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Functional Ecology

PERSPECTIVE

Non-lethal effects of climate change and infectious disease: An energetics approach to understanding population impacts

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Abstract

1. Climate change and infectious disease jointly impact species worldwide. In addition to causing conspicuous mortality events, these threats produce a range of non-lethal effects that are often overlooked, yet can affect individual survival and fecundity, and ultimately, population and species viability.
2. We develop an energetic framework that structures the study of non-lethal effects of climate change and infectious disease and their downstream demographic consequences. The framework identifies pathways by which climate change and infectious disease affect the acquisition, storage and mobilisation of energy required for organismal survival and reproduction.
3. The joint energetic effects of climate change and infectious disease, while often non-lethal, can reduce fitness by increasing energetic demands, exacerbating energetic trade-offs and accelerating physiological ageing.
4. Considering the energetic mechanisms underlying non-lethal effects can explain when and why recurrent and/or chronic events associated with climate change and infectious disease can be important limiting forces for populations and species.

KEYWORDS
allostasis, ecology, global change, infectious disease, non-lethal effects, population demography

1 | INTRODUCTION

Among the many ecological concerns arising from global change, impacts from climate and infectious disease are at the forefront (Drake et al., 2023; Lafferty, 2009; Rohr et al., 2011). Populations of virtually all species face the simultaneous threats of a changing climate concurrent with rising threats from infectious disease, calling attention to the joint impacts the two stressors have on species' populations. While attention has centred on catastrophic, compound disturbances of climate change and infectious disease (Cohen et al., 2019; Drake et al., 2023), many events associated with climate change and infectious disease do not kill organisms quickly or conspicuously (Conradie et al., 2019; Daversa, Hechinger, et al., 2021). Rather, they elicit behavioural and physiological

changes that have downstream consequences for survival and fecundity over longer timespans, falling under the general category of non-lethal effects (Conradie et al., 2019; Daversa, Hechinger, et al., 2021). Such events include (but are not limited to): more frequent days of extreme high temperatures (Paniw et al., 2022; Sinervo et al., 2010), more frequent and longer-lasting droughts (Chiang et al., 2021; Williams et al., 2022), habitat loss and alteration following wildfires (Driscoll et al., 2021; Sanderfoot et al., 2021), invasions by novel infectious agents (e.g. viruses, fungi, bacteria, worms) enabled by shifting climate (Drake et al., 2023; Mora et al., 2022), and shifting distributions and intensifying transmission of endemic pathogens (Eby et al., 2023; Mora et al., 2022). We submit that the demography of populations subjected to the non-catastrophic consequences of climate change and infectious disease is likely shaped in large part by the non-lethal effects of these stressors (Conradie et al., 2019; Daversa, Manica, et al., 2021; Li et al., 2013). While intuitively appealing, tests of this hypothesis are largely lacking, in part because there are no formal frameworks to structure investigations of joint non-lethal effects of both stressors. This research gap exposes substantial uncertainty surrounding the long-term viability of wild populations living with climate change and infectious disease.

At a basic physiological level, the non-lethal costs of many stressors, including climate change and infectious disease, are governed by energetic mechanisms. Unfavourable conditions presented by stressors raise the energetic costs of completing 'normal' activities essential to survival and fecundity (Bobba-Alves et al., 2022; Galic et al., 2018; Wingfield, 2005). They also elicit physiological and behavioural stress responses that deplete energy reserves (Buckley, 2008; Tomlinson et al., 2014; Wingfield et al., 2017) and lead to physiological malfunction (Bobba-Alves et al., 2022; McEwen, 1998; Smith et al., 2009; Wingfield, 2005). Because energetics is central to the biology of all organisms, tracking energetic changes should offer generalisable, mechanistically grounded metrics to quantify the role of non-lethal effects in limiting populations confronted with climate change and infectious disease. Although there are existing energetic models that predict population responses to climate change (Buckley, 2008; Buckley et al., 2014; Tomlinson et al., 2014), infectious disease has not been incorporated into these models.

We present an energy-based framework for examining demographic change that arises through joint non-lethal effects of climate change and infectious disease. We focus on scenarios where climate and infectious disease jointly create physiologically challenging, but not immediately lethal, conditions. Such scenarios threaten homeostasis—the maintenance of evolved physiological setpoints essential to survival—without abrupt death, and thus allow organisms to respond and adjust to the threats. We develop this approach in four steps. First, we outline the broad links between climate, infectious disease and demography, and we point out relevant organism–environment interactions underlying these linkages. Second, we present a dynamic energy budget (DEB) framework to identify non-lethal effects of climate change and infectious disease on organism-level processes associated with fitness. Third, we consider interactions between infectious disease and energy budgets that may indirectly affect demography by altering pathogen transmission and other epidemiological processes. Fourth, we draw from allostasis theory to establish testable hypotheses linking non-lethal effects on organism energy budgets to population decline. Fifth, we consider how the development of tolerance may attenuate non-lethal effects of climate change and infectious disease. We finish by offering methodological guidelines for applying our framework.

2 | THE DEMOGRAPHIC EFFECTS OF CLIMATE CHANGE AND INFECTIOUS DISEASE

At the coarsest level of detail, one can model climate, disease and their interactions (P1 in Figure 1) as directly altering survival and fecundity (grey arrows P2 and P3 in Figure 1). For catastrophic events that cause rapid mortality, this phenomenological approach may be sufficient. However, for the many events that do not directly cause rapid mortality, explicit consideration of influences on organismal biology is needed to consider slower, non-lethal mechanisms of demographic change. Climate and infectious disease influence organisms

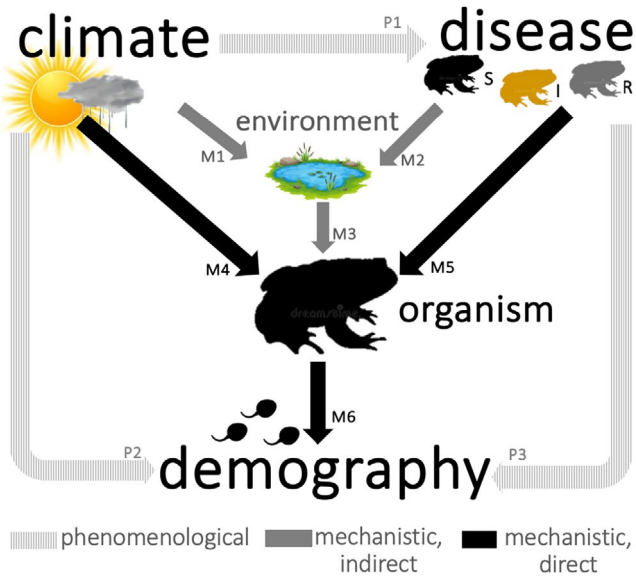

FIGURE 1 General framework for evaluating non-lethal effects of climate change and infectious disease. Demographic impacts of climate and infectious disease are often approached phenomenologically (light grey arrows, P1–P3), with climate having direct effects on infectious disease and demography, and with infectious disease also having direct effects on demography. Underlying these non-mechanistic linkages are effects on organismal biology, both directly (black arrows, M4–M6) and indirectly via environmental change (dark grey arrows, M1–M3). A mechanistic approach that incorporates environment and organismal biology accounts for non-lethal effects of climate change and infectious disease.

non-lethally by affecting behaviour and physiology (arrows M4 and M5 in Figure 1) and by changing resources in the non-climatic environment (arrows M1, M2, and M3 in Figure 1). Having incorporated these direct and indirect influences of climate and disease on organisms, we can consider non-lethal effects leading to demographic change (arrow M6 in Figure 1) and their underlying energetic mechanisms.

3 | ENERGETIC MECHANISMS OF NON-LETHAL EFFECTS

Bioenergetic models, including the DEB (Kooijman, 2009; Martin et al., 2013), provide a scaffold for specifying the energetic mechanisms driving non-lethal effects of climate change and infectious disease at the organismal level. DEB models describe how organisms convert energy from the environment into fitness (Figure 2). DEB models portray a sequence of energy fluxes that starts with an environmental (i.e. external) energy supply that is accessible to organisms. Through the process of assimilation, external energy sources are stored in the tissues of organisms and are transformed into an internal energy supply. In animals, assimilation includes feeding and digestion, and internal energy supplies are mobilised to meet the energy demands of fitness. The standard DEB model distinguishes four types of energetic demands (Kooijman, 2009; Martin et al., 2013): somatic maintenance, growth, maturation and reproduction. DEBs summarise resource supply and demand that drives organismal function (along with other physiological systems and resources, Cohen et al., 2012), with energy as the underlying currency. Demographic parameters, like survival and fecundity, depend on the capacity of organisms to assimilate and mobilise sufficient energy to meet the demands of maintenance, growth, maturation and reproduction.

Identifying how climate and infectious disease influence DEBs creates an energy-based accounting system for evaluating the direction and magnitude of non-lethal effects. The models are flexible; energetic demands can be tailored to match specific life history and/or life stage characteristics (e.g. juvenile stages do not mobilise energy towards reproduction) (Kooijman, 2009; Lika & Nisbet, 2000; Nisbet et al., 2010). DEBs can also be tailored to distinguish specific behaviours and physiological functions associated with maintenance, growth, maturation and reproduction for more targeted

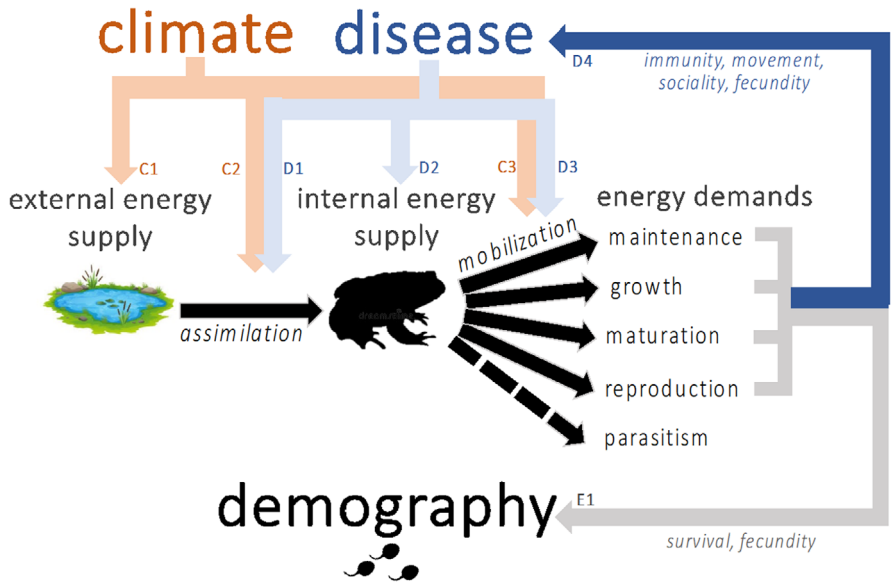

FIGURE 2 Non-lethal effects of climate change and infectious disease on energy budgets. Conceptualising an organism's biology as a dynamic energy budget highlights energetic mechanisms that are influenced by climate change and infectious disease. Energy from environmental resources (external energy supply) flows into organisms though assimilation (Kooijman, 2009), which includes feeding and digestion in animals and photosynthesis in plants. Within organisms, energy is held in reserves (internal energy supply) and mobilised to meet energetic demands for somatic maintenance, growth, maturation and reproduction (Kooijman, 2009). In the case of infection, infectious agents detract a proportion of internal energy reserves by parasitising hosts to meet their own energetic demands (Hall et al., 2009). Climate change influences external energy supply (C1) by altering resource quality and quantity. Both climate change and infectious disease influence assimilation (C2, D1) and mobilisation (C3, D3) by eliciting behavioural and physiological stress responses (McEwen, 1998; Sapolsky et al., 1986) and causing physiological wear-and-tear (McEwen, 1998; McEwen & Wingfield, 2003). Infectious disease directly influences internal energy supply (D2) because the causal infectious agents (e.g. viruses, fungi, bacteria and worms) consume host resources for energy (Hall et al., 2009). Survival and fecundity, which link to population demography (E1), are a function of an organism's energetic demands, energy mobilisation and physiological wear-and-tear over the lifespan. Reciprocal influences of energy budgets on infectious disease dynamics (D4) arise through energy mobilisation towards structures involving spatial movement (Daversa et al., 2017; Paniw et al., 2022), social interactions (Ezenwa et al., 2016), immune defence (Hing et al., 2016; Tate & Graham, 2015) and offspring production, all of which affect disease parameters (contact, pathogen transmission, recovery, etc.).

examinations of non-lethal effects. For example, measuring energy mobilisation for immune defence, which is part of maintenance in DEB models, could afford more precise characterisation of the energetic influences of infectious disease (Tate & Graham, 2015). Additional constraints, such as specific nutrient availability, can also be incorporated into the framework to consider non-energetic influences on organism function.

Measuring changes to energy supply and demand enables informed predictions of the consequences of joint exposure to climate change and infectious disease, including their additive, synergistic and/or antagonistic effects on organisms, which is a dominant aim of 'multiple stressor' research (Tekin et al., 2020). As a proof of concept, Galic et al. (2018) simulated joint non-lethal effects of hypothetical stressors on the energy budgets of amphipods. The simulations showed that joint exposure to two stressors led to reductions in amphipod body size and reproductive output, yet the magnitude of the reductions was less than predicted assuming additive effects of the two stressors (Galic et al., 2018). Furthermore, the impacts of joint stressors on organisms depended on the specific energetic process affected; stressors that reduced energy assimilation had negative effects on both body size and reproductive output, potentially reducing both survival and fecundity, whereas stressors that compromised reproduction only affected fecundity (Galic et al., 2018). These results underscore two key points that are incorporated in the proposed framework. First, multiple stressors, including climate change and infectious disease, should be considered both individually and jointly when evaluating their non-lethal effects. Second, predicting the non-lethal consequences of stressors is improved by examining specific energetic processes associated with energy budgets.

4 | INTERACTIONS BETWEEN INFECTIOUS DISEASE DYNAMICS AND DEBs

Contracting an infectious disease has several well-documented effects on energy budgets. First, perceived or actual contact with parasites or other disease-causing agents prior to infection elicits avoidance behaviours that affect energy mobilisation and impose energetic trade-offs with feeding, growth, maturation and reproduction (Daversa, Hechinger, et al., 2021; Giorgi et al., 2001; Luong et al., 2017; Scantlebury et al., 2007). Second, infectious agents impose a direct energetic cost by parasitising host energy to increase their own fitness (Figure 2, dashed mobilisation arrow), which reduces host internal energy supply and can cause tissue damage that mobilises energy towards repair (Cressler et al., 2014b; Hall et al., 2009; Scantlebury et al., 2007). Third, infections elicit immunological defences that are energetically costly (Hart & Hart, 2018; Hawley et al., 2012; Tate & Graham, 2015). There can also be additional, system-specific energetic effects, such as disruption to thermoregulation in wolves driven by mange infection (Cross et al., 2016).

Energy budgets, in turn, influence infectious disease parameters, such as host contact rates, pathogen transmission, recovery and host recruitment (Hall et al., 2009), creating a feedback loop (Figure 2, blue arrow). One such influence occurs via energy mobilisation for movement and social interactions, which for free-living organisms are important components of maintenance, growth, maturation and reproduction. Movement and sociality are critical factors in disease dynamics because they affect host contact and interaction patterns, which determine the efficiency of transmission for many pathogens (Daversa et al., 2017; Eby et al., 2023; Ezenwa et al., 2016). Energy mobilisation for immune defences, which is classically considered part of maintenance, has strong impacts on disease dynamics through influences on pathogen transmission rates and probabilities of host recovery from disease (Cressler et al., 2014a; Hing et al., 2016). Pathogen exposure typically increases energy mobilisation for immunity, and this can reduce transmission and speed recovery. However, some pathogens can co-opt immune activity to promote transmission (Lazzaro & Tate, 2022), creating positive relationships between transmission and energy mobilisation towards immunity. Acknowledging that immunity and the outcomes of pathogen exposure are shaped by a range of physiological factors (Cohen et al., 2012; Downs et al., 2014), the specific impacts of energy budget changes to epidemiological processes provide a route of demographic change under the combined stress of climate change and infectious disease. Susceptible-Infected-Recovered models (Keeling & Rohani, 2008) can be modified to complement the proposed framework and capture feedback between energy budgets and disease parameters.

Kalahari meerkats (*Suricata suricatta*) offer a case study for the demographic importance of feedbacks between energy budgets and infectious disease dynamics. Rising maximum air temperatures in the Kalahari increased energetic demands for thermoregulation in meerkats, which led to reduced survival due to energetic stress (Paniw et al., 2022). In addition, warming temperatures elicited stress responses in the form of dispersal (requiring energy mobilisation for movement), which increased transmission of the tuberculosis-causing pathogen *Mycobacterium suricattae*, leading to higher overall prevalence of tuberculosis and disease-driven reductions in survival (Paniw et al., 2022). The synergistic effects of climate change and infectious disease, mediated by energetics, greatly amplified the predicted risk of extinction for this population, highlighting the importance of a framework to address these stressors jointly and systematically.

5 | ALLOSTASIS AND THE FITNESS COSTS OF NON-LETHAL EFFECTS THAT LEAD TO POPULATION DECLINE

With climate and disease linked to specific energetic processes associated with fitness, we can draw on allostasis theory to generate predictions for the fitness costs of the two stressors. Allostasis denotes 'stability through change' (Wingfield, 2005), where

stability refers to homeostasis. Under allostasis theory, homeostasis reflects organismal physiology and behaviour under normal conditions, whereas allostasis encompasses physiology and behaviour exhibited over the spectrum of environmental conditions encountered by organisms over their lifespan, including periods with unfavourable conditions, like those created by climate change and infectious disease. The conceptual model of allostasis is based on the energy budget, but critically, includes a temporal dimension; the model tracks energetic demand, referred to as 'allostatic load' (Wingfield, 2005; Word et al., 2022), relative to external energy supply over time as environmental conditions fluctuate (Figure 3a). Allostasis highlights three fitness costs associated with non-lethal stressors: increased energetic demand, energetic trade-offs and accelerated physiological ageing. All three arise gradually and accumulate during recurrent or prolonged exposure to unfavourable conditions.

5.1 | Increased energetic demand

Completing routine activities under unfavourable conditions requires more energy (Bobba-Alves et al., 2022; Wingfield, 2005; Word et al., 2022), resulting in faster depletion of internal energy supplies. Infectious disease further depletes internal energy supply through direct consumption of host resources or host tissue by parasitic infectious agents (D2 in Figure 2) (Hall et al., 2009). As challenging conditions deplete internal energy supplies at heightened rates, organisms face an increased risk of going into an energy deficit. Energetic stress, a type of 'allostatic overload' (McEwen & Wingfield, 2003; Wingfield, 2005), describes the state of energy deficit in which organisms deplete internal energy supplies faster than they can replenish them. Under energetic stress, organisms cannot mobilise sufficient energy for maintenance, growth, and reproduction (Cressler et al., 2014b;

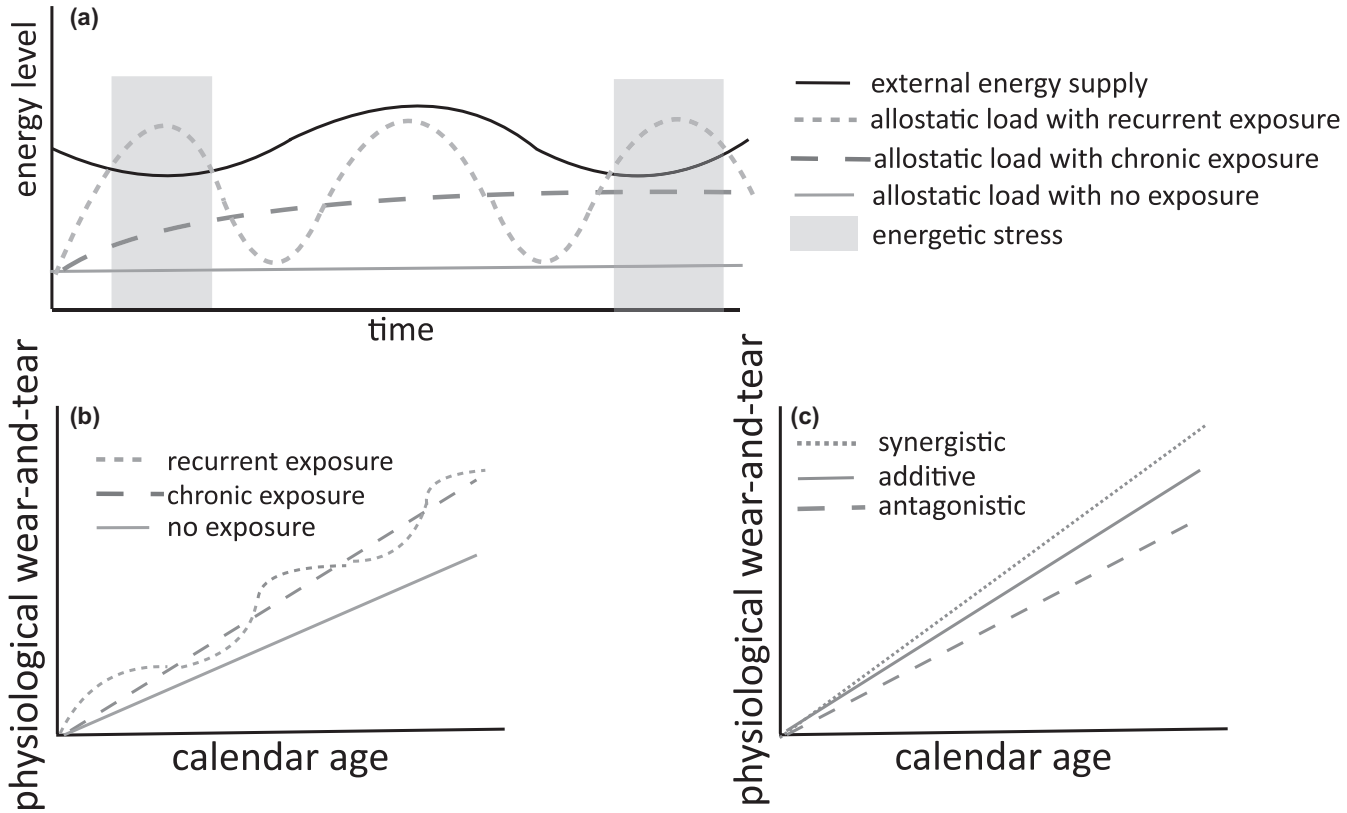

FIGURE 3 Temporal dynamics of allostatic load and hypothesised rates of physiological damage. (a) Adapted from Wingfield (2005); the allostasis model conceptualises the balance of external energy reserves (solid black line) and energy demands, (i.e. allostatic load, grey solid and dashed lines) over organism lifespans. Recurrent and chronic exposure to climate change and infectious disease impose energetic costs by increasing allostatic load (dashed grey lines) above baseline levels (solid grey line). Energetic stress occurs when allostatic load exceeds external energy supply (Type I allostatic overload (Wingfield, 2005); greyed areas). Energetic stress is less likely with chronic exposure than with recurrent acute exposure. However, both recurrent and chronic exposure cause costly physiological wear-and-tear that accumulates over organism lifespans (Type II allostatic overload, Wingfield, 2005) at rates expected to depend on (b) the frequency and duration of exposure to climate change and infectious disease, as well as (c) how climate change and infectious disease interact to jointly affect allostatic load. In (b), recurrent exposure to climate change and infectious disease are expected to cause temporary spikes in the rate of physiological wear-and-tear (denoted by sleeper slopes of the relationship, long dashed line), whereas chronic exposure should cause a continuously elevated rate of wear-and-tear (short dashed line), relative to background rates under no exposure (solid lines). In (c), additive (solid line) and synergistic (short dashed line) non-lethal effects of climate change and infectious disease should cause the highest rates of wear-and-tear because joint exposure results in stronger non-lethal effects than single exposure, which is not necessarily the case when their non-lethal effects are antagonistic (long dashed line). The relationships outlined in (b) and (c) are hypothetical and in need of testing.

Merila & Wiggins, 1997; Sokolova, 2013), which leads to physiological malfunctioning, reproductive failure, and heightened risk of mortality through starvation, predation, disease, etc. (McEwen & Wingfield, 2003). Organisms can avoid energetic stress by increasing energy assimilation rates, but this clearly depends on the availability of external energy and having the capacity to expand internal energy reserves sufficiently to sequester this added energy demand. All else being equal, limited external (e.g. food) or internal energy supply (e.g. fat stores) heightens the risk of energetic stress under climate change and infectious disease. Increases in energetic demand caused by climate change and infectious disease may be evaluated by comparing energy assimilation rates (C2 and D1 in Figure 2), energy mobilisation rates (C3 and D3 in Figure 2) and external energy supply (C1 in Figure 2) during exposure to the two stressors (see Section 7 for specific methodological approaches).

5.2 | Energetic trade-offs

Challenging conditions elicit stress responses that promote immediate survival at the expense of activities supporting long-term survival and fecundity. Stress responses may heighten energetic demand (McEwen, 1998; Sapolsky et al., 1986), exemplified by 'fight-or-flight' behaviours (Kay et al., 2021; Wingfield, 2013), or lower energetic demand, exemplified by shelter use (Creel et al., 2007; Sinervo et al., 2010) and self-isolation (Stockmaier et al., 2021). Although stress responses have evolved to increase short-term survival in challenging situations, they often elicit reductions in energy mobilisation for feeding, reproduction and other fitness-related activities (Sapolsky, 2004). Such trade-offs can gradually reduce lifespan and fecundity when stress responses are repeatedly or chronically expressed. A study of lizard extirpations illustrates how even subtle stress responses can lead to dangerous reductions in survival and fecundity (Sinervo et al., 2010). Climatic changes that increased daily hours of unsuitably high temperatures forced lizards to behaviourally thermoregulate by spending more time sheltering from direct sunlight to avoid overheating (Sinervo et al., 2010). This response was itself not energetically costly but reduced energy allocated to foraging and mating, and ultimately led to a reduction in the population growth rate to below-replacement levels (Sinervo et al., 2010). Modeling the energetic trade-offs of more frequent sheltering explained population extirpations and established a mechanistic, non-lethal link between climate warming and population collapse (Sinervo et al., 2010). Challenging conditions, like those created by climate change and infectious disease, can force unsustainable shifts in energy away from activities and physiological functions supporting long-term survival and fecundity, and eventually drive population decline. The contribution of energetic trade-offs in shaping non-lethal effects of climate change and infectious disease can be evaluated by measuring changes in the mobilisation pathways (C3 and D3 in Figure 2) under different climate-disease scenarios.

5.3 | Accelerated physiological ageing

Challenging conditions presented by climate change and infectious disease can also generate fitness costs due to physiological strain, or progressive 'wear-and-tear' on physiological function over time (McEwen, 1998; McEwen & Wingfield, 2003). Wear-and-tear on physiological functions occurs as organisms age and is associated with a range of internal malfunctioning, from the build-up of cholesterol and the inhibition of neurogenesis (McEwen & Wingfield, 2003) to down-regulation of adaptive immune cell gene expression and dysregulation of protein homeostasis (Watowich et al., 2022), which together increase the risk of age-related diseases, and ultimately, mortality (Parker et al., 2022). The rate of wear-and-tear on physiological function is expected to be proportional to allostatic load (i.e. energy demand), such that heightened allostatic loads essentially accelerate the ageing process and shorten lifespan, presenting another mechanism by which repeated and/or chronic exposure to stressors can reduce survival and fecundity. Incorporating the combined stresses of climate change and infectious disease may result in additive, synergistic or antagonistic effects on physiological ageing (Figure 3c), depending on how climate and disease interact to alter energy budgets.

Few studies have investigated climate- and disease-induced accelerated ageing, but one illustrative example comes from free-ranging rhesus macaques (*Macaca mullata*) inhabiting the island of Cayo Santiago (Watowich et al., 2022). Watowich et al. (2022) compared age-related gene expression and transcription profiles of macaques surveyed before and after Hurricane Maria. The surviving macaques exhibited gene expression and transcription profiles typically found only in older individuals, profiles which were associated with inflammatory disease, disruption to protein folding networks and other age-related mortality factors. The authors used the gene and transcription profiles to estimate that the hurricane accelerated the age of surviving individuals by 1.96 years, which when scaled to human lifespans equated to 7–8 years. The study highlights that during severe, acute climate or disease events that cause conspicuous mortality, even the survivors are prone to non-lethal effects that degrade physiological function over time. The findings support the allostatic load model by showing that stressors expedite physiological wear-and-tear, perhaps due to heightened energetic demand under duress. The energetics underlying this physiological malfunctioning were not evaluated, nor were the duration of malfunctioning or consequences for lifespan, leaving data gaps concerning the mechanistic basis and demographic outcomes of stress-driven accelerated ageing (Table 1). Incorporating biomarkers of ageing, such as DNA methylation (Farrell et al., 2020; Horvath & Raj, 2018; Seale et al., 2022), could help fill existing knowledge gaps concerning climate- and disease-driven accelerated ageing and their impacts on lifespan and fitness.

5.4 | Hypothesised drivers of population decline

The fitness costs associated with non-lethal changes to energy budgets establish three testable hypotheses for how recurrent

TABLE 1 Guidelines and case studies for measuring the non-lethal effects of climate change and infectious disease. Data that can be collected to parameterize a model based on our framework (Figure 2) are listed. Data needed for testing the three proposed hypotheses for how non-lethal effects reduce fitness and lead to population declines (grey cells) are denoted with checkmarks. Joint non-lethal exposure to climate change and infectious disease may reduce fitness by increasing energetic demands beyond energy availability in the environment (Hypothesis 1), by eliciting stress responses that divert energy away from reproduction and long-term survival (hypothesis 2), and/or accelerating the aging process and associated physiological wear-and-tear (hypothesis 3). Case studies (blue cells) highlight data availability (checked cells) and data gaps (empty cells) in exemplar systems, including Case 1: Kalahari meerkats (*Suricata suricatta*) impacted by extreme heath and the parasitic agent causing tuberculosis (Paniw et al., 2022); Case 2: Various speciese of North American bats exposed to the parasitic agent causing white nose syndrome (Hoyt et al., 2021); Case 3: Lizards exposed to shifting climatic regimes (Buckley, 2008); Case 4: *Atelopus* frogs jointly exposed to unfavourably high temperatures and the parasitic agent causing chytridiomycosis (Cohen et al., 2019); and Case 5: Rhesus macaques exposed to hurricanes (Watowich et al., 2022). Checkmarks under case studies denote data that have been collected and reported in the cited articles. The list of data examples is not exhaustive. Data for case studies come from select articles and may not reflect the totality of data available for the case system.

			Hypothesized non-lethal drivers of population decline							
			Hypothesis 1	Hypothesis 2	Hypothesis 3	Case 1: Kalahari meerkats, extreme heat, & tuberculosis	Case 2: North American bats & white nose syndrome	Case 3: *Sceloporus* lizards & climate shifts	Case 4: *Atelopus* frogs & chytridiomycosis	Case 5: Rhesus macaques & extreme weather
	Variable	Data example	Energetic stress	Stress-induced energetic tradeoffs	Accelerated aging					
Environment 	External energy supply	Resource density	✓					✓		
		Resource quality (energy content)	✓				✓	✓		
	Disease risk	Parasite density	✓	✓	✓				✓	
	Weather	Temperature	✓	✓	✓	✓	✓	✓	✓	✓
		Moisture	✓	✓	✓	✓	✓	✓		✓
Individual 	Internal energy supply	Size	✓	✓		✓	✓	✓		
		Weight	✓	✓		✓	✓	✓		
		Food consumption	✓	✓		✓	✓	✓		
		Digestion efficiency	✓	✓				✓		
		Fat reserves	✓	✓			✓			
	Energy demands	Energy expenditure (metabolism)		✓			✓	✓		
		Activity budget		✓				✓		
	Stress level	Glucocorticoid levels		✓						
		H:L (or N:L) ratio		✓						
	Disease status	Parasite load	✓	✓	✓		✓		✓	
		Morbidity	✓	✓	✓	✓	✓		✓	
Population 	Demography	Population size	✓	✓	✓	✓	✓	✓	✓	✓
		Births	✓	✓	✓	✓	✓	✓		✓
		Deaths	✓	✓	✓	✓	✓	✓	✓	✓
		Dispersal		✓		✓	✓	✓		
		Biological aging			✓					
	Disease status	Infection prevalence/incidence	✓	✓	✓		✓		✓	
		Disease prevalence/incidence	✓	✓	✓	✓	✓			

and/or chronic exposure to climate change and infectious disease can drive population declines:

1. Climate change and infectious disease cause faster depletion of an individual's energy supply, reducing lifetime fitness.
   a. Key variables: weather, disease risk, internal energy supply (Table 1).
2. Climate change and infectious disease place organisms in a state of stress; individuals prioritise immediate survival mechanisms while diverting energy from reproduction, feeding and long-term maintenance.
   a. Key variables: weather, disease risk, internal energy supply and energy demand (Table 1).
3. Climate change and infectious disease accelerate ageing and age-related risk factors through progressive physiological dysregulation.
   a. Key variables: weather, disease risk and ageing (Table 1).

Specific variables associated with our framework, which are outlined in Table 1, can be measured to test the above hypotheses. Yet, multiple non-lethal effects may act in tandem and potentially interact to reduce fitness, calling for holistic measures of all framework variables. For example, immune defences and infection outcomes can be shaped by the physiological state of the organism in addition to energy availability (Cohen et al., 2012; Downs et al., 2014). The importance of holistic measures is illustrated in hibernating bats with white-nose syndrome (caused by the fungal parasite *Pseudogymnoascus destructans* [*Pd*]), which experience chronically elevated metabolic rates, impairment of numerous physiological functions and recurrent arousals from torpor (Hoyt et al., 2021; Verant et al., 2014). These initially non-lethal effects increase energetic demand up to twofold above normal levels (Verant et al., 2014), which rapidly depletes internal energy supplies and, together with physiological dysregulation, reduces the chances that bats survive to the end of hibernation (Hoyt et al., 2021; Verant et al., 2014). *Pd* is not directly lethal, but its multiple non-lethal effects on energy budgets indirectly reduce host survival and fecundity and lead to demographic decline (Grimaudo et al., 2024). A key untested question is how climate-driven changes in the timing and environmental conditions of hibernation act in tandem with *Pd* to impact the energetics of overwintering bats.

6 | TOLERANCE AS A BUFFER AGAINST ENERGY-DRIVEN POPULATION DECLINES

The costs associated with non-lethal effects predict a grim future for populations facing joint pressures from climate change and infectious disease, but evidence for tolerance provides some cause for optimism. In many cases, recurrent exposure to unfavourable conditions leads organisms to develop new sets of beneficial, homeostatic states that align with changed environments. These physiological adjustments may develop within or across generations due to plasticity (Beever et al., 2017; Buckley & Kingsolver, 2012), epigenetic modification (Jeremias et al., 2018) or natural selection (Buckley & Kingsolver, 2012; Campbell-Staton et al., 2020). In any case, the resulting physiological adjustments make unfavourable conditions more tolerable, meaning they become less threatening to homeostasis and less energetically costly. There is ample evidence to support the role of tolerance in allaying the damaging impacts of infectious disease (Medzhitov et al., 2012; Raberg et al., 2009). Evidence for tolerance to climate change comes from urban environments, where recurrent extreme high temperature events have selected for warmer thermal performance ranges in *Anolis* lizards (Campbell-Staton et al., 2020, 2021). In the context of energy budgets, tolerance reduces allostatic load under changed environmental conditions. Tolerance can also reduce the extent to which climate change and infectious disease elicit stress responses, which further reduces both energetic demand and energetic trade-offs with maintenance, growth and reproduction. Reducing the energetic costs associated with environmental change yields clear benefits to survival and reproduction of individuals and, in turn, the viability of populations.

7 | EMPIRICAL TESTING OF NON-LETHAL EFFECTS OF CLIMATE CHANGE AND INFECTIOUS DISEASE

The difficulty in empirically measuring energy flow in organisms marks a key challenge of bioenergetic models. Measuring allostatic load and accumulated physiological damage over an organism's lifespan is also not straightforward. Numerous tools for indirectly measuring energy flow and physiological damage have been developed and can be leveraged to test the consequences of non-lethal effects (Table 1). The multifaceted influences of climate and disease on energy budgets call for multidisciplinary methods integrating multiple assays. We highlight several methods that could be harnessed to advance this empirical research agenda.

7.1 | Energy assimilation and internal energy reserves

Internal energy reserves, such as stored fats, can be mobilised for maintenance, growth, maturation, or reproduction. 'Body composition models' provide estimates of internal energy reserves using easily obtainable length and mass measurements (Molnár et al., 2009). Some researchers have used ultrasound and magnetic resonance to measure fat and protein content directly (Schmaljohann & Eikenaar, 2017).

7.2 | Allostatic load

Metabolic rate is often used to estimate an organism's gross energetic demand. Direct measures of metabolic rate can be

obtained through respirometry, which measures oxygen consumption and/or carbon dioxide production (Mautz & Pekins, 1989; Niewiarowski & Waldschmidt, 1992); calorimetry, which measures heat exchange between organisms and the environment (Compher et al., 2006); and doubly labelled water, which measures carbon dioxide production from water labelled with 'heavy' hydrogen (^{2}H) and 'heavy' oxygen (^{18}O) (Westerterp, 2017). Calorimetry is preferred for measuring resting metabolic rates (Compher et al., 2006), whereas field metabolic rates (metabolic rates during normal activity) are often obtained using doubly labelled water (Hudson et al., 2013; Westerterp, 2010). Studies in mice have also used calorimetry to estimate activity-specific energy demands (Van Klinken et al., 2012), which creates opportunities for distinguishing energy mobilisation towards maintenance, growth, maturation, and reproduction.

7.3 | Energy mobilisation

Activity budgets, which are measures of time allocated to specific behaviours, can be combined with metabolic rate to link energetic demand to specific activities (Table 1). For example, data on the amount of time lizards spent moving and foraging across different temperatures were used to parametrise a species distribution model that predicted changes in lizard ranges under climate warming scenarios (Buckley, 2008). Because energy expenditure for many organisms involves movement, accelerometry is a contemporary method for estimating activity budgets (Brown et al., 2022; Nickel et al., 2021; Wilson et al., 2020). A study of pumas (*Puma concolor*) used accelerometry coupled with metabolic rate measurements to quantify the increased energetic expenditure of avoiding humans (Nickel et al., 2021). Similar approaches could be used to measure energetic consequences of avoiding infection (e.g. moving to lower risk habitat or self-isolation) and extreme climatic events (e.g. fleeing from wildfires and searching for water during drought), keeping in mind that accelerometry overlooks energetic demands not associated with movement (Wilson et al., 2020).

7.4 | Stress responses

Stress responses are marked by the activation of numerous physiological processes that change energy budgets, with the intention of restoring homeostasis. Measurements of physiological variables involved in stress responses offer estimates of the magnitude and duration of responses (reviewed by Gormally & Romero, 2020). The most commonly measured physiological variable to estimate stress responses are glucocorticoid (GC) hormones (cortisol and corticosterone) (Angelier & Wingfield, 2013; Davis & Maney, 2018; Sheriff et al., 2011). GCs are released into the bloodstream several minutes into the stress response. GCs initiate a cascade of processes aimed at restoring homeostasis, and temporary spikes in GC concentrations are indicative of responses to acute stress (Angelier & Wingfield, 2013; Davis & Maney, 2018). Although GC-based studies of stress have been criticised for being overly simplistic (Breuner et al., 2013; MacDougall-Shackleton et al., 2019; Romero & Beattie, 2022), documented correlations between GC levels from blood samples and energy expenditure suggest that GC measures can be used to factor acute stress responses into estimates of allostatic load (Johns et al., 2018).

Immune cell concentrations in the bloodstream, namely the ratio of circulating heterophils (neutrophils in mammals) to lymphocyte concentrations (H:L ratio or N:L ratio) are also a widely used physiological measure of stress, with higher values indicative of a stress response (reviewed by Davis & Maney, 2018). Importantly, heterophils get released later than GCs, and concentrations do not wane under repeated or chronic stress as do GC concentrations, making them a strong measure of sustained stress over longer time-scales (Davis & Maney, 2018). Ideally, both GCs and H:L (or N:L) ratios would be measured to develop a comprehensive stress response profile (Davis & Maney, 2018; Gormally & Romero, 2020), which can be integrated with metabolic rate to get a sense of the changes in allostatic load that accompany the stress response.

7.5 | Ageing

Measuring progressive physiological dysregulation, or 'wear-and-tear', associated with allostatic load requires persistent measures. Previous studies have used GC measures from samples that represent cumulative GC concentrations over longer periods (weeks-months) than blood samples (hours-days), including GC levels in feathers of birds (Johns et al., 2018) and the fur of mammals (Gormally & Romero, 2020; Sheriff et al., 2011). Telomere length, which predicts lifespan in numerous taxa (Boonekamp et al., 2014; Whittemore et al., 2019), may also be leveraged to quantify physiological wear-and tear and ageing (Nettle et al., 2017). Another promising biomarker of ageing is the epigenetic process of DNA methylation (Farrell et al., 2020; Horvath & Raj, 2018; Seale et al., 2022). DNA becomes methylated throughout organism lifespans, and the rate of DNA methylation increases under unfavourable conditions (Farrell et al., 2020; Horvath & Raj, 2018; Parrott & Bertucci, 2019; Pinho et al., 2022), which can result in accelerated ageing (Farrell et al., 2020; Horvath & Raj, 2018; Parrott & Bertucci, 2019; Pinho et al., 2022). Characterising DNA methylation levels in relation to calendar age has the potential to reveal the physiological consequences of an organism's lifetime stress history, but this approach needs more testing.

8 | CONCLUSIONS

The ecological relevance of non-lethal effects is well recognised in the context of consumer–resource interactions (Daversa, Hechinger, et al., 2021; Preisser et al., 2005) but has been less prominent in

global change research. By developing a framework that links climate and infectious disease jointly to energy budgets, we hope to motivate theoretical and empirical studies that lead to a more mechanistic understanding of how non-lethal effects challenge populations confronted with rapid environmental change. Assessing the fitness costs of non-lethal effects arising from energetic stress, energetic trade-offs and accelerated ageing highlights the relevance of recurrent and chronic events to population demography. Regardless of their threat to immediate survival, such events impose energy-mediated fitness costs that accumulate and act gradually to erode population viability, yet the demographic consequences of recurrent and chronic events associated with climate change and infectious disease are not captured by existing models. We hope the proposed framework enables more complete investigation of the ecological impacts of global change.

AUTHOR CONTRIBUTIONS

All authors contributed equally to idea formulation and development. David R. Daversa led the writing of the manuscript, which received multiple rounds of revisions by Daniel T. Blumstein, Jamie O. Lloyd-Smith, Gary M. Bucciarelli, and H. Bradley Shaffer. All authors contributed critically to the drafts and gave final approval for publication.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

There are no data associated with this manuscript.

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