Thermal biology in insect-parasite interactions

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Recently, several applied studies exploring the use of pathogens for insect biocontrol have demonstrated significant effects of environmental temperature on the outcome of infection. For example, host resistance, host recovery, pathogen virulence and replication can alter considerably with sometimes very small changes in temperature. Moreover, the effectiveness of certain insect parasitoids and the activity of endosymbionts can vary across the range of realistic temperatures experienced in the field. These responses are not necessarily linear or immediately predictable, because they derive from a complex 'genotype-by-genotype-by-environment' interaction. Given the importance of parameters such as virulence and resistance in determining the course of a host–parasite interaction, such effects of temperature could have profound implications for host–parasite dynamics and coevolution.

That temperature affects biochemical, physiological and behavioural processes in animals is well established and there has been much work exploring the thermal sensitivity of organisms and factors influencing the evolution of thermal performance curves (Box 1). Ecological and evolutionary studies of interactions between hosts and their pathogens and parasites have a similar pedigree. What has been somewhat neglected is the integration of these two research areas to consider the effects that temperature can have on interactions between organisms [i.e. genotype-by-genotype-by-environment, or G × G × E, INTERACTIONS (see Glossary)]. To some extent, this might have arisen out of a tendency to abstract systems down to their basic biotic components to make research problems more tractable. However, there is an increasing number of examples, many from more applied disciplines, such as biological pest control, which point to environmental temperature having a crucial role in mediating the outcome of host–parasite interactions (for the sake of brevity, we refer simply to host–parasite interactions to include pathogens, macroparasites and parasitoids). Our aim here is to highlight some of these examples, examine the mechanisms involved and then explore some of the implications for our fundamental understanding of the ecology and evolution of host–parasite interactions. In drawing on the biocontrol literature and examples from other common 'model' systems, we emphasize studies of

insect or other invertebrate hosts. However, many of these issues apply to a range of ECTOTHERMS and their natural parasites.

Effects of temperature on host–pathogen/parasite interactions

Driven by the desire to develop alternatives to chemical insecticides, there have been many studies exploring the potential of pathogens and parasites for use in biological pest control [1]. A common starting point for most of this work is the investigation of dose responses and host mortality rates under constant laboratory conditions. The resulting measures of LD_{50} and LT_{50} are then used to select the most promising (virulent) agents for further testing in the field. This approach is also typical of many of the more

Glossary

Acridid: family of grasshoppers with short antennae. The acridids include the locusts that are a select group of grasshoppers able to pass into a swarming phase subject to the right environmental conditions.
Behavioural thermoregulation: the use of behaviour, such as avoiding or seeking sources of heat, to regulate body temperature.
Cellular and humoral defense mechanisms: refers to the two components of the insect immune system. The humoral response consists of soluble factors in the blood such as antimicrobial peptides and phenoloxidases. The cellular component involves the activity of specialized cells collectively called haemocytes. Haemocytes secrete antimicrobial peptides and are involved phagocytosis of small foreign objects and encapsulation of large objects.
Ectotherm: an animal that derives much of its body heat from external heat sources.
Encapsulation: an immune response in which large foreign bodies, such as eggs of parasitoids, are surrounded and suffocated by blood haemocytes.
Entomophthoralean (fungi): an order of entomopathogenic fungi that have complex life cycles involving a sexual stage and resting spores.
Frequency dependent selection: An evolutionary process where the fitness of a genotype is dependent on the relative frequency of other genotypes in the population. In a host–parasite context, we would expect initially rare resistant hosts to become more common under the pressure of parasite selection.
G × G × E interaction: The phenotype of an individual plant or animal is a consequence of the interaction between its genotype (G) and the environment (E) in which it exists i.e. a 'G × E' interaction. In considering the role of environment on a host and its parasite we extend the concept to include both interacting genotypes i.e. G × G × E.
LT_{50}: The median lethal time is the time required to produce death in 50% of the infected individuals exposed to a specific dosage of pathogens.
LD_{50}: The median lethal concentration or dose of a pathogen that produces death in 50% of the treated subjects. Usually a time period is stated, e.g. number of days post inoculation.
Mitosporic fungi: a group of fungi with a simple life cycle with no known sexual stage.
Reaction norm: describes the phenotypic variation among individuals of a single genotype when it is exposed to different environments.
**Thermoconformer:** an organism that does not attempt to regulate body temperatures but allows its body temperature to closely track environmental temperatures.

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Box 1. Thermal sensitivity profiles

The thermal sensitivity of ectotherm performance has been extensively studied and representation of ectotherm performance across temperature is traditionally summarized as a nonlinear asymmetric curve [70–72] (Fig. Ia). Optimal temperature (T_o) is defined as the temperature at which the trait measured (e.g. sprint speed in reptiles [73], or pathogen growth [39]) occurs at its maximal rate. CT_{min} and CT_{max} are the critical thermal minimum and maximum of thermally dependent performance (although not necessarily the lethal temperatures), respectively. Operative range is the difference between CT_{min} and CT_{max} across which some level of performance can be measured. Such curves have been used to explore hypothetical changes in trait performance owing to selection pressure from, for example, a warming environment [70,74].

In considering the interaction between a host and parasite, each organism or genotype will have its own thermal performance curve. These might superimpose directly on one another such that no relative effects of temperature would be expected. Alternatively, as exemplified by locust–fungal systems (and probably several other systems), host and parasite performance curves can show some form of separation (Fig. Ib) [39]. In this case, the course of infection could be strongly influenced by temperature and, particularly, by how temperature varies across the respective operative ranges of host and parasite. In Fig. Ib, high temperatures favour the host by both optimizing defence responses and directly limiting pathogen growth. As a result, there might be little pathogen-induced mortality [2,39]. At lower temperatures, pathogen growth might be optimized and host defence is less effective, with the result that the same pathogen appears highly virulent [2,39].
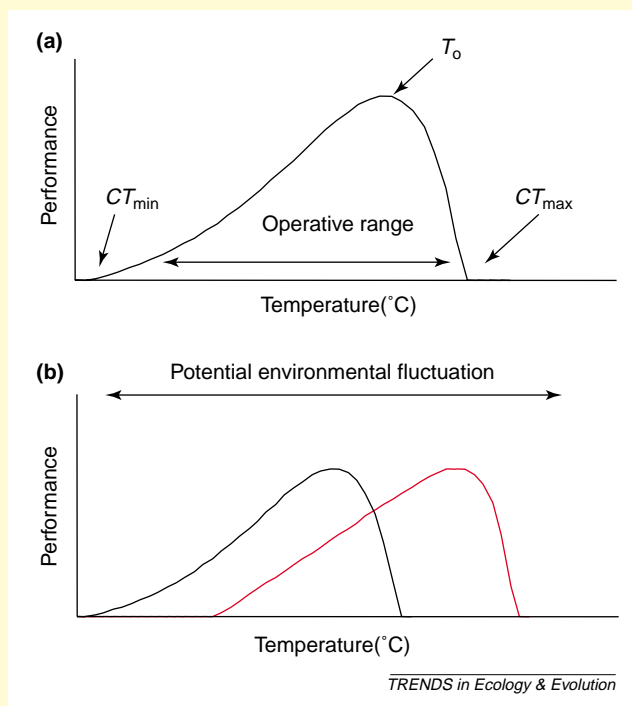
Fig. I. Variation in ectotherm performance (e.g. immune response, growth or feeding rate) across temperature. (a) A typical asymmetrical thermal performance profile indicating upper (CT_{max}) and lower critical temperatures (CT_{min}) and the temperature at which performance is maximum (T_o). (b) Performance curves of host and parasite can show some degree of separation. Here, host performance (red) is optimized at a higher temperature than that of the parasite (black).

fundamental studies exploring, for example, variation in resistance and virulence among natural populations. However, an additional common feature of this research is that, when the agents are taken to the field, performance

tends to be highly erratic [2,3]. Although there are many factors that can contribute to this variability, recent work on the use of fungal pathogens for biocontrol of locusts and grasshoppers has shown that, in particular, the ability of a pathogen to kill the host depends crucially on host body temperature and how this fluctuates with external environmental conditions. For example, both the speed of kill and overall mortality caused by MITOSPORIC FUNGI, such as *Beauveria bassiana* and *Metarhizium anisopliae* var. *acridum*, vary greatly with changes in environmental temperature [2–7]. Thus, these pathogens might appear either very virulent, causing extensive and rapid mortality of locusts or grasshoppers in 5–10 days, or virtually benign, with the same hosts surviving for weeks or even months.

In the context of biocontrol, it is clearly important for us to understand such temperature-dependent variability because biopesticide products based on these fungi will achieve excellent control under some conditions, whilst they might be largely ineffective and inappropriate for use under other conditions [8]. However, the effects also extend to natural host–pathogen interactions. For example, under certain environmental conditions, the ENTOMOPHTHORALEAN fungal pathogen *Entomophaga grylli* can act as the key mortality factor in the population dynamics of the variegated grasshopper *Zonocerus variegatus* [9]. However, during sunny periods, an increase in daytime maximum temperature of just 2°C enables infected hosts to recover from disease, creating an effectively immune population [10]. Similar results are revealed from studies of disease dynamics in rangeland grasshoppers in the USA [11].

That relatively small and realistic changes in temperature can dramatically alter apparent levels of susceptibility is not unusual or specific to ACRIDID–fungal systems. For example, strong thermal effects on virulence have been shown for *Entomophthora muscae* against flies [12], *Entomophaga maimaiga* against larvae of gypsy moth [13], *Erynia radicans* against potato leafhopper [14] and *Erynia neoaphidis* against the pea aphid [15,16], independent of factors acting on transmission and germination. Moreover, thermal effects on host–parasite interactions are not confined to fungal pathogens but include other microbial pathogens, such as rickettsia, microsporidia, bacteria and viruses [17–22], together with nematodes [23].

Temperature can have considerable effects on host susceptibility and/or parasite virulence with parasitoids. For example, there has been extensive research on *Drosophila*–parasitoid interactions, including studies of the selection of host resistance under high parasitoid pressure and concomitant tradeoffs [24]. But, temperature also plays an important role in determining the relative susceptibility of *Drosophila* larvae to their different parasitoids. For example, Fellowes *et al.* investigated cross-resistance against three parasitoid species in *Drosophila* lines that had been selected for increased defence [25]. Resistance was measured at 15°C, 20°C and 25°C in unselected and selected lines. Depending on the specific host–parasitoid combination, a range of interactions was possible, with resistance sometimes remaining

Box 2. Effects of temperature on the outcome of a host–parasite interaction

The influence of temperature on the outcome of a host–parasite interaction will depend on how temperature affects the ability of the parasite to colonize a host, versus the effect of temperature on the ability of the host to defend itself. If we define virulence as the net effect of this dynamic interaction (i.e. virulence is not necessarily a constant, nor simply an inherent property of the parasite alone) then this provides us with a metric to represent some of the varied effects of temperature highlighted in the main text (although it will not necessarily capture effects, such as changes in pathogen reproductive rate or expression of latent infections, which are also reported).

If both host and parasite are insensitive to temperature or share the same response over a given temperature range, the resulting virulence reaction norm will be flat (Fig. Ia). However, such insensitivity to temperature might be true only of very few systems, or be representative of a limited temperature range. More typical is some kind of positive or negative response, which can be linear (Fig. Ib) or more complex (Fig. Ic,d). Linear reaction norms appear to result where the range of temperature is still small relative to the operative ranges of host and/or parasite [25,75]. Exploring effects of temperature across a wider range that encompasses a greater proportion of the thermal performance profiles, and even exceeds the operative ranges, generally reveals more complex nonlinear responses. For example, with insect viruses (Fig. Ic), virulence is generally low at cool temperatures, increases with elevations in temperature and reaches an asymptote as temperature rises further [17,76]. Short-term exposures to yet higher temperatures, however, can enable host recovery effectively reducing virulence to zero on return to more permissive temperatures [18]; this is illustrated in (Fig. I) by the broken line, which represents a critical upper temperature at which virulence falls to zero. For other parasites, virulence might increase with an initial rise in temperature and then fall off as temperature increases further (Fig. Id). This pattern is reported for a range of systems described in the main text. However, the reverse pattern is also reported in which virulence is lowest at intermediate temperatures and then increases as temperatures either rise or fall (at least over a limited range) [25].
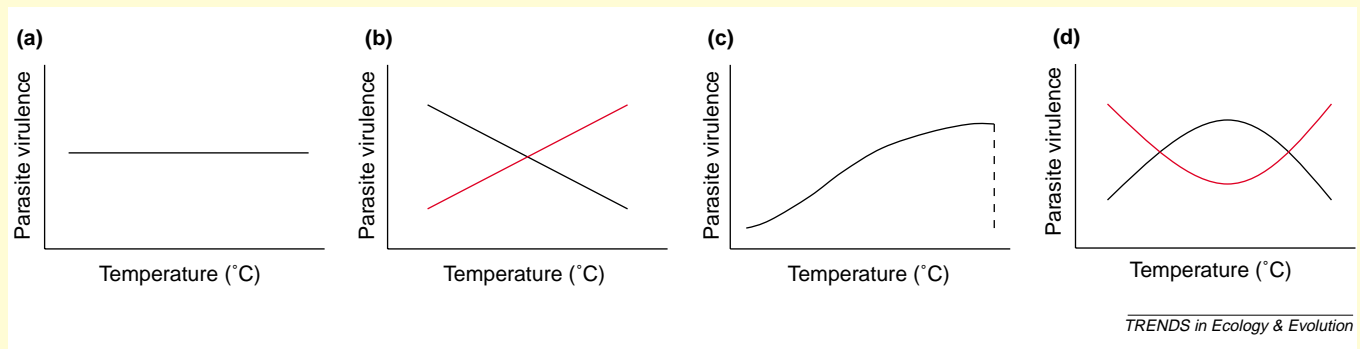

(a) Parasite virulence / Temperature (°C)
(b) Parasite virulence / Temperature (°C)
(c) Parasite virulence / Temperature (°C)
(d) Parasite virulence / Temperature (°C)

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Fig. I.

more or less constant irrespective of temperature, decreasing linearly with temperature, or following a complex pattern either increasing or decreasing at the extremes. Moreover, Fellowes *et al.* also showed that a change in thermal regime from 20°C to 25°C could confer an equivalent level of resistance in an unselected line to that exhibited by the selected line at the base-line temperature. Thermal effects on different aspects of host–parasitoid interactions have also been shown in other systems [26–29], with a fairly common observation that high temperatures can enhance survival of parasitized hosts (although whether this is due to increased ENCAPSULATION of the parasite larvae or pupae by the host, or greater direct mortality of the parasite owing to a higher thermal sensitivity, is often not determined).

There is therefore a range of possible influences of temperature on host–parasite interactions (Box 2) including effects on latent periods of infection [2], expression of latent disease [19], host recovery and parasite mortality [11,18,21,28], parasite replication [18] and parasite virulence and/or host resistance [2,6,23,25–27]. Importantly, and in contrast to the prevailing assumption that temperature acts as a simple scaling factor on rate processes [25], most studies reveal complex nonlinear effects in line with an interaction between host and pathogen thermal sensitivity profiles (Box 1). Evidence for a simple linear effect of temperature on aspects such as susceptibility or mortality rate is quite rare, especially when accounting for the full range of realistic environmental temperatures. Temperature fluctuations and temperature extremes can also result in markedly different effects from those predicted under constant temperatures [15,16,27].

Beyond hosts and their parasites

The examples we have discussed concern insect hosts and their parasites. However, it is also possible to identify effects of temperature on associations that are not strictly parasitic. For instance, endosymbiotic rickettsia and other bacteria have been isolated from a diversity of host organisms and have a range of effects on host biology [30–32]. Interestingly, although at least some of these endosymbionts are obligate partners, several studies illustrate a difference in thermal sensitivity between these microorganisms and their hosts. For example, relatively short exposure to high temperatures reduces the abundance and even eliminates endosymbionts, such as *Wolbachia* [33,34] and *Buchnera* [35], without direct effects on the host (although with obvious indirect effects resulting from loss of the endosymbiont). Moreover, interactions between symbionts are possible whereby one species can reduce the effects of environmental temperature on another [35]. Therefore, the differential effects of temperature on all the 'interacting genomes' should be considered if the ecology of natural insect populations is to be fully understood [35]. This extends also

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Box 3. Influence of thermal behaviour and environment on host body temperature

Most insects (and other ectotherms) adopt one of two thermal strategies: they either regulate body temperature behaviourally, or they conform to the external thermal environment and, within certain extremes, show little thermally mediated behaviour. BEHAVIOURAL THERMOREGULATION (see Box Glossary) enables an organism to achieve optimal body temperature for physiological functions, and is seen most often in insects inhabiting environments that are diurnally or seasonally unpredictable [77–79]. Body temperatures of behavioural thermoregu-lators tend to vary nonlinearly with ambient temperature [80]. Body temperatures of thermal conformers, however, are expected to track ambient temperatures closely [79]. In practice, thermoregulating insects can exhibit a linear relationship with ambient temperature if there is no source of radiant energy, or the cost of finding suitable thermoregulatory sites is high [7,81]. Conversely, THERMOCONFORMERS can exhibit marked nonlinearity in their ambient–body temperature relationship, tending to show an increasing thermal excess as ambient temperature increases [82]. Thus, on a hot day (red line, Fig. I), a behaviourally regulating insect might reach and maintain its preferred body temperature for several hours by balancing heat gain and heat avoidance [7,83] (black line, Fig. I, Day 1). Body temperature of a thermal conformer (blue line, Fig. I) might also reach high levels but for a shorter duration. Under slightly cooler conditions (Fig. I, Day 2), behavioural thermoregulation might again lead to large differences between body temperature and ambient temperature, although the preferred tem-perature might not be reached. Body temperature of a thermoconform-ing insect, however, will tend to be much closer to ambient. On a cool day with no radiant source of heat (Fig. I, Day 3), both thermoregulators and thermal conformers will tend to have body temperatures close to
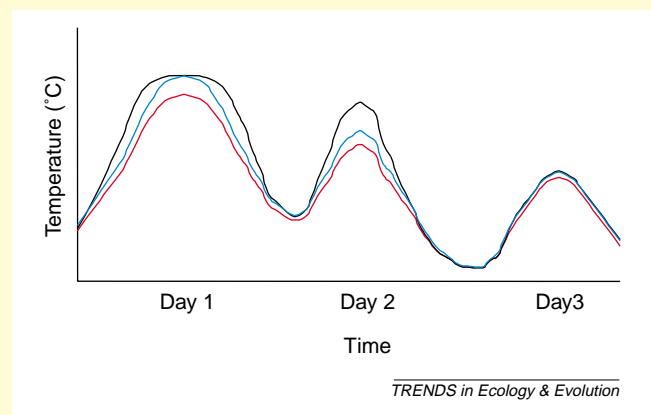

Fig. I.

ambient and will be little different to one another. In addition, irrespective of thermal behaviour in the day, body temperatures of ectotherms will equilibrate with ambient temperatures at night (which will, of course, vary). Moreover, body temperature and behaviour will be affected by the size (and so age and sex), thermal profile, distribution and colour of the insect. Thus, basic measures of ambient temperature, or simplifying assumptions that consider just the mean temperature, will tend not to be representative of the thermal environment in which the host–parasite interaction is actually played out.

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to our understanding of the ecology of vector-borne diseases, for which differential effects of temperature on the parasite and disease vector (together with possible further interactions with endosymbionts [32]) have been identified [36–38].

Mechanisms: parasite growth and host defence

The effects of temperature on a host–parasite interaction depend on the thermal sensitivity profiles and the environmental variability (Box 1). When both host and parasite share the same thermal optima and are adapted to perform similarly across a temperature range, the effects of temperature can be simple. However, when host and parasite have more discrete thermal performance profiles and temperatures regularly fluctuate across the range of these REACTION NORMS, the nonlinearities in the respective responses of host and parasite can lead to large G × G × E effects [15,39].

In many cases, the mechanisms whereby temperature acts on the host–parasite interaction remain poorly understood. In the first instance, the influence of environ-mental temperature on host body temperature is deter-mined by host thermal behaviour (Box 3). Most locust and grasshoppers, for example, are good thermoregulators and have a suite of behaviours for maintaining a preferred body temperature, which is relatively independent of ambient diurnal temperatures [40]. Given suitable environmental conditions, this thermoregulatory beha-viour enables locusts to raise their body temperatures to a preferred set point around 38°C–40°C [2,5,11,40,41]. Given that the optimum temperature for growth of fungi such as *B. bassiana* and *M. anisopliae* is in the range of 25°C–30°C, such body temperatures will have a direct

effect on pathogen growth, contributing to the substantial delay in the rate of fungus-induced mortality referred to previously [2,5–7,39].

An additional dimension to thermoregulation in locusts and grasshoppers is that certain species exhibit fever responses following infection, whereby thermoregulatory behaviour is modified to enable hosts to attain a new preferred body temperature some 2°C–5°C higher than normal [4,39,41]. Until very recently, the significance of this 'behavioural fever' response was unclear, because the growth of many pathogens at normal host preferred temperatures of 38°C–40°C is already negligible or zero. However, Elliot *et al.* [39] have now demonstrated that behavioural fever does provide additional survival benefits above and beyond normal thermoregulatory behaviour. That said, it still remains unclear to what extent the survival benefit of fever in Orthoptera and other arthro-pods results from a direct negative effect of temperature on the pathogen, or from indirect effects via changes in host immune response, or from a combination of both. There is some evidence of a differential fever response depending on the thermal sensitivity of the pathogen, with fever only detected against a pathogen that is negatively affected by higher host temperatures [2,20]. Similarly, fever in honeybee colonies has been suggested as a defense response to *Ascosphaera apis* (the fungus which causes the disease 'chalk brood'), again through a direct thermal effect on a heat-sensitive pathogen [42]. A recent study of migratory locusts, however, indicated that fever tempera-tures could play a role in maintaining haemocyte population levels and enhancing host immunity by promoting phagocytosis [43]. Studies of other arthropod taxa report a range of fever responses with temperatures

elevated by up to 20°C and suggest both direct and indirect effects of increased temperature on pathogenesis [44,45]. Moreover, the possibility also exists that parasites can manipulate behaviour (including thermal behaviour) of their hosts to enhance their own virulence and/or fitness [36,46,47]. Interestingly, the mechanisms and adaptive significance of fever responses in mammals is also of some debate [46]. Increasingly, analogies and homologies identified between the immune system of insects and innate immunity of higher animals suggest common mechanisms and common effects of temperature and fever across taxa [48–51].

Thermoconforming organisms do not regulate body temperature to the same extent as behavioural regulators at least within the bounds of upper and lower lethal temperatures (Box 3). This might be because the stability of the thermal environment they inhabit [7,10], and/or the size of the insect, preclude tight control of body temperature [26]. As such, body temperature will tend to track ambient temperature more directly and the capacity to utilize temperature actively in defence is minimal (as is the potential for the parasite to manipulate host body temperature for its own fitness). However, lack of thermoregulation does not equate to lack of thermal sensitivity. Thus, environmentally driven variation in body temperature will still influence key elements of the innate immune system, such as CELLULAR AND HUMORAL DEFENSE MECHANISMS [52]. For example, encapsulation of eggs of the parasitoid *Metaphycus stanleyi* by the pyriform scale *Protopulvinaria pyriformis* is correlated with ambient temperature creating marked seasonal variation in the effectiveness of the parasitoid [26]. In addition, changes in host body temperature can also impact directly on the parasite. With the entomoptherealean fungi, for instance, exposure to high temperatures can cause cell lysis enabling hosts to recover from infection [11,15]. For other pathogens, such as nucleopolyhedroviruses, effects can be more complex, with individual traits (e.g. incubation time and yield) showing different thermal sensitivities within and between hosts [17,22,53].

In summary, the body temperature of ectotherms is dependent on environmental temperature, with the exact relationship determined by several ecological factors, particularly thermal behaviour (Box 3). In turn, host body temperature determines performance of the parasite, either directly, or through effects on host defence mechanisms. In addition, in many systems, biotic factors, such as the host density and extent of melanization, also influence overall resistance levels [54,55]. The interaction between such biotic and abiotic influences has been largely neglected [3] and is only now receiving attention [56,57].

Implications for population dynamics and the evolution of host–parasite interactions

Although there are very few published studies that examine the consequences of temperature-induced effects on host–parasite population dynamics, measures of host susceptibility, host recovery, latent period of infection, pathogen-induced mortality rate (virulence) and pathogen replication are included in some form in most host–

parasite models. As previously discussed, temperature can impact on all of these parameters, either singly or in combination. It follows then that temperature must impact on host–parasite dynamics, either quantitatively or qualitatively, a conclusion that is supported when temperature is modeled explicitly [13]. Of particular relevance is that variation in environmental temperature can be considerable across even very small temporal and spatial scales. In addition, host body temperatures can also vary with biotic factors, such as behaviour, density and body colour [58,59]. Such variation might severely limit the generality of model predictions based on parameters estimated under just one set of conditions.

In an evolutionary context, numerous studies have identified genetic variation in defence and virulence in host–parasite associations [60–63]. However, there has been difficulty in demonstrating that laboratory-observed genetic variation can lead to FREQUENCY-DEPENDENT SELECTION in the field [62,64]. Given that environmental temperature has the potential to alter virulence and resistance in such systems (demonstrated for the *Drosophila* and pea aphid systems, which show marked nonlinearities in responses to temperature [15,16,25]), this suggests that variability in environmental temperature could play a role in mediating the pattern of frequency-dependent selection. In other words, with temperature altering virulence and resistance and causing the selection pressure to shift over time and/or space, the net effect of frequency-dependent selection at the population level might be very different from that predicted under one set of conditions in the laboratory [15,16]. Although this remains to be tested, it is interesting to note that, under controlled laboratory conditions, genetic variation has been shown to provide the basis for selection of resistant host genotypes [24]. More generally, coevolutionary dynamics have most clearly been demonstrated in endothermic host–parasite associations [65,66]. Both these situations represent environments in which extrinsic variability is removed.

Conclusions and recommendations

Our key message here is that, in studying host–parasite interactions, environment matters. More specifically, the course of an interaction is determined by host body temperature, which, depending on thermal behaviour, might not be close to ambient. Where some distinction exists between thermal sensitivity profiles of host and parasite, environmental temperature, via its influence on host body temperature, might have complex effects that are not necessarily immediately predictable.

Clearly, understanding such complex interactions poses considerable experimental challenges, even more so when one considers that temperature is unlikely to be the only environmental determinant [67,68]. However, there are several basic approaches that could provide relatively rapid insight into the possible importance of environmental temperature on host–parasite interactions. First, in considering whether the influence of temperature is likely to lead to any complex effects, a useful starting point is to determine the temperature-dependent performance curves for each interacting species (or genotype). For many

host−parasite systems, such information probably exists, so valuable inferences can already be made. Second, the potential importance of any interactions can be revealed by conducting experiments under a range of temperatures, exploring temperature variation and realistic upper and lower extremes, as well as constant temperatures. Finally, a recent study exploring the effects of assay conditions in life-history experiments with *Drosophila* has revealed that conclusions about responses to selection depend upon the environment in which the assay was performed [68]. Studies of other model communities indicate that costs of resistance (i.e. tradeoffs with other traits, such as competitive ability) might also vary depending on the environment [69]. We suggest, therefore, that in studying coevolution of hosts and parasites and other correlated responses, it might be more valuable to derive measures of life-history traits from the response of the genotype across the relevant environmental range (i.e. the area under the reaction norm), rather than use a snapshot measure estimated under just one set of conditions.

Acknowledgements

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