

# Ruminating on complexity: macroparasites of wildlife and livestock

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**Recent advances in ecology have improved our understanding of the role of parasites in the dynamics of wildlife populations. However, conditions that prevail in many wildlife systems, such as host movement, contact with livestock, and heterogeneity in the environment of the parasite outside of the host, have largely been ignored in existing models of macroparasite transmission. We need to refine these models if we are to stand a chance of developing effective parasite control strategies. New quantitative approaches enable us to address key complexities and make better use of scarce data, and these should enhance our efforts to understand and control emerging problems of interspecific parasite transmission.**

The role of parasites in the ecology of their hosts is attracting increasing attention [1]. Gastrointestinal nematodes regulate wild populations of red grouse and reindeer [2,3], whilst a wider range of parasites and pathogens are of practical importance to conservation efforts [1,4]. MACROPARASITES (see Glossary) are of particular interest because they often have broad host ranges, and transmission between wildlife, domestic animals and humans can occur, with consequences for conservation, livestock production, animal welfare and human health [5]. Predicting the risk, scale and even direction of such transmission presents a challenge. Contact between multiple host groups need not be direct, and macroparasite development outside the host depends on climatic factors and/or the presence of INTERMEDIATE HOSTS. Where these factors vary both spatially and temporally, environmental heterogeneity interacts with host movement to generate a shifting mosaic of transmission within and between host species.

Theoretical approaches, especially when used in tandem with field studies, have rapidly advanced our understanding of other complex problems of host–parasite population dynamics [1,6], and mathematical models of the parasite life cycle have long underpinned advances in the control of helminths in livestock and humans [7,8].

However, innovative refinements to existing model frameworks are required if we are to consider additional complexities that are common in nature, especially host movement and environmental heterogeneity. Here, we examine some shortfalls of existing macroparasite transmission models in this context, and indicate areas where recent theoretical and empirical progress and technical advances in computing enable key refinements to be made. Gastrointestinal nematodes of ruminants have been well studied and are used to illustrate many points. However, the approaches are applicable to a wide range of systems, and their use as part of a fuller consideration of parasite transmission in complex natural systems is long overdue.

## Multiple host populations

Existing models tend to consider only one DEFINITIVE HOST population. In fact, many macroparasites can infect more than one host species, and single-species models give misleading results where more than one sympatric host species or population is significant in parasite dynamics. Experiences with microparasites, such as the failure to eradicate bovine TB in areas in the UK with high densities of badgers [9] and invasions of endangered wildlife populations by diseases [10], demonstrate the potential importance of disease transmission between wildlife and domestic animals in both directions. Generalist macroparasites, meanwhile, pose a much greater threat to

## Glossary

**Definitive host:** species in which sexual reproduction of the parasite occurs.

**Direct life cycle:** the parasite is transmitted to another host by means of a stage that lives free in the environment.

**Indirect life cycle:** transmission necessarily involves development in species other than the main or definitive host.

**Intermediate host:** species in an indirect life cycle in which onward development or asexual reproduction of the parasite occurs.

**Macroparasite:** multicellular parasite, whose life cycle generally includes stages off the host (e.g. helminths and arthropods). With some exceptions, macroparasites cannot increase their populations without leaving the host: factors affecting transmission are therefore important to the level and outcome of infection in individual hosts, as well as to exchange of parasites between hosts [1].

**Microparasite:** smaller parasite that can reproduce without leaving the host, e.g. protozoa, bacteria and viruses.

vulnerable wildlife populations than do those with greater host specificity [11,12]. Ignoring the role of sympatric hosts in parasite dynamics compromises efforts to control parasites in multi-species systems.

For macroparasites, multiple sympatric host populations contribute to and draw from overlapping pools of infective stages (Figure 1), and can therefore be modeled by adding duplicate equations for parts of the life cycle in each area or group of hosts. Multiple group models have been used to estimate the relative contribution and exposure of different risk groups within a human population to *Schistosoma japonicum* in snails, and to guide control strategies accordingly [13]. Host age structure can be incorporated in a similar way [14]. Numerical simulations involving the parallel calculation of large numbers of duplicate equations pose few problems to modern computers, so the behaviour of complex systems of linked equations can be thoroughly investigated.

With a shared pool of infective stages, the comparative abundance and patterns of habitat use of wild and domestic animals underpin the magnitude of the flow of parasites between them, making a nonsense of early but persistent views that host populations with high typical parasite burdens are a probable source of infection for those populations with low burdens [15]. A lightly infected host population could be a significant source of infection if it is abundant or its movement patterns are appropriate (e.g. parasite persistence during temporary absence of the 'main' host might depend on infection of alternative hosts, even at a low level). This appears to be a factor in the persistence of the nematode *Haemonchus* spp. in nomadic livestock in Mauritania [16], and might also explain the reinfection of salmon with the monogenean *Gyrodactylus salaris* in Norwegian rivers [17]. Transmission between host groups depends additionally on the timing of contact between them, and not just on their total contribution to the pool of infective stages. A period of development is often required before parasites reach the infective stage [18], and the order and timing of host presence – and the

conditions for parasite development and survival in the intervening period – then become crucial to transmission.

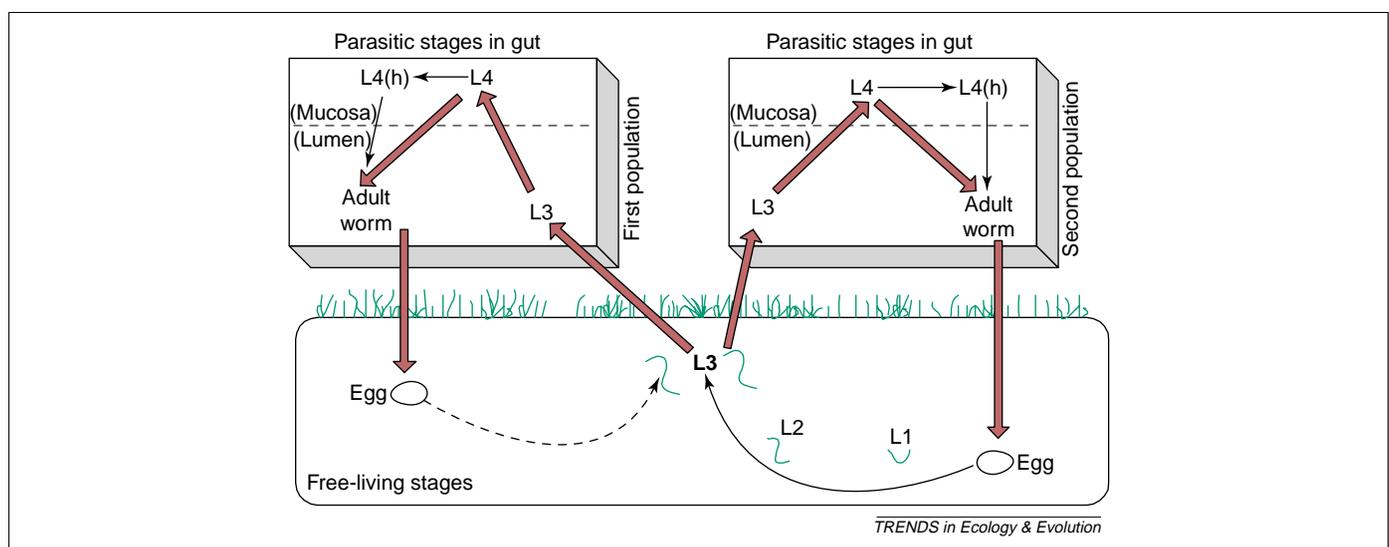
In many natural systems, the assumptions of single-host models are therefore unrealistic, and even multiple-host models that fail to consider differences in host abundance and habitat use are likely to ignore factors that are important to transmission.

### Host movement

Spatial models of microparasite transmission that are based on direct transmission between animals [19] are inherently inappropriate for macroparasites, whose life cycles usually involve development outside the main or definitive host. In DIRECT LIFE CYCLES, infective stages generally develop close to the point of deposition, and dissemination of infection relies on host movement [20]. Even if the life cycle is indirect, intermediate hosts often travel short distances relative to definitive hosts. The contrasting mechanisms of spread of microparasites and macroparasites are well illustrated by diseases of racoons in North America: whereas rabies undergoes long distance lateral spread through the host population [21], the nematode *Baylisascaris procyonis* can only move with the host, and transmission inevitably involves interaction with the external environment [22]. Gastrointestinal nematodes of grazing animals, which usually have direct life cycles, populate the vegetation layer, recruiting from and donating infective larvae to hosts that move over this layer. Parasite acquisition depends on hosts being present in an area at the time of infective larval availability, and host movement patterns are, therefore, a major determinant of transmission.

### Environmental heterogeneity and host dynamics

Even where host movement is relatively constrained, transmission models that assume a homogeneous environment can be misleading. For instance, predictions of the abundance of the liver fluke *Fasciola hepatica* in domestic ruminants based on the effects of climate on the free-living



**Figure 1.** Typical life cycle of gastrointestinal trichostrongylid (nematode) parasites of ruminants, illustrating the contribution of multiple host populations to a common pool of free-living infective stages. L1, 2, 3 and 4, first–fourth larval stages, respectively; L4(h), hypobiotic larvae in the gut mucosa. The common pool of L3 is specific to the geographical area, and can be duplicated to represent areas of contrasting climate, host density, and so on.

and intermediate stages are unreliable unless modified to take account of local conditions, such as topography [23,24]. For most parasites, conditions for infective stage development and survival vary in space and time. Eggs of trichostrongylid nematodes, for example, need warmth and moisture to develop to the infective third larval stage and migrate onto the herbage, and larvae survive best in cool (but not cold) conditions [18]. Infective stage availability is therefore strongly seasonal, whilst areas with a microclimate that is favourable to development or survival can become foci of early or prolonged infection [25]. Where intermediate hosts are involved in the parasite life cycle, seasonal and geographical variation in their abundance further affects infective stage availability [26].

Landscape epidemiology recognizes this by distinguishing between the infective risk of areas with differing underlying characteristics of, for example, vegetation or climate [26,27]. However, such static approaches ignore process uncertainty, such as the effect of climatic stochasticity on parasite development, as well as neglecting host distribution and habitat use. An area that is very suitable for parasite development poses no infective risk if hosts do not visit it at appropriate times to deposit or acquire parasites. Unpredictable variation in the weather, meanwhile, could mean that infective stages are present when hosts use certain habitat patches in some years, but not in others. This is likely to be most important in free-ranging wildlife whose life history includes well defined periods of movement or differential habitat use. Because this is common in nature, and movement of one or both host groups often forms the basis of contact between wildlife and livestock, climatic stochasticity is likely to play an important role in the transmission of parasites across host species boundaries. The mechanistic models of infective-stage development that prevail in the literature are ill placed to capture this in their present form, and must be extended to consider temporal and spatial heterogeneity in the external environment.

Interestingly, the movement of wildlife (and some livestock) itself often depends on climate, and seasonal host absence could drive fluctuations in parasite abundance [28]. In the long term, parasite transmission mechanisms might be expected to evolve to cope with long periods of host absence (e.g. monogeneans of desert toads [29]), and mobile hosts to avoid times and places of high infection risk (e.g. reindeer migrating away from 'hotspots' of warble fly density [30]). Some features of parasite life cycles, such as hypobiosis, equip them to survive periods that are unsuitable for transmission [31]. Variability in life-history traits within parasite populations, meanwhile, might itself be an adaptation to variable host availability [32]. Fluctuations in host nutrition and immunity that increase exposure or susceptibility [33,34] could coincide with additions to the pool of infection (e.g. through the periparturient rise in egg output [35]). Given host movement, such temporal heterogeneities are likely to generate spatial foci of infection.

Spatial variability in the external environment will therefore be important to parasite transmission only if it combines with host distribution to alter infection rate.

Changes in land use that affect host distribution and movement might then alter patterns of infection in wildlife (and transmission to livestock) even if they have no direct effects on parasite development and survival at a given location. The importance of landscape heterogeneity to microparasite transmission has been under increasing scrutiny [36–39], and the rarity of spatial models of macroparasite transmission is surprising given the additional relationships between free-living stages and the environment. There is scope in some systems to reduce modeled spatial variation to broad climatic patterns and mass host movements (Box 1). In others, the important spatial heterogeneities will differ (e.g. local contact patterns between hosts [21,40] or concurrent effects of global climate change on hosts and parasites [41]). Deciding the scale of spatial and temporal variation in host and environmental factors is an important dilemma when modeling disease transfer between wildlife and livestock.

#### Scales of variation and uncertainty

The usefulness of increasing complexity in transmission models is constrained by the resolution of data that are available to test predictions. Spatial models of *Echinococcus multilocularis* in foxes, for instance, have considered variation in the suitability of the environment for eggs and intermediate hosts at the resolution of the intermediate host home range (Box 2). Foxes in fact roam over much larger areas, and a high level of infection in a particular group of rodents is as likely to be due to the chance visit of a heavily infected fox as to favourable conditions for parasite development. Given overdispersion in adult parasite burdens in foxes [42], data collected at a spatial resolution equal to the size of individual fox ranges could also be confounded by individual variation in susceptibility. Prudence must therefore be exercised in the interpretation of results at such fine spatial resolution. A spatial model would only be useful if it predicted patterns of infection at a level at which enough hosts could be sampled to obtain a real measure of parasite abundance in the context of parasite aggregation. Parasites are aggregated in most wild animal populations [43] as a consequence of spatial variation in the environment (and consequently in parasites sampled by hosts as they move around [44]), as well as of genetic differences between individual hosts and the aggregated deposition of infective stages. Patterns of parasite aggregation in hosts and the environment are, therefore, interrelated [45] and could be fundamental to phenomena such as the rapid spread of anthelmintic resistance in livestock [20]. In practical terms, researchers should take into account host abundance and distribution, and levels of parasite aggregation, when deciding the level of spatial complexity to include in their models, and not just the resolution of environmental variation likely to be significant to developing parasites.

Uncertainty in parameter values might also dissuade us from incorporating too much complexity in transmission models. Our understanding of macroparasite population dynamics in livestock provides a good starting point for wildlife studies (e.g. in Soay sheep [46]). However, direct extrapolation of parameters from livestock is often

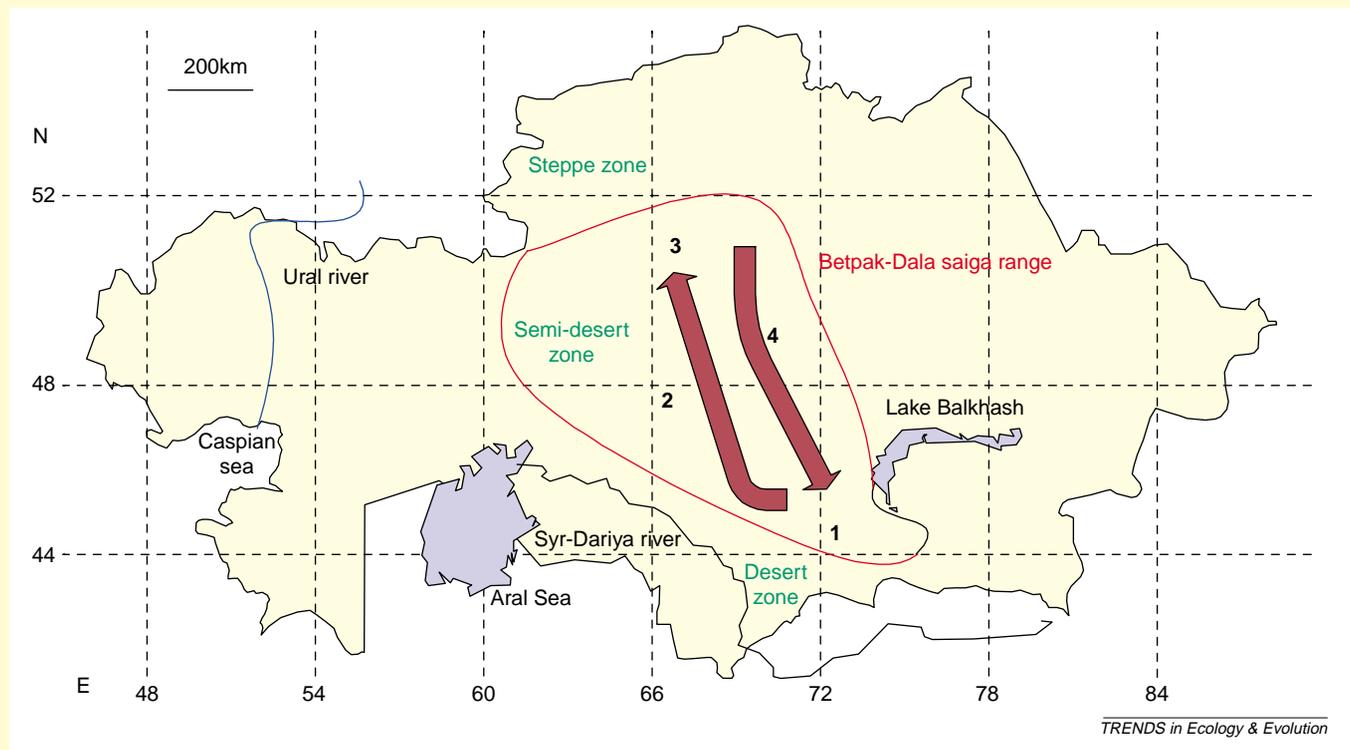
### Box 1. Movement and parasite transmission between Saiga antelopes and livestock

Saigas *Saiga tatarica* in Kazakhstan migrate *en masse* across climatic zones (Figure 1), and share their range and several species of parasite with domestic sheep, goats, cattle and camels. Some helminths are transmitted predominantly from saigas to livestock, and others from livestock to saigas, and the geographical direction of transmission also differs between parasites.

Observed patterns can be explained by relating host susceptibility and movement to parasite vital rates. Thus, gastrointestinal nematodes specific to saigas (e.g. *Nematodirus gazellae*) are characterized by tough free-living stages that survive adverse climatic conditions during prolonged host absence [18,52]. More fragile parasites make use of the multiple ruminant species that share the saiga range. Nematodes of the genus *Marshallagia* appear in sheep in northern Kazakhstan only where they graze within the saiga range, hence saigas are assumed to be the source of infection [53]. Very low burdens of *Marshallagia* in saigas less than a year of age suggest that saigas are themselves infected from livestock pasture in the south, and transfer these parasites to livestock in the north in summer [54]. The role of saiga migration in the spatial dissemination of infection is likely to be related to the longevity of adult

parasites. Tapeworms of the genera *Avitellina* and *Moniezia* are found in saigas after they have grazed livestock pasture in the north; whereas *Avitellina* is then carried south to shed eggs on livestock pasture, *Moniezia* live for only a few weeks, and die on the journey [55]. Strategies to reduce parasite transmission between saigas and livestock would be most effective if the times and places of high infection risk were known for each host group [56].

Combined empirical and theoretical study of this and similar systems, characterized by relatively clear host movement patterns and well demarcated seasons, might help us to understand how parasite persistence in different host groups and in the environment contributes to overall parasite population dynamics. Large-scale perturbations in host populations, such as the recent dramatic declines in saiga and livestock numbers in Kazakhstan [57,58], can also provide rare opportunities to investigate the importance of host abundance and density to parasite persistence. The subcutaneous botfly *Pallasiomyia antilopum*, for instance, has not been recorded in Kazakhstan since a similarly profound decline in saiga numbers in the 1920s, but persisted in the more stable Mongolian population [59].



**Figure 1.** Saiga antelope migration in Kazakhstan. Saigas spend winter in the south (1), calve on the northward migration (2) before reaching summer range (3), and return south in autumn (4).

inappropriate, for example where differences in susceptibility affect values for larval establishment or egg production, or nutritional constraints limit the effectiveness of immunity. Conducting the experiments necessary for parameter estimation in wildlife, however, might present insurmountable logistical and ethical difficulties, whilst existing field data are scarce and often imprecise. In addition, by adopting model structures developed for livestock, we are led by what we already know, and not necessarily by what is important in natural systems. Models of trichostrongylid transmission in domestic ruminants generally make detailed assumptions concerning development rates at different air temperatures [47].

These are largely drawn from laboratory experiments carried out in fixed conditions, and probably poorly represent the microclimate experienced by nematode larvae in the field [48]. Moreover, estimation of key epidemiological indicators, such as the basic reproduction number,  $R_0$ , is frustrated by temporal variation in vital rates and host density (Box 3), even though these variations might be key to the probability of parasite transfer to and persistence in a new host group.

This level of process and parameter uncertainty might discourage us from modeling parasite transmission in wildlife at all. However, by making broader assumptions concerning vital rates, and focusing on the role of seasonal

### Box 2. Spatial distribution and control of *Echinococcus multilocularis*

*Echinococcus multilocularis* is a tapeworm of foxes and has been the subject of intense research because of the zoonotic threat that it poses [60]. Eggs are infective immediately after being passed in fox (or dog) faeces and, when eaten by the intermediate host (usually a rodent), develop into cysts. The fox is infected by eating rodents with cysts (Figure 1). Humans are aberrant intermediate hosts and life-threatening alveolar echinococcosis (AE) results when humans ingest tapeworm eggs.

The infection risk for canids depends on the extent to which their range includes those of susceptible rodents. Thus, in Switzerland, the small ranges of urban foxes do not include peri-urban areas with high rodent intermediate host densities, and the prevalence of AE is consequently lower closer to the centre of conurbations [42]. Fox densities are highest in the urban areas owing to the anthropogenic food supply, but the prevalence of infection reaches a peak in the countryside and peri-urban areas because of greater availability of infected prey species. Consequently, it is hypothesized that the highest risk of environmental contamination with parasite eggs is from fox populations on the urban periphery. In more general terms, the prevalence in foxes appears to depend on habitat suitability for both intermediate hosts and survival of the tapeworm eggs (Figure 1), leading to a spatial distribution on a local scale.

Land use can affect rodent density and, thus, parasite prevalence. The variation in human AE prevalence between villages in China is strongly influenced by the proximity of landscapes that support *E. multilocularis* in wildlife [61]. Furthermore, in Europe, infected foxes are more likely to be found in areas with higher pasture and lower forest cover [62]. Studies at a high spatial resolution have suggested that only by assuming a heterogeneous distribution of infected foxes affected by landscape structures was it possible to explain the rapid recrudescence of infection after a control programme was abandoned [63]. Furthermore, observed variation in the prevalence of *Echinococcus multilocularis* in vole intermediate hosts has been explained by landscape-related variation in parasite egg viability [64]. However, parasite aggregation in the fox was not considered in the underlying reference scenario in the model development, but as an additional scenario whose relevance was rejected.

Control programmes based on treatment of foxes can reduce the threat to humans [65], and any real spatial aggregation of infected foxes would enable control efforts to be targeted on particular areas. Models that are genuinely able to pinpoint these areas would be extremely valuable.

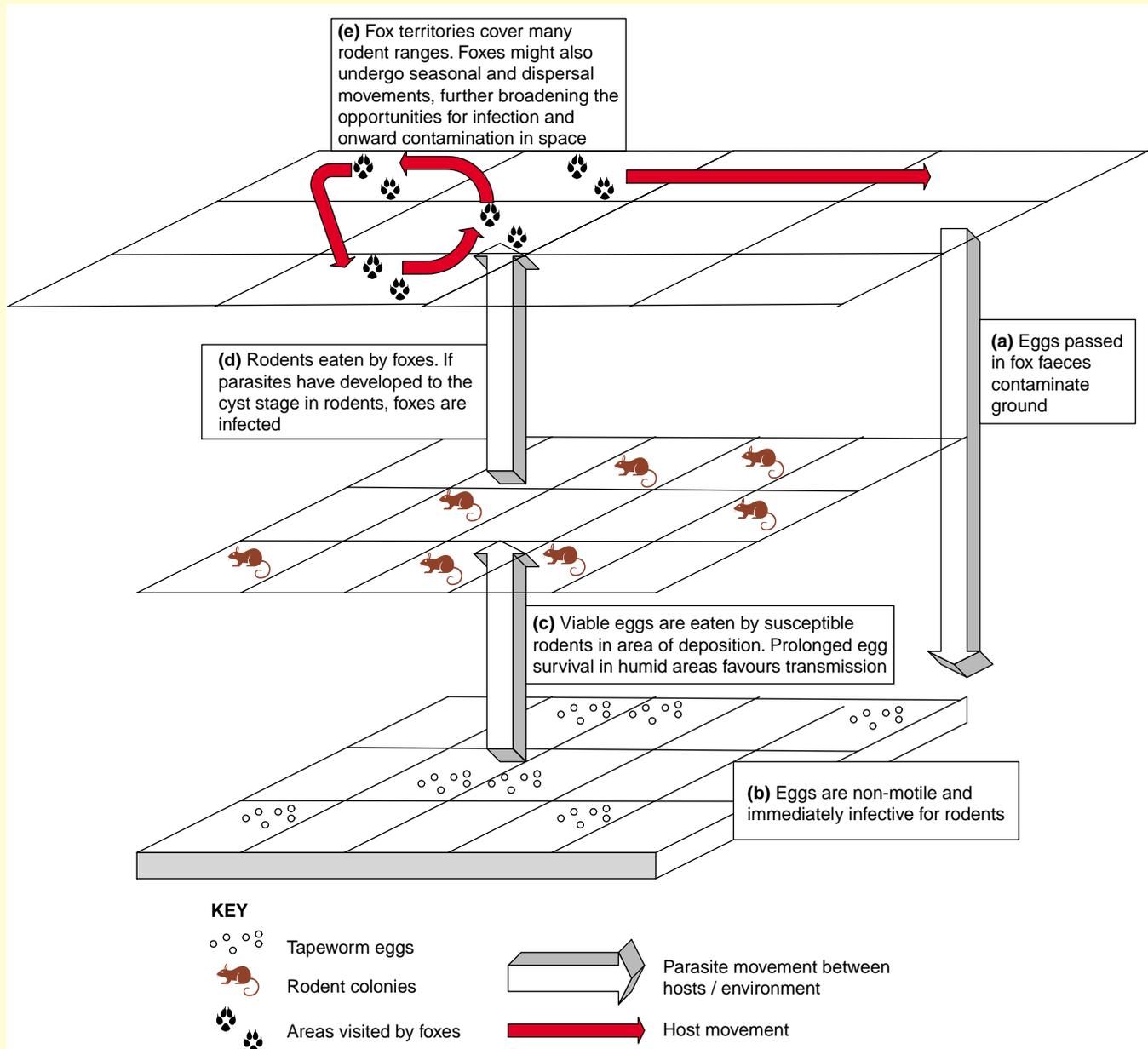


Figure 1. Spatial transmission of *Echinococcus multilocularis*.

### Box 3. Environmental heterogeneity and parasite persistence

The ability of a parasite to invade and persist in a host population is central to the dynamics of disease transmission across species boundaries. An extremely useful measure of this ability in both microparasites and macroparasites is the basic reproduction number,  $R_0$ , which must equal or exceed unity for a parasite to persist [66]. An equivalent quantity for macroparasites uses the average number of reproducing offspring produced by each in the present generation as a guide to the tendency of the population to increase or decrease in the long term. Roberts and Heesterbeek [67] formalized a definition for this quantity for the direct life cycle of nematodes of ruminants, which they called the basic reproduction quotient,  $Q_0$  (Eqn 1):

$$Q_0 = \frac{q\lambda(0)}{\mu(0)} \frac{\beta p(0)}{(\rho + \beta H)} H \quad (\text{Eqn 1})$$

with parameters listed in Table 1.

In its attempt to estimate the typical direction of population growth,  $Q_0$  assumes parameter values invariant in time. Although it can be refined to include periodic variation in parameter values [67], unpredictable variation (e.g. in climate and weather) is ignored (Table 1). Also, when hosts are absent,  $H(0)$ , and  $Q_0$  must therefore be zero, suggesting that the parasite population will decline to extinction. In a heterogeneous environment, vacant patches will have a  $Q_0$  of zero, even though developing larvae contribute to reinfection on the next host visit,

whereas brief occupation of patches might not enable transmission even if instantaneous parameter values give a high estimate for  $Q_0$ . In a host population moving between patches, then,  $Q_0$  cannot be estimated independently of host movement. Where more than one host population shares a pool of infective stages, an overall value for  $Q_0$  can only be estimated if they are present simultaneously [67]. Delays in patch occupation by different hosts can, in fact, provide time for larval development and enhance parasite transmission and persistence.

If temporal variations in parameters governing parasite transmission are known, we can simply simulate population dynamics in the absence of host immunity, and use overall predicted growth rates as a threshold quantity (growth rate  $\geq 0$  being equivalent to  $R_0 \geq 1$ ). By incorporating host movement explicitly in the model structure, and including climatic stochasticity as run-time variation in parasite vital rates, this simulation-based approach is a simple and potentially powerful way of considering the effect of host and environmental heterogeneities on  $R_0$ . Indeed, by ranking different heterogeneities according to their effect on growth rates, the important heterogeneities in a particular system can be predicted. Simulated instantaneous rates of change in parasite burdens in host (sub-) populations might also enable us to derive temporal variations in  $R_0$ , and hence identify the times and places most important to parasite population growth.

**Table 1. Parameters for the model of nematode transmission in a ruminant population and their reliance on variable factors in the external environment<sup>a</sup>**

Abbreviation	Parameter	Varies with
$q$	Probability that an egg becomes an infective larva	Temperature, humidity
$\lambda(0)$	Mean rate at which an adult worm produces eggs in the absence of acquired immunity	
$\beta$	Rate at which larvae are eaten by ruminants	Herbage intake, sward characteristics
$P(0)$	Probability that an ingested larva develops to become an adult in the absence of acquired immunity	
$\mu(0)$	Mean mortality rate of adult parasites in the absence of acquired immunity	Temperature, humidity
$\rho$	Rate at which the density of larvae on the pasture would decline in the absence of ruminants	
$H$	Number of host animals per unit area	

<sup>a</sup>Based on [67].

fluctuations in climate and host availability, we can still make useful predictions of patterns of larval availability in natural systems [47]. Stochastic approaches can then be employed to explore the effects of both parameter uncertainty and climatic variability, enabling parameter values to be explored across a distribution, alone or in combination [49,50]. New data can be incorporated by updating these distributions, rather than by changing the assumed 'correct' value. Our understanding of the system can be tested using competing alternative models [51]. In this way, we can incorporate uncertainty explicitly, rather than trying to build detailed deterministic models for which parameter estimates are poor or lacking. This rational reduction of uncertainty has been usefully applied to parasite transmission models using Bayesian techniques [13], and could be a cornerstone of our attempts to deal with parameter uncertainty in wildlife–parasite (and wildlife–livestock–parasite) systems.

Ultimately, models should aim to include biological variation that is important and ignore that which is not. It is unlikely that the relative importance of different sources of heterogeneity will be the same for all systems. By starting with a complex model and testing decreasing

levels of complexity against the available data, we can both identify the heterogeneities most important to parasite transmission in a particular system, and be ready to challenge these predictions as new data become available.

### Conclusions

In nature, multi-host, multi-parasite systems are the norm. Macroparasite transmission between populations relies on host movement, and the order and timing of habitat occupation can be crucial to parasite transmission, for example at the wildlife–livestock boundary. These complexities must be considered if we are to address problems of interspecific parasite transmission, and single-species, static host models are inadequate to this task. Parameter uncertainty and climatic stochasticity are significant challenges to complex models of unfamiliar systems, but should not deter us from building such models, provided that we aim to reduce uncertainty and complexity in a rational way. Greater complexity can then be incorporated without sacrificing our ability to explore model predictions thoroughly. Moreover, important parasite transmission between wild and domestic animals in both directions might occur at very specific times and

places, and recommendations concerning parasite control are then likely to be most useful to managers if they are explicit. Complex models that consider important temporal and spatial variation in host and parasite factors are in a better position to make such recommendations, and are an important and underused tool in the understanding and control of old and emerging problems of interspecific parasite transmission.

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