Kingdom) containing daily temperature (minimum and maximum) and rainfall figures. From these data, the average temperatures and total rainfall amounts were calculated for 1- to 4-week periods prior to the date of collection for each specimen submitted for Campylobacter examination.

Data analysis. Mammals and birds were analyzed separately. Generalized linear mixed-effects models (43) with logit link functions and binomial error structures were fitted to each of the two binary (presence or absence) Campylobacter data sets. The abiotic fixed effects were mean weekly “temperature” and natural log-transformed mean weekly “rainfall” recorded for the week preceding the identification of gastroenteritis (lag 1), as well as for the previous 3 weeks (lags 2, 3, and 4). The biotic fixed effect was the presence or absence of other “bacteria” and “parasites” (protozoans and helminths), which was determined at the time that gastroenteritis symptoms were observed and was not time lagged. The taxonomic order of the host animal was fitted as a random effect in all mixed-effects models. Fitting all fixed effects to our campylobacteriosis data, as well as all possible statistical interactions, would have resulted in a poorly specified, overparameterized model. Initially, we fitted biotic variables along with single temporal lag rainfall and temperature measurements. Therefore, four models were constructed separately for mammals and four models were constructed for birds, one for each temporal lag, incorporating all interaction terms with biotic factors. In order to ascertain appropriate model complexity, a comparison of simpler models, nested within each of the four full models, was performed using Akaike’s information criterion (AIC) (8). A difference in AIC of less than 2 between the best fitting and competing (nested) models indicated that the alternative model competed effectively for explaining the observed prevalence. Thus, the best-fitting nested models indicated the overall complexity in terms of statistical interactions between biotic and abiotic factors. We then applied the same effects modeling, incorporating all temporal lags of temperature and rainfall simultaneously along with the presence or absence of other bacteria and parasites, on this complexity of statistical interactions. Since no interactions were found between the two broad classes of explanatory variables (biotic [bacteria and endoparasites] and abiotic [lagged weekly rainfall and temperature]) in any of the single lag models, the final models (separate models for mammals and birds) included all four lagged “rainfall” and “temperature” main effects additionally, as well as terms for interactions between “rainfall” and “temperature” at each lag but not across temporal lags. In addition, “bacteria” and “parasites” were included, allowing an interaction between these two biotic covariates. Again, “order” was included as a random effect, allowing both random intercepts and random interactions with each of the fixed main effects. The data were analyzed using the “R” Project software for statistical computing (the R Project can be downloaded from http://www.r-project.org/).

Disease seasonality was assessed using all recorded data except the data for 1990 to 1992 due to missing monthly information for these 3 years.

RESULTS

The proportion of animals found to harbor Campylobacter belonging to each of the orders of mammals and birds tested at WZ is shown in Table 1, along with details of the number of species tested in each order. Summaries of seasonal effects,
which were not found to be significant, are shown in Fig. 1 and 2. Separate generalized linear mixed-effects models were fitted to explain differences in the observed levels of *Campylobacter* in mammals and birds.

**Mammals. (i) Single time lag models.** Across the whole range of mammals having symptoms of gastroenteritis, there was a significant increase \((t = 2.04, \text{df} = 717, P = 0.042)\) in occurrence of campylobacteriosis with increasing temperature and a less well-supported \((t = 1.84, \text{df} = 717, P = 0.067)\) increase in incidence with increasing rainfall in the week preceding the appearance of symptoms. However, the significant negative interaction between temperature and rainfall \((t = 2.08, \text{df} = 717, P = 0.038)\) indicates that the increases in the occurrence of *Campylobacter* spp. were reduced when both the levels of rainfall and the temperature were high. Models including temperature and rainfall measurements more than 1 week before symptoms occurred (lag 2, 3, or 4) did not support inclusion of temperature or rainfall as an explanatory fixed effect. In addition, we found that the occurrence of campylobacteriosis was positively associated with the presence of other parasites \((t = 2.01, \text{df} = 717, P = 0.045)\).

(ii) **Multiple time lag model.** Results from single time lag models provided strong support for restricting a multiple time lag model to only main effects and first-order statistical interaction terms. Analysis of variance for this fitted model corroborated our initial finding that temperature and rainfall only in the week before symptoms occurred interacted in order to explain the observed patterns of the occurrence of campylobacteriosis (Table 2). As described above, we found that the interaction between temperature and rainfall was negative.

**Birds. (i) Single time lag models.** As observed with mammals, the final models for birds were the most parsimonious models, based on AIC, nested within full models including all possible interactions between fixed effects. Summarized results for the four best-fitting models indicated that an increase in the occurrence of campylobacteriosis with increasing rainfall, both 3 weeks \((t = 1.79, \text{df} = 500, P = 0.074)\) and 4 weeks \((t = 1.80, \text{df} = 500, P = 0.073)\) prior to the onset of symptoms of gastroenteritis, was weakly supported. The presence of other pathogenic bacteria was also positively associated with the presence of *Campylobacter* \((t = 2.08, \text{df} = 500, P = 0.038)\).

(ii) **Multiple time lag model.** Although modeling time lags separately for birds indicated that only rainfall and bacteria explained the observed deviation in the occurrence of campylobacteriosis with any degree of confidence, we took a conservative approach and modeled *Campylobacter* in birds with the same overall model structure that was used for mammals (Table 3). We found that this full model broadly supported our single-model findings for birds (for rainfall lag 1, \(F_{1,487} = 0.35\)).

### Table 1. Occurrence of *Campylobacter*-related gastroenteritis in members of different orders in the zoological collection at ZSL WZ from 1990 to 2003

| Class      | Order         | No. of species | No. of positive samples | Total no. of samples | % of positive samples |
|------------|---------------|----------------|-------------------------|----------------------|----------------------|
| Mammals    | Diprotodontia | 3              | 4                       | 18                   | 22                   |
|            | Primates      | 11             | 12                      | 82                   | 15                   |
|            | Rodentia      | 4              | 11                      | 136                  | 8                    |
|            | Carnivora     | 8              | 4                       | 78                   | 5                    |
|            | Pinnipedia    | 1              | 0                       | 9                    | 0                    |
|            | Proboscidea   | 1              | 0                       | 14                   | 0                    |
|            | Perissodactyla| 6              | 1                       | 62                   | 2                    |
|            | Artiodactyla  | 36             | 43                      | 284                  | 15                   |
| Birds      | Casuariiformes| 2              | 1                       | 10                   | 10                   |
|            | Sphenisciformes| 3             | 4                       | 37                   | 11                   |
|            | Ciconiiformes | 4              | 16                      | 47                   | 34                   |
|            | Anseriformes  | 25             | 31                      | 104                  | 30                   |
|            | Falconiformes | 16             | 0                       | 48                   | 0                    |
|            | Galliformes   | 7              | 4                       | 27                   | 15                   |
|            | Gruiformes    | 8              | 52                      | 119                  | 44                   |
|            | Charadriiformes| 2             | 4                       | 15                   | 27                   |
|            | Psittaciformes| 7              | 0                       | 23                   | 0                    |
|            | Strigiformes  | 6              | 0                       | 23                   | 0                    |
|            | Coraciiformes | 3              | 0                       | 19                   | 0                    |
|            | Piciformes    | 2              | 0                       | 6                    | 0                    |
|            | Passeriformes | 4              | 5                       | 37                   | 14                   |
|            | Pelecaniformes| 1              | 0                       | 3                    | 0                    |
and $P = 0.55$; for lag 2, $F_{1, 487} = 0.19$ and $P = 0.67$; for lag 3, $F_{1, 487} = 3.73$ and $P = 0.05$; and for lag 4, $F_{1, 487} = 0.006$ and $P = 0.94$), with support for a negative association between the occurrence of campylobacteriosis and mean weekly rainfall 3 weeks before the onset of symptoms and a positive association between campylobacteriosis and other bacteria.

**DISCUSSION**

This study highlighted the biological and environmental influences which should be considered in order to understand the epidemiology of *Campylobacter* spp. in a zoological collection.

Across the whole range of mammals having symptoms of gastroenteritis, there was a significant increase in the occurrence of *Campylobacter* spp. with increasing temperature. At low temperatures there was some support for an increase in the occurrence with increasing rainfall, but this was not observed at higher temperatures, at which there was a significant negative interaction between temperature and rainfall. Similar results have been reported for humans, in which there was a positive association between the occurrence of campylobacteriosis and both temperature and the number of hours of sunlight. However, this effect was slightly mitigated by high humidity (40). It was hypothesized that the association with humidity was due to the inverse relationship between humidity and the number of hours of sunlight. The main difference between our results and those of Patrick et al. (40) is that Patrick et al. reported that a 3- to 4-week lag in climatic conditions was the best predictor of the occurrence of campylobacteriosis in humans, whereas we found that the week immediately preceding the occurrence had the greatest explana-

**TABLE 2. Analysis of variance for the full model fitted to the prevalence of *Campylobacter* in mammals, incorporating only main effects and the interaction terms found to be important using initial, single time lag models**

| Fixed effect* | df | Residual df | F value | P value |
|---------------|----|-------------|---------|---------|
| Temp lag 1    | 1  | 706         | 0.182   | 0.67    |
| Temp lag 2    | 1  | 706         | 0.0411  | 0.84    |
| Temp lag 3    | 1  | 706         | 0.0352  | 0.85    |
| Temp lag 4    | 1  | 706         | 0.235   | 0.63    |
| Rain lag 1    | 1  | 706         | 0.00002 | 1.00    |
| Rain lag 2    | 1  | 706         | 2.829   | 0.09    |
| Rain lag 3    | 1  | 706         | 0.0858  | 0.77    |
| Rain lag 4    | 1  | 706         | 2.08    | 0.15    |
| Bacteria      | 1  | 706         | 2.89    | 0.09    |
| Parasites     | 1  | 706         | 11.4    | <0.001  |
| Temp:rain lag 1| 1  | 706         | 4.52    | 0.03    |
| Temp:rain lag 2| 1  | 706         | 3.27    | 0.07    |
| Temp:rain lag 3| 1  | 706         | 1.03    | 0.31    |
| Temp:rain lag 4| 1  | 706         | 1.88    | 0.17    |
| Bacteria:parasites | 1  | 706       | 4.33     | 0.04    |

* Lag indicates a weekly time lag. Bold type indicates fixed effects explaining a significant amount of the observed deviation at $\alpha = 0.05$. A colon indicates a statistical interaction.

**TABLE 3. Analysis of variance for the full model fitted to the prevalence of *Campylobacter* in birds, with main effect and statistical interaction terms allowing comparison with mammals**

| Fixed effect* | df | Residual df | F value | P value |
|---------------|----|-------------|---------|---------|
| Temp lag 1    | 1  | 487         | 0.762   | 0.38    |
| Temp lag 2    | 1  | 487         | 0.0543  | 0.82    |
| Temp lag 3    | 1  | 487         | 0.247   | 0.62    |
| Temp lag 4    | 1  | 487         | 3.21    | 0.07    |
| Rain lag 1    | 1  | 487         | 0.351   | 0.55    |
| Rain lag 2    | 1  | 487         | 0.185   | 0.67    |
| Rain lag 3    | 1  | 487         | 3.73    | 0.05    |
| Rain lag 4    | 1  | 487         | 0.00572 | 0.94    |
| Bacteria      | 1  | 487         | 4.49    | 0.03    |
| Parasites     | 1  | 487         | 1.49    | 0.22    |
| Temp:rain lag 1| 1  | 487         | 0.751   | 0.39    |
| Temp:rain lag 2| 1  | 487         | 0.291   | 0.59    |
| Temp:rain lag 3| 1  | 487         | 0.872   | 0.35    |
| Temp:rain lag 4| 1  | 487         | 1.42    | 0.23    |
| Bacteria:parasites | 1  | 487       | 1.22     | 0.27    |

* Lag indicates a weekly time lag. Bold type indicates fixed effects explaining a significant amount of the observed deviation at $\alpha = 0.05$. A colon indicates a statistical interaction.
tory power for mammals. However, Patrick et al. noted that on average there was a 21-day delay between the onset of symptoms and reporting to medical authorities. Our results are further corroborated by another investigation of the seasonality of enterically transmitted diseases and ambient temperature (37), which demonstrated that the peak daily occurrence of campylobacteriosis in humans closely followed the peak ambient temperature with a lag of 2 to 14 days. At WZ, all animal species whose conservation is important are regularly checked in addition to regular observation of free-ranging species, the fecal worm egg count program, and veterinary examination of any ill animal. This is likely to be the reason that the time lags found in the human studies were not evident in our results. We believe that this is strong evidence for the hypothesis that in mammals the occurrence of Campylobacter-related gastroenteritis may be predicted by the weather conditions immediately prior to the onset of symptoms. However, it has also previously been reported that temperature may have a direct effect either on Campylobacter carriage or on vectors (26), indicating a potential mechanism for a temporal lag between transient weather conditions and the occurrence of Campylobacter spp. in some host species. Temperature may also have an indirect effect on the rate of Campylobacter shedding by necessitating changes in the animal management plan. For example, when domestic sheep (Ovis aries) were housed indoors due to cold weather conditions and supplied with feedstuffs, a low rate of shedding was observed (27). This variability in shedding might have been due to less exposure to sources of environmental contamination with Campylobacter spp.

In Campylobacter-related gastroenteritis cases with mixed concurrent infections, we found that the occurrence of campylobacteriosis is positively associated with the presence of a range of other pathogens, most notably parasites (helminths and protozoans). It has been suggested that shedding of Campylobacter spp. may be precipitated by parasitism (30). In addition, C. jejuni can infect amoebae (Acanthamoeba polyphaga) in vitro, and these or other protozoans might serve as potential vectors (3). Therefore, it is possible that protozoan species play a role in the transmission of Campylobacter spp. and maintain infections. Taken together, these observations correspond with the results of this study. However, more investigations should be considered as climatic factors may also influence the proliferation of parasitic diseases (41).

Our results for birds at WZ differ from those for mammals in a number of interesting ways. All birds sampled during the period investigated were examined due to suspicions of gastrointestinal disease, and therefore, the sample did not include many possible asymptomatic carriers. We did not find a significant association between temperature and the occurrence of campylobacteriosis, despite the sample size (518 birds, compared to 683 mammals). However, we did find some support for a negative association between rainfall and the occurrence of Campylobacter-related gastroenteritis. Moreover, this association was found only for mean weekly rainfall 3 or 4 weeks prior to detection of Campylobacter. However, Campylobacter is known to be intolerant of dry conditions (39), so the observed (weak) trend appears to be at odds with predictions. While the most rigorous way to explore this result further would involve experimental manipulation of environmental conditions, a more practical and ethical approach would require regular sampling surveys of wild birds and their environment, correlating the results with the temperature and rainfall. However, given that the birds at WZ benefit from the same close health monitoring as mammals, it seems likely that the difference in temporal dynamics represents the effects of a real biological mechanism in the disease process.

For birds, as for mammals, we found a positive association between the occurrence of campylobacteriosis and the presence of other pathogens. We found that Campylobacter-related gastroenteritis in birds is associated with other concurrent bacterial infections rather than the presence of helminths and protozoans. Enteric challenge with a variety of organisms and the synergistic interaction between parasites and bacteria may influence Campylobacter-related gastroenteritis and associated disease manifestations. The difference in broad types of associated pathogens is informative for health management of wild animal parks.

In general, the susceptibility of species to enteric diseases is regulated by many factors. It may be that mammalian species have different susceptibilities to colonization by C. jejuni whatever the degree of exposure to this bacterium (46). For Campylobacter spp. in rhesus monkey (Macaca mulatta) there were significant differences among age and sex groups (2). Furthermore, the behavior of different species may affect their propensity to become infected with Campylobacter spp. (53). Therefore, the low frequency of Campylobacter-related gastroenteritis in carnivores and perissodactyls might be influenced by species and behavioral differences. For many animals, foraging behavior, diet, and habitat might affect the likelihood of encountering Campylobacter spp. In a previous study, it was reported that shoreline-foraging birds and ground-foraging and opportunistic feeders were commonly infected by Campylobacter spp. Here, the prevalence of Campylobacter spp. was explained by ecological guilds, based on feeding habits (55). Similarly, in our study, Ciconiformes and Gruiformes, both feeding at the edges of ponds in their exhibit, showed a high prevalence of Campylobacter spp. Since Campylobacter spp. isolated from surface water have been found to be capable of surviving in different moist environmental conditions (20, 28), these animals are the most likely to be exposed to and harbor the bacteria from their habitats.

Previous investigations of the seasonal patterns of the prevalence of Campylobacter spp. showed that there were significant variations among the species studied (6, 40), the regions, and the countries (49). Furthermore, the rate of survival of Campylobacter spp. varied seasonally and daily in different sampled environments. This was attributed to the changes in temperature and other climatic factors (28, 37). In this study, we could not interpret differences in the occurrence of Campylobacter-related gastroenteritis among seasons for either mammals or birds (Fig. 1 and 2). Numerous factors are likely be involved in determining the levels of Campylobacter shedding and the development of gastroenteritis in different seasons. Our analysis did not account for relative humidity, the number of hours of sunlight, and the level of UV radiation. These factors are crucial in interpreting the effects of complex interactions among climatic factors (28, 40) on both the biological survival of Campylobacter spp. in the environment and the occurrence of these microorganisms in wild animals. In addition, seasonal differences might also be associated with the
activity of animals, such as migrations, and in turn contamination of the environment and the spread of Campylobacter spp. to other animals via feedstuffs or water (6, 21). Therefore, the link between seasonality and the occurrence of Campylobacter spp. is difficult to interpret when season is considered in isolation. In our view, more investigation is required through regular sampling surveys. These surveys should link all climatic factors that might be associated with the biological survival of Campylobacter spp. in the environment and the occurrence of Campylobacter spp. in wild animals. Further, in translating findings from such surveys into management decisions, practical responses to associations between Campylobacter incidence and covariates must be integrated with all other aspects of wild animal population health and welfare.

In conclusion, while our results represent an important retrospective study of Campylobacter epidemiology, we were not able to test any mechanistic explanations of cause and effect found in our statistical associations. Further investigations are required to determine the impact of climatic factors, particularly at different times of the year, in order to evaluate any seasonal variations based on climatic changes. Monitoring the movement of pathogens or vectors should facilitate minimizing the risk of infection and enable intervention procedures to be developed at WZ. Given that Campylobacter infection has been evaluated as an emerging disease, there is a pressing need to develop different models and risk assessment tools for Campylobacter infection. This is essential in order to improve our understanding of Campylobacter epidemiology, particularly in captive wild animal populations.

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