Campylobacteriosis, like many human diseases, has its own ecology in which the propagation of human infection and disease depends on pathogen survival and finding new hosts in order to replicate and sustain the pathogen population. The complexity of this process, a process common to other enteric pathogens, has hampered control efforts. Many unknowns remain, resulting in a poorly understood disease ecology. To provide structure to these unknowns and help direct further research and intervention, we propose an eco-environmental modeling approach for campylobacteriosis. This modeling approach follows the pathogen population as it moves through the environments that define the physical structure of its ecology. In this paper, we term the ecologic processes and environments through which these populations move “pathogen survival trajectories.” Although such a modeling approach could have veterinary applications, our emphasis is on human campylobacteriosis and focuses on human exposures to Campylobacter through feces, food, and aquatic environments. The pathogen survival trajectories that lead to human exposure include ecologic filters that limit population size, e.g., cooking food to kill Campylobacter. Environmental factors that influence the size of the pathogen reservoirs include temperature, nutrient availability, and moisture availability during the period of time the pathogen population is moving through the environment between infected and susceptible hosts. We anticipate that the modeling approach proposed here will work symbiotically with traditional epidemiologic and microbiologic research to help guide and evaluate the acquisition of new knowledge about the ecology, eventual intervention, and control of campylobacteriosis. Key words: aquatic environments, Campylobacter, campylobacteriosis, disease ecology, eco-environmental modeling, ecologic filters, feces, pathogen survival. Environ Health Perspect 111:19–28 (2003). [Online 7 November 2002] doi:10.1289/ehp.5312 available via http://dx.doi.org/111:19–28 (2003).
the structure of an eco-environmental model for campylobacteriosis. In this paper, we plan to lay the necessary groundwork for the future development of quantitative eco-environmental models for campylobacteriosis and other enteric diseases.

**Approaches to Modeling Human Disease Ecologies**

Models are human constructs that are superimposed upon real-world complexity. Therefore, a model can be described as a device used to simplify an object of study. Models come in all shapes and sizes and are used for many purposes. However, researchers have become used to the idea of using quantitative computer-based simulation models, particularly stochastic models used to describe relationships, e.g., regression models. Another type of model that may be less familiar to epidemiologists and public health professionals is the dynamic or process model.

Dynamic modeling underlies the proposed eco-environmental approach to campylobacteriosis. This type of modeling focuses on the components of the system, which drive important ecosystem dynamics. For example, changes in environmental temperature appear to play an important role in the survival of *Campylobacter* outside the animal host. Consequently, temperature is a variable important to the process of pathogen survival (20, 21). This dynamic modeling approach is well entrenched in the earth sciences (6) and disease vector modeling (13). Within the practice of dynamic modeling, a number of tools have been used, including deterministic modeling, stochastic modeling, and more recently, fuzzy logic, neural networks, genetic algorithms (22), implementation of Markov Chain Monte Carlo in application of Bayesian methods (23), and quantitative risk assessment methodologies (24). Regardless of the tools used, the focus should be on those components known to contribute to the dynamics of the system being modeled. Ultimately, modeling should lead to the development of a better understanding of those dynamics and refocus attention on areas where knowledge is lacking. The use of dynamic modeling in the study of human disease ecology has led to significant gains in knowledge that can be used to tackle new problems such as understanding potential impacts from climatic change (25). However, the modeling of disease ecology has yet to be applied to campylobacteriosis or other enteric diseases.

Anderson and May (19) have written what is arguably the single most important text on the modeling of human disease dynamics. Yet, enteric diseases such as campylobacteriosis are not covered in this important work. The reason for this omission can be surmised from their recollection that

Our interest in the work described in this book originally grew out of our attempts to understand the extent to which parasites—broadly defined to include viral, bacterial, protozoan, and fungal pathogens along with the more conventionally defined helminthic parasites—regulate the numerical abundance or geographical distribution of non-human animal populations (19).

Although without doubt their contributions are now focused on animals of the two-legged variety, enteric diseases have not in general "regulated the abundance or geographical distribution" of animals, particularly humans, at least in the developed world. Even in developing countries where mortality from enteric disease is high compared with other health issues, the availability of enteric disease interventions such as oral rehydration therapy (26) and engineering solutions that provide clean water have perhaps reduced interest in developing models to improve knowledge. There might be a greater sense of urgency in understanding the campylobacteriosis disease ecology if relatively simple interventions were known to be available and morbidity and mortality more severe. The modeling of human disease ecology has instead focused on the person-to-person spread of diseases between susceptible and infective hosts using mathematical devices such as the basic reproductive rate, \( R_0 \), for diseases with more dire outcomes (19).

\( R_0 \) is the principal mechanism used to model the number of secondary host infections that are produced from one primary infection. This is a particularly useful approach to take when the mechanism(s) of spread and environmental pathways of the pathogen are well understood, as is the case in person-to-person spread and some vector-borne diseases. However, it poses a problem when the environmental pathways of the pathogen are only partially understood, as is the case with most enteric disease (27). Modeling of enteric disease has consequently been limited to those enteropathogens whose primary mechanism of spread is person to person rather than those where animal reservoirs are the primary source of pathogens (26–29). Indeed, we have been unable to find any evidence of an attempt to model campylobacteriosis or similar enteropathogen zoonoses.

The way forward may be to construct a model disease ecology from the perspective of pathogens trying to survive until they find their next host, rather than the more traditional host-centric models typified in population dynamics-based modeling. Modeling pathogen survival as pathogen survival trajectories with various ecologic filtering processes (e.g., cooking food), intermediate environmental vectors (e.g., flies or food preparation surfaces), and associated constraints to survival (e.g., environmental temperature) constitutes an eco-environmental approach. This might also be termed a “Lagrangian” model that follows the flow of a particular group of pathogens through the environment, whereas traditional models calculate fluxes at fixed points within the model (e.g., time or space).

The advantage of this Lagrangian approach is that it directly focuses on the unknowns of greatest interest (i.e., the survivability of pathogens as they move through the environment to their next host).

**The Ecology of Campylobacteriosis**

Although campylobacteriosis is the most common known cause of gastrointestinal illness, our understanding of its ecology is incomplete. Overall, what we do know is that the *Campylobacter* spp. pathogens replicate almost exclusively within the intestinal tract of warm-blooded animal hosts (20), within a narrow temperature range of approximately 32–45°C (21). Pathogen dispersal from its animal host is through the excretion of feces or the contamination of an animal’s carcass by the intestinal contents during slaughter. It is this contamination of meat products that is believed to be the major source of campylobacteriosis in the human population (30). This belief is supported by case-control studies that have identified the consumption of untreated water, unpasteurized milk, and certain meats, often poultry, with the additional risk of campylobacteriosis (31–35).

Even if we accept that the major mechanism for the spread of campylobacteriosis is through food contamination, our understanding is incomplete. What is it about animal management practices and food processing systems that allow this pathogen to be so widely spread? Food preparation practices, which are the final safety check, do not appear to effectively remove *Campylobacter* pathogens. Why? The seasonal oscillation in human disease incidence is one of the most remarkable features of campylobacteriosis that we observe through human disease surveillance systems (Figure 1). This seasonal pattern of raised summer incidence appears in all countries where human campylobacteriosis is under surveillance (36).

There are at least two possible foodborne explanations for the observed seasonal oscillation in incidence. The first explanation is that during the warmer summer months, human exposure to the pathogens increases through outdoor grilling. It is thought that this might reduce the likelihood of thorough cooking and also increase the possibility of cross-contamination on relatively crowded barbecue grills (35, 37). A second explanation is seasonality in the number of animals with campylobacteriosis, which in turn drives the seasonality of human disease incidence (38).
However, there is contradictory evidence indicating that in some animal industries, carcasses may be contaminated throughout the year (38). Additionally, the seasonal increase in human cases has been acknowledged to precede seasonal rises in animal infections in some instances (38).

Non–food-related explanations for the seasonality of campylobacteriosis include the survival and consequent prevalence of Campylobacter in environmental reservoirs (39,40) and the seasonal effectiveness of the human immune system response (41). For a pathogen that is so difficult to culture in a laboratory, Campylobacter have been shown to have a rather remarkable capacity for survival in aquatic environments (42). Indeed, it appears that greater numbers of pathogens are found in aquatic environments during winter and spring periods because of the relatively lower water temperatures in winter (43). A potential relationship can be hypothesized between the seasonal accumulation of pathogens in the environment and the eventual rise in human disease during the following summer season. However, this is speculative, as we know of no direct studies of this potential relationship, and there are additional confounders such as the potential for increased human exposure through summer recreational contact with water.

The human immune system is another variable with a seasonal oscillation, and is impaired with greater exposure to ultraviolet (UV) radiation (41). This establishes another possible explanation that variation in immune response supports a component of the seasonality in human disease incidence, where the annual summer peak in notifications coincides with the population’s greatest exposure to UV radiation. The potential credibility for this mechanism has been established through the study of the role of the immune system in responding to campylobacteriosis and other enteric pathogens in a group of captive primates (41).

The viable but nonculturable (VNC) form of the Campylobacter pathogen has presented a major hurdle in developing an understanding of the disease ecology by limiting our ability to detect, classify, and quantify the Campylobacter pathogens in the environment. The literature on this topic of microbiologic techniques development is vast, and it is not reviewed here. Nonetheless, models will need to capture or account for our lack of knowledge stemming in part from our inability to see Campylobacter in the environment.

The physiologic role of the VNC form of Campylobacter is not clear, but its ability to use this form to survive in cold water has been demonstrated (44). Furthermore, there is some evidence that after surviving for considerable periods of time in aquatic environments, the passage of VNC Campylobacter through an animal host will restore it to its culturable form (45).

The dose–response relationship may also be an important component of the disease ecology of campylobacteriosis and may differ for various types and strains of the Campylobacter pathogen (46). The dose–response relationship for the number of pathogens required to cause disease is less certain than that required to cause infection. In one study, the greatest dose studied (1 × 10^8 pathogens) produced no disease in five nonimmune human subjects (46,47). A beta-Poisson model of the challenge studies of Black et al. (46) has been used to define a dose–response relationship for humans (48) and to assess the risk of consuming mussels (49). Although only a few challenge studies of human campylobacteriosis have been conducted, these studies do provide a starting point from which to base a modeling approach.

Although it is thought that many or even most cases of campylobacteriosis arise through the consumption of contaminated food and water, it is less clear how the pathogens move through the environment. The majority of campylobacteriosis cases appear sporadically and not in outbreaks, but there is an acknowledged occurrence of common-source milk (50,51) and waterborne disease outbreaks (52–54), which does nothing to clarify the disease ecology. The one mechanism of spread that can be modeled using established techniques is person-to-person spread, but this mechanism has not been widely implicated in the literature (55). Despite the many studies already published, uncertainty dominates our understanding of the ecology of campylobacteriosis, and this justifies an evaluation of the potential for applying dynamic modeling.

Pathogen Survival Trajectories: An Eco-Environmental Modeling Approach

An eco-environmental model for campylobacteriosis must focus on the environmental transmission pathways of the Campylobacter pathogen (Figure 2). This approach describes the ecology of campylobacteriosis from the perspective of a pathogen’s survival as it moves through the environment—a survival trajectory. We use the pathogen’s survival in the environment as the mechanism to quantify human exposure. Our lack of knowledge about pathogen survival hinders our ability to understand the ecologic dynamics and potential public health intervention points.

Figure 1. Seasonal pattern of campylobacteriosis in New Zealand. Aside from the steady rise in incidence, summer–winter seasonality dominates the temporal picture of campylobacteriosis in New Zealand, even from the period shortly after notifications were first recorded in 1980. Note that monthly data from 1986 were not archived [data from the Department of Heath (Wellington, New Zealand) records for before 1993 and from the New Zealand disease notification system (EpiSurv) for 1993 and later].
The *Campylobacter* pathogens replicate primarily within the intestinal tract of warm-blooded animals. Therefore, it is the postexcretion of pathogens that defines the ecology of campylobacteriosis, and all survival trajectories begin with pathogens being dispersed from animals through feces (Figure 2). Feces then serve as the primary environmental dispersal mechanism (Figure 2), including animals slaughtered for food processing, where the carcass is often contaminated by their *Campylobacter*-laden intestines. Secondary mechanisms of dispersion may occur, and all successful survival trajectories end with the new exposures (Figure 2).

There are three general nonoccupational pathogen survival trajectories: (a) direct exposure to feces; (b) exposure through food consumption, food processing, or food preparation activities; or (c) exposure through aquatic environments (Table 1). These mechanisms, which all describe oral routes for pathogen exposure, have associated ecologic filters that provide natural public health intervention points. In turn, the pathogen burden with which each of these filters must cope depends upon environmental factors that control the size of the pathogen load that arrives at a filter point (e.g., rainfall, stocking density, speed and magnitude of runoff, proximity to water body). Grouping pathogen survival trajectories according to common environmental elements (Table 1) facilitates comparative ecologic risk assessments of pathogen survival and human exposure routes (Figure 2). It is worth noting that the above ordering of ecosystem components is in order of increasing risk, identified through case-control studies. It also appears, at least at face value, to be in increasing order of the complexity of the pathogen survival trajectory. In other words, what might appear to be the best survival prospect for *Campylobacter* pathogen, from epidemiologic studies, appears to be the least survivable. This is a consequence of the relatively greater number of ecologic filters a pathogen would need to traverse to find a new host and replenish its numbers.

From an ecologic perspective, direct exposure to feces or indirect exposure to feces through human-to-human contact or animal-to-human contact would appear to be the most likely survival trajectory (Figure 2). Feces, as the direct excretion of materials from the intestines of warm-blooded animals, must be the environments of greatest *Campylobacter* concentration outside their animal hosts. From feces through the exposure of the next animal host, *Campylobacter* numbers must decrease. Their survival depends only on reaching their next host before their number declines below what is required for an infective dose.

Feces of warm-blooded animals must therefore represent the environmental site of greatest hazard. Animal hosts are many and varied, including birds and wild, domestic, and domesticated animals of all shapes and sizes as well as humans [Table 2 (55–82)]. There does not seem to be a shortage of *Campylobacter*-contaminated feces in the environment. From a modeling perspective, we might well consider the supply of pathogens...
from feces to be infinite. Why is it, then, that epidemiologic studies and disease surveillance information do not suggest that either direct feces contact or indirect (human-to-human or animal-to-human) feces contact to be a major component of the disease ecology? There may be at least two reasons why neither direct nor indirect contacts with feces are the major pathogen survival trajectories of Campylobacter. First, most human contact with animal feces is probably of an occupational nature in the agricultural and food industries.

### Table 2. Animal reservoirs of human Campylobacter pathogens from intestinal samples, fresh feces, or surfaces of freshly slaughtered carcasses.

| Animal reservoirs | Proportion of animals infected | Pathogen load | Seasonality and other notable study features | References |
|-------------------|-------------------------------|--------------|---------------------------------------------|------------|
| Humans            |                               |              |                                             |            |
| United Kingdom    | Poultry workers: short-term (<1 month) employees, one tested positive (n = 43); long-term employees, seven tested positive (n = 78) | —            | Only one short-term worker had symptoms indicating longer-term immunity in workers | [56] |
| Chile             | Clearance from food handlers averaged 17–19 days | —            | Associated analysis of domestic pets: dogs 43% (n = 214); hens 67% (n = 150); ducks 73% (n = 100); pigeons 11% (n = 104) | [57] |
| Wild animals and domesticated pets |                               |              |                                             |            |
| United States     | Thirteen orders of wild and domesticated birds 10% (n = 445) | —            |                                             |            |
|                   | Young dogs with diarrhea infected with C. jejuni | —            |                                             |            |
| Norway            | Urban Oslo: crows 90% (n = 48); gulls 50% (n = 54); pigeons 4% (n = 71) | —            | 11 of 40 species tested positive for C. fetus spp. C. jejuni | [59] (59), [60] |
|                   | Nonurban coastal area: puffin 51% (n = 76); common tern 6% (n = 28); common gull 19% (n = 37); black-headed gull 13% (n = 53) | —            | Another study found no evidence of C. jejuni in takeaway inhabited by over 600,000 water fowl | [61] |
|                   | Migratory waterfowl 35% (n = 445) | —            | 25% of gut contents of crows was human refuse | [62] |
| Japan             | Crows 34% (n = 87); blue magpies 20% (n = 10); gray starlings 14% (n = 36); domestic pigeons 13% (n = 16); bulbuls 11% (n = 36); eastern turtle doves 2% (n = 62) | —            | Considerable monthly variation in isolation rates was found | [63] |
| United States     | Migratory birds: sandhill cranes 81% (n = 91); ducks 73% (n = 113); Canada geese 5% (n = 94) | —            |                                             | [64] |
| Sweden            | Migratory passerines 3% (n = 101) | —            |                                             | [65] |
| Portugal          | Poultry 60% (n = 59); swine 59% (n = 65); black rats 57% (n = 31); sparrows 46% (n = 61); ducks 41% (n = 21); cows 20% (n = 32); sheep 15% (n = 27) | —            |                                             | [66] |
| Swine             | Norway 100% (n = 111) | —            | More than 1,200 wild and domestic mammals were surveyed | [67] |
|                   | Netherlands At slaughter (SI contents) 79%; carcasses postslaughter 9% | (4,000 cfu/g) ND | Dry cool conditions thought responsible for rapid Campylobacter die-off, while Salmonella persisted | [68] |
|                   | United States Gilts 76% (n = 50); pregnant sows 100% (n = 9); Newborn piglets 58% (n = 73); weaned piglets 100% (n = 20) | —            | 78% C. jejuni, 21% C. coli, and 3% C. lari | [69] |
|                   | Netherlands Pigs during fattening (feces), 85% (n = 7, n = 10) | 4.1 log N/g ND | 87% C. jejuni, 13% C. coli | [70] |
| Poultry broilers   | United States 50–100% Birds 27% (n = 12,233) | 4–16,000 | Spring has lowest flock positivity | [71]–[72] |
|                   | Netherlands Flocks 82% (n = 187) | —            | Flock positivity was seasonal, with 100% in June–September and 96% in March | [73] |
| Dairy herds        | United Kingdom Feces, 10 of 12 herds positive, 10–72% of test cows 4 average size dairy herds | ND | Spring and autumn, “evidence of true seasonality” | [74] |
|                   | United States Feces 38% (n = 2,085) | —            | Extensive study of farm practice including whether herds consumed chlorinated water, which appears to have no predictive effects | [75] |
|                   | New Zealand Feces, summer 24% (n = 72); autumn 31% (n = 106); winter 12% (n = 95) | —            | Rectal swabs taken in summer, autumn, and winter, with a fairly even split between C. jejuni and C. coli | [76] |
| Beef cattle        | United Kingdom At slaughter (SI contents) 89% (n = 360) | 6.1 × 10^2 MPN/g (SE = 2, n = 1,380) | No significant seasonal periodicity | [77] |
|                   | United Kingdom Calves (SI contents), ND | 3.3 × 10^4 MPN/g (SE = 180, n = 32) | ND | [78] |
| Sheep              | Norway Feces 8%, (n = 197) | —            | More than 1,200 wild and domestic mammals were surveyed in this study | [79] |
|                   | United Kingdom SI contents 92%; feces 30–46% | —            | Survived in feces for up to 4 days; shedding is seasonal, peaking with lambing, weaning, and movements to new pasture | [80] |
|                   | United Kingdom Lambs at slaughter (SI contents) 92% (n = 360) | 4 log 10 MPN/gfw (n = 1,080, SD = 0.16) to 7 log 10 MPN/gfw ND | Seasonality evident; time series not indicative of annual peaks | [81] |
|                   | United Kingdom Sheep (feces) 29% (n = 420) | —            | ND | [82] |

Abbreviations: cfu, colony-forming units; gfw, gram fresh weight; MPN, most probable number; ND, not done; NF, significant association not found; SI, small intestine.
processing sectors. Therefore, the number of humans exposed in this way is relatively small in developed countries, and there is some evidence that persons exposed in an occupational setting build up an immunity to these enteric pathogens (56). Second, there is evidence that the ecologic filter represented by personal hygiene is very effective (89). Even in situations where people are very exposed, such as changing babies’ diapers, washing one’s hands is likely to remove virtually all pathogens. Where hygiene is poor, as is expected of the very young (under 5 years of age), our disease surveillance information has shown an increased incidence of disease (1).

In terms of modeling human exposure to Campylobacter through direct and/or indirect contact with feces and to simulate the pathogen survival trajectories, we need to know the following:
• How many Campylobacter pathogens are excreted with feces?
• How long do Campylobacter survive in feces?
• How many can be transferred onto fingers, skin, fur, or other relevant intermediate environmental vectors?

### Table 3. Survival of Campylobacter pathogens in aquatic environments and on intermediate environmental vectors outside of animal hosts.

| Environmental setting | Positive environmental samples and pathogen load | Seasonality and other notable features | Reference |
|----------------------|------------------------------------------------|---------------------------------------|-----------|
| Finished drinking water environments New Zealand | 29% (n = 24), median MPN < 0.07 100/mL | Occurrence significantly higher when | (84) |
| Greece | 1% (n = 500) | coliform bacteria present, but no difference in frequency of occurrence between chlorinated and nonchlorinated water | (89) |
| Wastewater environments Netherlands | 2–50 × 10^2/100 mL | May–June peak, sewage effluent from abattoir and animal processing plants | (89) |
| United Kingdom | 2.2 × 10^2/100 mL to 5.1 × 10^3/100 mL | Positive samples year-round; E. coli not indicative of Campylobacter presence | (90) |
| 46% of pond samples and 45% of all drain samples were positive | | | |
| Netherlands | 80–1,600 MPN/100 mL | Minimum June–August | (91) |
| Italy | 630–3,200/100 mL | Maximum May–July | (91) |
| Aquatic environments Greece | 16% (n = 86), < 10/100 mL | Contamination not predicted by the standard indicator bacteria | (92) |
| New Zealand | Rivers 60% (n = 30), median MPN 0.18/100 mL | Recovery rates highest in fall/winter, lowest in spring and summer | (84) |
| Shallow ground water 75% (n = 18), median MPN 0.12/100 mL | | | |
| United States, Washington State | Sampled a number of mountain streams and lakes | Recovery rates highest in fall/winter, lowest in spring and summer | (93) |
| United Kingdom | 22% (n = 49), 10–230 MPN/100 mL | Coastal and estuary samples | (94) |
| 16% (n = 44), 10–360/100 mL | River samples | | |
| Filtration method 43% (n = 312); 21% by MPN (< 10 Campylobacter/100 mL) | Autumn and winter peaks; greatest MPN downstream of sewage outfalls; rural and urban samples | (39) |
| Groundwater spring contamination, isolated from 550-mL and 100-mL filter enrichment | Not isolated in the absence of fecal indicators | (95) |
| Netherlands | 46% of pond samples and 45% of all drain samples were positive | Higher numbers in winter; lower or none in May, June, and July; negative correlation with infection incidence in community | (38) |
| United Kingdom, river, canal, estuary | Seasonal variation in C. jejuni, C. coli, UPTC, C. lari, with higher numbers found in winter | | (96–98) |
| Human skin and hair surfaces Fingertips | Suspensions of 10^6–10^7 C. jejuni dried on fingertips for 1–4 min | Organisms removed by hand washing with either soap and water or just water and drying hands on paper towels | (83) |
| Food surfaces United Kingdom | Grown at 37°C on high-pH meat (pH 6.4), but not on normal pH meat (pH 5.8); population decay rates same for both pHs; very slow decay rate at 1°C for high-pH meat | 83% chicken samples positive; all meats had some positives; C. jejuni, 57 sero/phage types | (99) |
| Kitchen meat samples 73% (n = 489); | Multiple visits to four large commercial kitchens; Internal and external swabs of various meats | (100) |
| Chicken giblets 41%, thawed chicken juices 22%, fresh chickens 88% (n = 34) | | (101) |
| Insect vectors House flies | Sample of 32 house flies allowed to ingest C. jejuni, 20% recovery from feet and ventral surface, 70% recovery from viscera | | (102) |
| House flies | Chicken farm 51% (n = ?) Piggery 43% (n = ?) | Authors suggest that flies may be an important vector between animals | (103) |
| Other Beach sand | 45% (n = 182) and > 30% dry sand samples also contaminated | Presence greater in wet sand, but still 30% of dry sand samples positive | (104) |
| Beach sand | Sediment samples showed no seasonality, unlike water samples taken at the same time; sediment samples had greater numbers of Campylobacter than overlying water samples | | (96) |

Abbreviations: ?, unknown; MPN, most probable number; UPTC, urease-positive thermophilic campylobacters.
to be much greater with respect to food and water-exposure mechanisms.

There are three major issues: a) complexity of the survival trajectories in terms of the environmental constraints and ecologic filters; b) uncertainty in terms of the VNC state of Campylobacter; and c) the variability among strains of Campylobacter. Many studies have found Campylobacter in aquatic environments; in fact, its recovery is common and widespread [Table 3 (38,39,83–104)]. Sewage treatment (86–91) and water-treatment processes (84,85) are less than perfect ecologic filters. Animal access to drinking water catchments and proximity to rivers would seem to be an important factor in determining exposure through aquatic ecosystems. The role of birds in the survival trajectory of the pathogens may be especially important in the longer-distance movement of Campylobacter (Table 2).

The VNC state of Campylobacter, also referred to as a cocoid form, introduces additional uncertainty into pathogen survival trajectories through aquatic environments. Uncertainty arises in the interpretation of many observational studies of the disease ecology by increasing the number of false-negative analyses where Campylobacter exists but is not found because it is in the VNC state. Additionally, infectivity after recovery of Campylobacter in the intestinal tract of animal hosts appears possible, but is not well documented (105). Changes in virulence after its recovery may also be possible (45), and the modification of its genotypic structure may be obfuscating attempts to observe flows in the environment (106).

Drinking water contaminated with Campylobacter pathogens appears to be an efficient exposure mechanism. The degree to which water is regularly contaminated appears to be the largest unknown, but from disease surveillance, small waterborne outbreaks appear to be common. The identification of the ratio of identified to unidentified outbreaks might be a key piece of epidemiologic information that could help resolve the relative proportion of the population regularly being exposed to contaminated water. In terms of modeling the pathogen survival trajectories through aquatic environments, we need to know a) how many Campylobacter pathogens are entering aquatic environments; b) how long they survive in various aquatic environments; c) how effective sewage treatment and drinking-water treatment are at removing pathogens from contaminated water; and d) how often an infective dose makes it to an oral exposure?

Consequently, there are three primary modeling problems: a) survival during the period required to get into an aquatic environment, b) survival during the residence time in an aquatic environment, and c) the ecologic filters concerning sewage treatment, water treatment, and human behaviors in the use of water. All of the modeling issues associated with the survival of Campylobacter in water

![Table 4. Environment factors controlling survival of Campylobacter pathogens outside of the intestinal tract of host animals.](#)

| Factor | Environmental temperature | Campylobacter spp. | Comments | Reference |
|--------|--------------------------|-------------------|----------|-----------|
| Replication conditions | | | | |
| Minimum | 34–36°C | C. fetus spp. jejuni, 12 strains | — | (99) |
| | 31–32°C | C. jejuni, 2 strains | — | (21,43) |
| Optimum | 37–42°C | C. jejuni, 2 strains | — | (21) |
| Maximum | 45°C | C. jejuni, 2 strains | — | (21) |
| Survival conditions | | | | |
| Review papers | Various | Various | — | (42,107) |
| 30–65 days | 4°C | C. jejuni | Isolated from wastewater using various culturing techniques | (43) |
| 18–45 days | 12°C | C. jejuni | Half-shelled and unopened oysters; survival better at 3°C than at 10°C in half-shelled; survival better in bottled oysters at same temperature | |
| 4–7 days | 25°C | C. jejuni | | |
| 2–9 days | 20°C and 30°C | C. coli, C. jejuni | | |
| 8–14 days | 3°C and 10°C | | | |
| Viable for months | −20°C and −24°C | Frozen half-shelled | (108) |
| 18–28 days and 16 weeks to VNC | 4°C | C. jejuni, 4 strains | VNC recovered for two strains using mice<sup>b</sup> | (109) |
| > 4 months, filtered stream water in lab flask | 4°C | C. jejuni | Filtered stream water in lab flask; shaking and aeration increases survival; increasing temperature decreases recoverability | (110) |
| 202 hr (avg) | 4°C | C. jejuni, 17 strains | Water microcosms and biofilm studies show consistency in survival across C. jejuni strains | (111) |
| 176 hr (avg) | 10°C | | | |
| 43 hr (avg) | 22°C | | | |
| 22 hr (avg) | 37°C | | | |
| 7 months, laboratory | 4°C | C. jejuni | Identified viability through respiration | (105) |
| 12 hr to nonculturability | 37°C in darkness | C. lari, C. jejuni, C. coli | C. lari and UPTCs survived longer, survival in sea water | (98) |
| 5 days to nonculturability | 4°C in darkness | | Slightly better than in river water | (98) |
| 30–60 min to nonculturability | Not temperature dependent | Exposed to UVB in lab simulating sunny June day | (98) |

*UVB, ultraviolet B radiation.

<sup>a</sup>Spiral forms of thermophilic Campylobacter (i.e., C. jejuni, C. coli, C. lari, and C. upsaliensis) are gram-negative, oxidase-positive, S-shaped morphology. C. jejuni strains (21,43) have been isolated from wastewater using various culturing techniques. Half-shelled and unopened oysters; survival better at 3°C than at 10°C in half-shelled; survival better in bottled oysters at same temperature. VNC recovered for two strains using mice<sup>b</sup>.
appear to indicate that water temperature may be the primary determinant of pathogen longevity [Table 4 (21,42,43,98,99,105–111)].

Food preparation and consumption of certain meats, particularly chicken, are identified through observational studies as the greatest risk of campylobacteriosis, despite the number of ecologic filters between source and exposure (Figure 2). From a perspective of pathogen survival, food preparation is the final ecologic filter before human exposure. It appears that food derived from poultry, cattle, and sheep are regularly, if not perpetually, contaminated with Campylobacter (Table 2). Animal management and the subsequent slaughtering and food processing represent ecologic filters, but they do not appear to be entirely effective in removing Campylobacter from the environment (Table 3). However, these filters may be important both in reducing the pathogen load and in providing identifiable break points for future interventions.

In terms of modeling pathogen survival trajectories through food processing, preparation, and consumption exposures, we need to know a) how many Campylobacter pathogens are transferred onto foodstuffs destined for human consumption; b) how long they survive on food and food preparation surfaces; c) how many pathogens can be transferred between these intermediate environmental vectors (e.g., kitchen implements); d) how many are transferred between foodstuffs in the process of cross-contamination; e) how effective the food-related ecologic filters of food processing and food preparation are; and f) how often an infective dose makes it to an oral exposure.

These considerations reduce to the following modeling problems: a) survival on food surfaces, b) survival during the transfer between intermediate environmental vectors, and c) the ecologic filters of food processing, food preparation, and personal and kitchen hygiene.

Discussion

An examination of the literature dealing with the environmental constraints of pathogen survival does not clearly indicate how the Campylobacter pathogens are moving through the environment, but only ample evidence of its occurrence. The examination of animals, aquatic environments, and food-related surfaces indicates that there is an abundance of animal reservoirs (Table 2) and a variety of survival trajectories through the environment (Table 3) with rather common environmental survival parameters (Table 4). The ecologic model for campylobacteriosis we propose in this paper (Figure 2, Table 1) is not intended to predict how Campylobacter moves through the environment, but will provide a tool to study the dynamics of this system.

The proposed eco-environmental modeling will attempt to do three things. First, it will assist in bringing together the existing information on the survival of Campylobacter in the environment. In this paper, we have labeled the organizing principle “pathogen survival trajectories.” This Lagrangian approach seeks to define the relative survival potential for pathogens moving through the environment. Two key aspects define the movement of pathogens outside their animal hosts: a continuous decay in population numbers and movement of a proportion of the population into the VNC state, and passage through ecologic filters, which further reduces the pathogen population size. Although the first component is shaped by environmental factors such as temperature and nutrient availability, the ecologic filters are also defined by a number of factors, including behavioral factors such as personal hygiene or culinary practices.

Second, the proposed eco-environmental modeling provides an alternative perspective on the ecology of this disease. Three types of information shape our current understanding of human campylobacteriosis: human disease surveillance, epidemiologic case-control studies, and microbiologic investigations. Disease surveillance information has shown, for the last decade at least, a high incidence of campylobacteriosis within developed countries and large seasonal swings in the incidence of human disease. However, disease surveillance information is limited by the nature of the surveillance systems. It is generally accepted that the incidence is many times higher than we can observe and that there is no clear explanation for the seasonality. Case-control studies point to food, particularly poultry and undercooked poultry, raw water, and unpasteurized milk, to name a few, as sources of elevated risk. Case-control studies are not infallible, however. Case-control studies rely on recall ability for food and other activities and particularly on the ability of the control group to recall diarrhea events. This is problematic, particularly where controls may have had only minor symptoms or no symptoms. There have been hundreds of microbiologic studies of Campylobacter pathogens that have examined survival in a myriad of environments (Tables 2, 3, and 4) (e.g., pathogenesis, virulence, and strain typing). Despite the immense effort, there does not appear to be any overarching structure into which these new pieces of knowledge are being organized. It is difficult to see where this accumulation of knowledge is taking us. The model proposed here operates from a pathogen perspective that would be at home in the microbiologic discipline, but that also allows epidemiologic studies of human and animal disease incidence to be integrated into and evaluated against microbiologic knowledge.

Third, and perhaps most importantly, the proposed ecologic model for campylobacteriosis may provide a tool to help us better identify what we do not know and to evaluate how important these unknowns are likely to be in the context of the overall ecology. For example, our disease surveillance systems do not provide strong evidence of human-to-human spread. It is difficult to assess the effectiveness of human-to-human contact as an exposure route compared with the relatively more complex trajectories through water and food. A working mathematical model may allow us to replicate enough of the dynamics to assess whether our lack of knowledge is really important in this area. In short, it is a tool that can assist in the building and evaluation of knowledge.

Whether the proposed ecologic model provides a useful alternative perspective on the ecology of campylobacteriosis will be judged in part on the success of this perspective in fostering new insight. Consequently, the ultimate success of the proposed model, beyond whatever conceptual attractiveness the model may hold, will lie in its implementation. Modeling should work symbiotically with empiric research to help guide and evaluate the acquisition of new knowledge, as has been ably demonstrated with the effort of the climate research community to bring these two approaches together (112).

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