

The consequences of rising temperatures for animal fertility

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Abstract

Thermal stress reduces fertility and fecundity in animals at temperatures below lethal. Reproductive output is impaired across taxa under diverse heat-exposure regimes, with consequences for individual fitness, population persistence and ecosystem dynamics. This pattern holds across terrestrial and aquatic systems, with implications for conservation, livestock, aquaculture and human health. Yet these sublethal effects remain underrepresented in biodiversity forecasts. In this Review, we synthesize evidence for the biological mechanisms associated with thermally induced declines in fertility and fecundity, and assess how life history and exposure regime can shape thermal sensitivity. Fertility-based thermal limits can predict species distributions and extinction risk better than survival-based measures, albeit tested across a limited taxonomic range. Evolutionary responses to fertility loss under warming seem constrained but increased mutational variation, local adaptation and hybridization might increase fertility resilience. Key research priorities include broader taxonomic evaluation of both evolutionary potential and ecological outcomes under more sophisticated conditions, assessing how fertility is affected when different environmental stressors interact, and understanding how community and ecosystem dynamics will change if fertility-sensitive taxa either shift distributions or go extinct. Recognizing and addressing fertility-based vulnerability is essential for anticipating biodiversity change and designing more effective responses to climate impacts.

Sections

Introduction

Patterns of heat-induced sterility

Mechanisms of heat-induced fertility loss

Variation in thermal vulnerability

Ecological and evolutionary effects

Evolutionary potential and constraints

Summary and future perspectives

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Key points

- Elevated temperatures can decrease reproductive output at temperatures below those causing mortality; hence an understanding of this phenomenon is critical for predicting organismal responses to climate change, including downstream effects on humans
- Variation in thermal vulnerability within and between species is shaped by how temperature affects the fertilization environment experienced by gametes, the interaction between the magnitude, duration and frequency of heat exposure, and by the relative sensitivity of each sex and life-history stage; additional complexity arises under multiple stressor conditions and from uncertainty as to whether phenotypic plasticity aids or exacerbates responses
- Heat-impaired reproductive output alters predictions of climate change impacts on species distributions and population persistence; however, evidence for ecological and evolutionary consequences is taxonomically limited, and scenarios involving shifting species interactions and community-level variation in thermal fertility sensitivity are unexplored
- The potential for adaptive increases in reproductive heat tolerance depends on heritable genetic variation, sex-specific genetic architecture and linked traits, and temperature-dependent DNA mutation; yet evidence for adaptive responses is sparse, limited by a small number of studies in few taxa
- Recognizing fertility-based vulnerability is essential for assessing the effects of climate change on populations, species, communities and ecosystems, including subsequent ecosystem service effects; improved taxonomic coverage under more complex conditions is required to anticipate ecological and evolutionary consequences and responses across scales

Introduction

Rising global temperatures are restructuring ecosystems and threatening biodiversity worldwide. Both the mean and variance in temperature are increasing, with more frequent and long-lasting extreme weather, including heatwaves¹. Although much attention has focused on the lethal effects of warming², reproductive processes are well known to be negatively affected at sublethal thermal stress in both sexes³. The thermal range over which fertility and fecundity (also referred to holistically as reproductive output) is possible is typically narrower than the survival range⁴ and can fail at temperatures that do not affect survival^{5,6}. Although fertility and fecundity are major drivers of population persistence, these non-lethal effects on reproduction remain underrepresented in predictions of species vulnerabilities and responses to climate change³. A thermal performance curve (TPC) can be used to measure thermal sensitivity and make these predictions (Box 1). Research has found that upper thermal fertility limits (TFLs) are better predictors of both current species ranges and temperature-related extinction than critical thermal limits (CTLs), such as the critical thermal maximum (CT_{max}) or lethal temperature^{5–7} (Box 1), and so are likely to be of critical importance to biodiversity. Importantly, the societal effects are potentially far-reaching because heat-induced sterility occurs in livestock⁸, pollinators⁹ and humans too¹⁰ (Fig. 1). Thus, temperature-mediated

effects on fertility and fecundity merit closer examination given their potential to influence ecosystem services and population persistence, thereby altering species interactions and community structure.

In this Review, we focus on how elevated temperatures reduce reproductive output by directly impairing gamete production, fertilization success and fecundity. We begin by outlining patterns of heat-induced reductions in reproductive output, then review physiological and molecular mechanisms underlying thermal damage to gametes, fertilization and fecundity. We highlight factors influencing variation in thermal effects on reproduction, such as sex, fertilization mode and exposure duration and how phenotypic plasticity might mediate responses. To gauge potential effects on biodiversity, we assess the ecological and evolutionary consequences of thermal limits to fertility, including on predictions of species redistribution, extinction risk and sexual selection. We also discuss the potential for adaptive evolutionary responses to increased warming. Finally, we outline research priorities for better predictions of the extent to which thermally induced reproductive decline will affect biodiversity under conditions of ongoing climate change.

Although temperature also alters traits that influence reproductive success – such as mating behaviour¹¹, reproductive timing and/or phenology¹² or parental care¹³ – our emphasis is on thermal effects on parental fertility and fecundity. Temperature can also influence post-fertilization mortality of embryos or offspring; experiments have frequently not disentangled the effects of heat stress on parental fertility and fecundity from those on offspring viability¹⁴. To minimize this problem, we highlight studies in which gametes or parents, rather than embryos or offspring, experienced heat stress.

Patterns of heat-induced sterility

Research on thermal effects on fertility is dominated by terrestrial invertebrates (particularly insects) and laboratory animal models¹⁴ (Supplementary Table 1). Nevertheless, there are examples from amphibians¹⁵ (Fig. 1a), reptiles¹⁶ (Fig. 1b), birds¹⁷ (Fig. 1c) and wild mammals^{18,19} (Fig. 1d). There has also been focus on economically important livestock⁸ (Fig. 1e; Supplementary Table 1). Even human sperm quality is declining, some of which is epidemiologically linked to temperature variability¹⁰ (Fig. 1f). Conservation and captive breeding programmes might also be at risk when animals are kept outside at ambient temperatures – owing to negative thermal effects on fertility and fecundity²⁰. Warming can affect pollinators by reducing sperm viability²¹, which is compounded by mated queens storing sperm for months or years⁹. Conversely, heat suppression of fertility and fecundity in pests or disease vectors might offer economic benefits²².

However, responses are heterogeneous (Fig. 1); temperature-induced changes in fecundity and fertility might be positive, negative or absent depending on a variety of factors such as species, sex, life stage and type of thermal exposure (that is constant, fluctuating and/or extreme). For example, in the mussel, *Mytilus galloprovincialis*, warmer temperature increases fertilization rate²³ whereas the opposite occurs in the clam, *Tridacna maxima*²⁴. Both sexes might be negatively affected (Fig. 1c), or one sex may be more susceptible. Effects on fecundity might result from complex interactions with species-specific ecological traits (Fig. 1d) or life-history traits, such as body size¹⁶ (although see elsewhere^{25,26}). Higher winter temperatures can also influence spring fecundity, such as in the wood frog, *Rana sylvatica*¹⁵ (Fig. 1a). Predicting whether fertility and fecundity themselves are negatively affected, and for which species under what thermal conditions, remains challenging, and might depend on the approach taken to measure the effects of heat stress^{7,27,28} (Box 1).

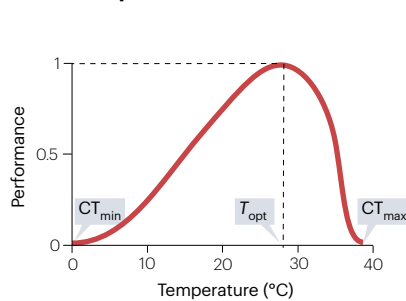
Box 1 | Measures of thermal tolerance

Thermal tolerance can be summarized by a thermal performance curve (TPC), which plots trait performance across temperatures¹⁶⁵ (figure **a**; we note that curves in all panels are hypothetical). TPCs are typically unimodal and asymmetric, with performance declining steeply at higher temperatures^{166,167} (figure **a**). TPCs describe thermal tolerance by determining the temperature that maximizes performance (T_{opt} ; dashed line in figure **a**), and the critical thermal limits (CTLs) that describe the lower (CT_{min}) and upper (CT_{max}) thermal limits at which performance fails (figure **a**). Given that TPCs across different performance traits and taxa can be quantified with the same variables, and that temperature has large consequences on organismal function, TPCs have been used to predict how organisms will respond to climate change^{137,168,169}.

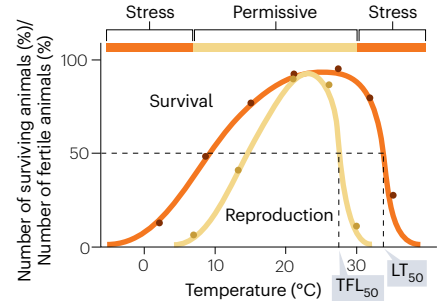
The effect of temperature on survival (binary trait) can be quantified using a TPC measuring either the CTLs, the temperatures at which individuals lose coordination or motor function (CT_{max} and CT_{min}), or the lethal temperature, the temperature at which a percentage of individuals die² (LT_{50} indicates the temperature at which 50% of test subjects die, marked by a dashed line to the orange curve in figure **b**). These assays usually apply heat acutely, either statically for a set time period at a particular temperature or by ramping the temperature at some rate until physiological failure occurs⁵. Similarly, fertility endpoints can be assessed using thermal fertility limits (TFLs)⁷ (TFL_{50} indicates the temperature at which 50% of test males are sterile, marked by a dashed line to the yellow curve in figure **b**) after heat exposure. Permissive temperatures are those that allow both successful reproduction (yellow curve) and survival (orange curve) to occur; anything outside this region is stressful (figure **b**).

A higher TFL indicates greater heat tolerance (dark blue line in figure **c**). High and low tolerance can represent variation between the sexes, populations, species, life-history stages and fertilization mode. Because fertility cannot be directly observed, TFL assays use static rather than ramping exposures⁷. Heat-stress effects on reproductive output can also be assessed using thermal sensitivity of fertility, in which fecundity (such as egg or offspring number) at a small number of test temperatures is determined⁷⁸⁵. The greater the difference between temperature treatments, the greater the sensitivity. Designs can vary by using either acute exposure of a few hours to mimic the hottest part of the day during a heatwave, or longer exposure across several days to mimic the full duration of a heatwave, or chronic exposure across weeks or months to mimic long-term changes in climate⁷¹⁴.

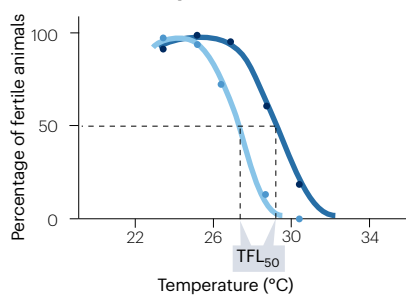
a Thermal performance curve



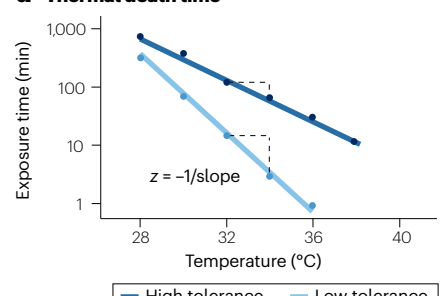
b Thermal limits



c Thermal fertility limit



d Thermal death time



Another design intended to capture the effect of thermal stress on performance is the thermal death time approach (figure **d**). Here, the effects of temperature on performance are determined by integrating a variety of exposure times and temperatures, assuming that thermal injury accrues in a dose-dependent way²⁷. Integrating exposure time and temperature with log-transformed exposure duration results in some performance outcome (such as a 50% loss of fertility) plotted against temperature⁴ (figure **d**). This approach captures how milder stress over longer periods can cause similar damage to acute extremes. Higher thermal tolerance is indicated by a shallower negative slope⁴ (dark blue line in figure **d**) with sensitivity quantified by parameter z (dashed lines in figure **d**). Some studies have shown that the thermal death time approach improves predictions of organismal response to high temperatures^{170,171}. Only one study of thermal death time has assessed fertility, finding that heat-induced sterility in *Drosophila suzukii* followed a dose-dependent relationship, with sterility occurring at significantly lower temperatures than would cause death⁸⁷.

This brief summary of different ways to measure the effects of temperature on organismal performance reflects a continuing debate on the most ecologically relevant way to measure thermal tolerance, including other approaches that, for example, integrate exposure duration and thermal intensity^{28,172}.

Mechanisms of heat-induced fertility loss

High-temperature stress damages fertility and fecundity through diverse physiological and molecular pathways, including effects on the following: gamete quantity and quality; reproductive fluids; sperm

storage; rate of fertilization; the conserved heat-shock response; and gene expression (Fig. 2; Supplementary Table 1). Reproductive impairment can occur in both sexes and can have transgenerational effects. Understanding these mechanisms, along with the potential

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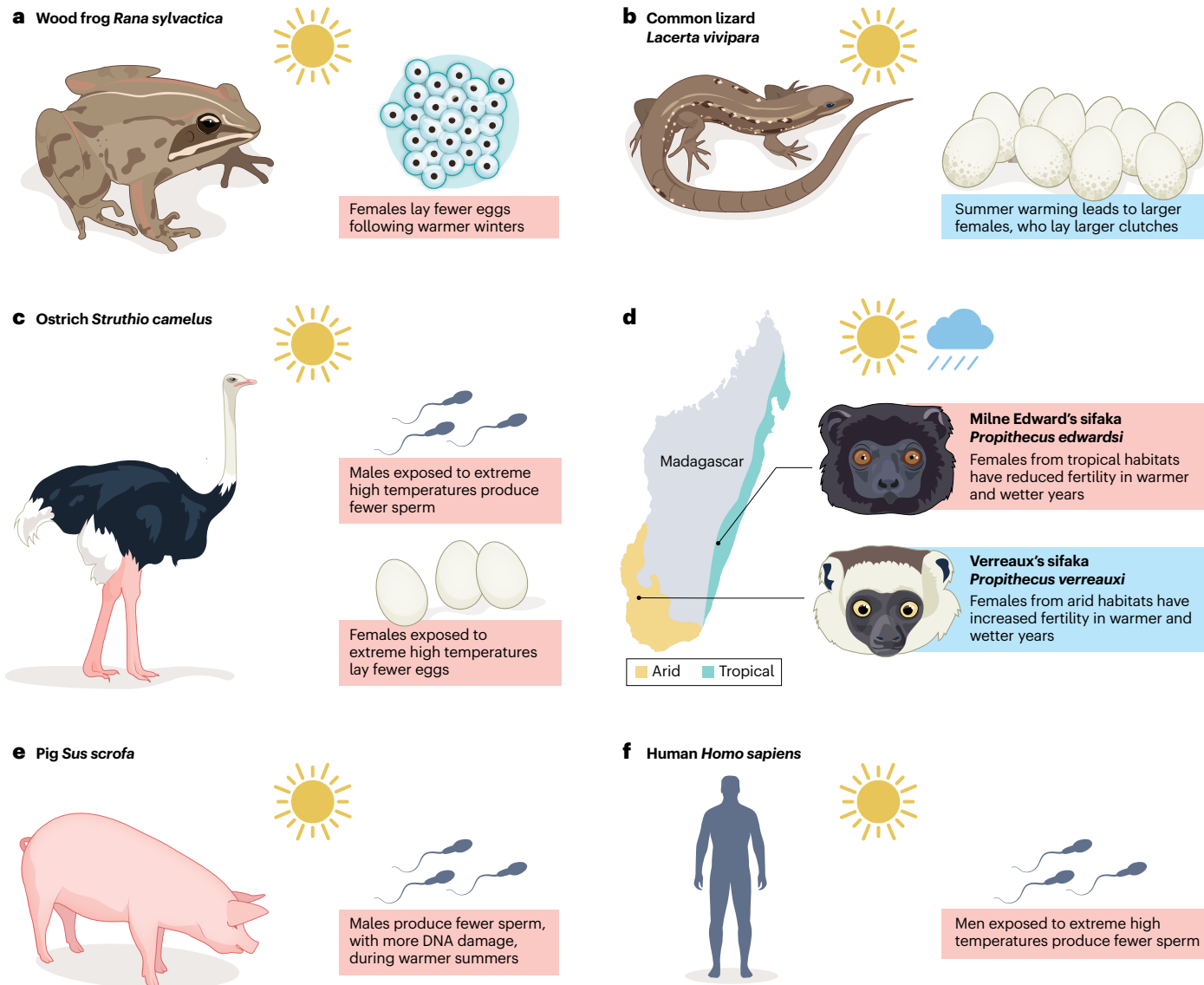


Fig. 1 | Temperature effects on reproductive output are variable and taxonomically widespread. **a**, Warmer winters in the midwestern USA reduce female fecundity in the wood frog, *Rana sylvatica*¹⁵. **b**, By contrast, female common lizards (*Lacerta vivipara*) from southern France born in warmer years mature at a larger size and so produce larger clutches¹⁶. **c**, Ostriches (*Struthio camelus*) breeding in South Africa are regularly exposed to large temperature extremes; extreme high temperatures cause females to lay fewer eggs, and males to transfer fewer sperm during mating¹⁷. **d**, In Madagascar, the effect of climate on breeding in sifakas differs across species. Female Milne Edward's sifakas (*Propithecus edwardsi*)

living in the tropical, mountainous east of the island have reduced fertility in warmer and wetter years¹⁹. By contrast, female Verreaux's sifakas (*Propithecus verreauxi*) living in the arid south of the island have higher fertility in warmer and wetter years¹⁸. **e**, High temperatures can also influence the fertility of species used in an agricultural setting. For example, domestic boars (*Sus scrofa domesticus*) reared in tropical Australia produce fewer sperm with more DNA damage in warmer summers⁸. **f**, High temperatures also impair the fertility of men. For example, Chinese men exposed to high temperatures 0–90 days before they visited a sperm bank had reduced sperm quality¹⁰. Data in **d** adapted from ref. 155.

for recovery, is vitally important for predicting fertility resilience, conservation, human health and protection of agriculture in the face of global warming.

Gamete effects

Elevated temperatures can impair gametogenesis in both sexes, resulting in reduced gamete quantity (Fig. 2; Supplementary Table 1). For example, heat stress can result in smaller testes or ovaries^{29–31},

increased apoptosis of developing gametes^{32–36} and impaired gamete maturation^{29,35,37} resulting in reduced gamete production^{30,32,38–40}. Mature gametes can also have impaired function (Fig. 2; Supplementary Table 1). For example, heat stress can result in sperm that swim more slowly^{38–40} or have reduced viability^{29,40–42}, and sperm and eggs that exhibit more DNA damage^{34,37,43}. In some species, a brief heat shock causes immediate and long-lasting male sterility, consistent with irreparable damage to spermatogenesis (Supplementary Table 1). In other

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species, males remain fertile initially but become sterile days later⁵. The causes of this variation are unclear but, in species with delayed sterility, one possibility is that immature sperm are more heat-sensitive than mature sperm. As existing mature sperm are depleted, they are gradually replaced by sperm that were developing during the heat stress, leading to a delayed onset of sterility^{5,44,45}.

Heat stress can induce oxidative stress through disruption of mitochondrial function, generating reactive oxygen species. An increase in

reactive oxygen species in ovaries and testes after heat stress causes DNA damage and triggers apoptosis and autophagy pathways that decrease gamete quantity and quality^{39,46}. In *Drosophila melanogaster* laboratory lines, male fertility sensitivity to heat stress correlated with other work on these lines showing lifespan reduction following oxidative stress⁴⁷; these patterns were mirrored at the genetic level, for which susceptibility to heat-induced male sterility was associated with genes involved in DNA-damage repair and autophagy⁴⁸. Similarly, in

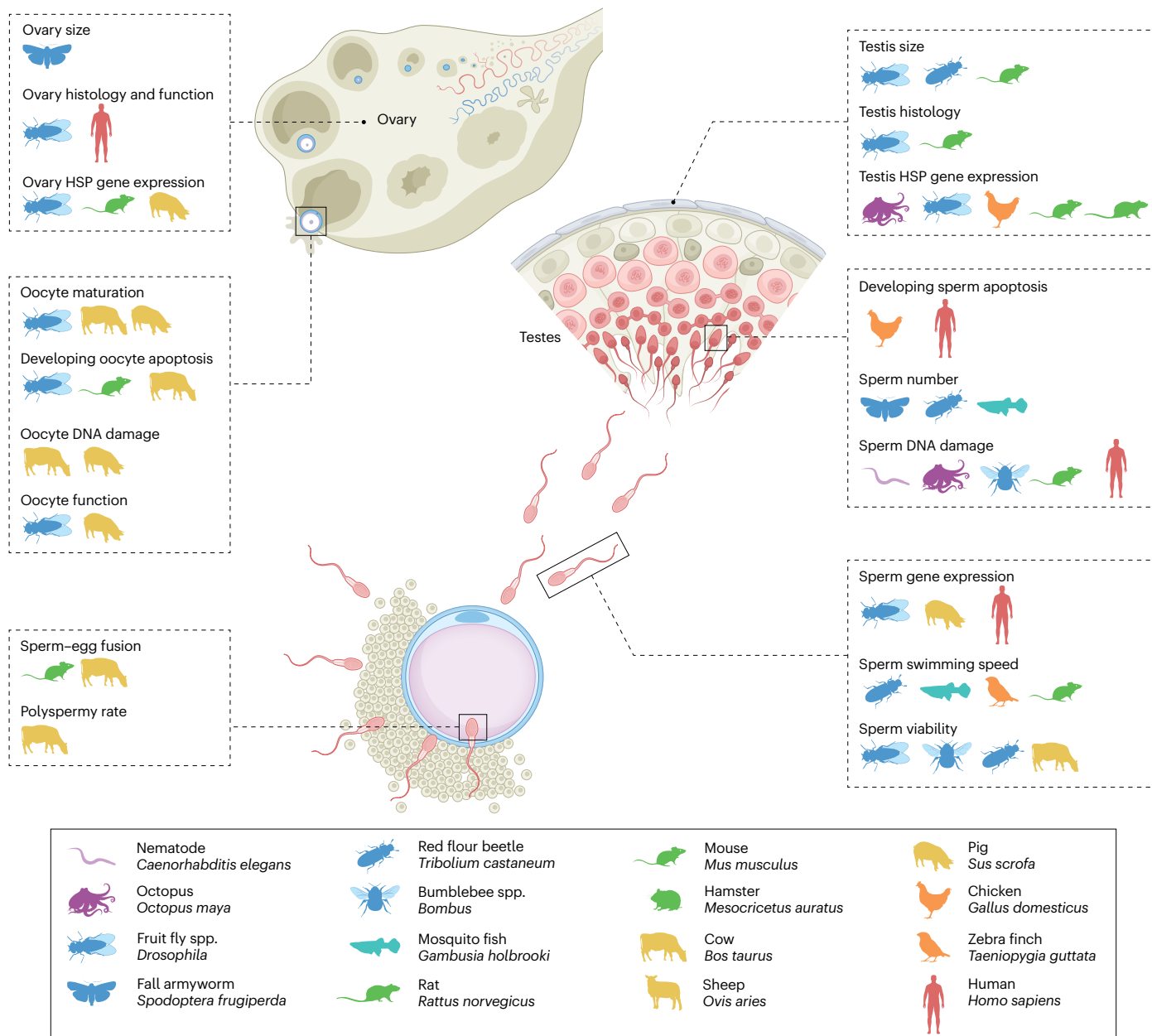


Fig. 2 | Generalized schematic of processes of animal reproduction, identifying the timing and mechanism of heat-stress effects, where evidence shows abnormal processes under heat stress compared to benign conditions.

We focus on processes from gamete formation to fertilization, including the following: ovary function (including size³⁰, histology and function^{32,114,156}, and ovary heat-shock protein (HSP) gene expression^{33,51,69,157,158}); oocyte development

(maturation^{35,37,159}, apoptosis^{32,33,35,114}, DNA damage^{35,37} and function^{32,160}); fertilization success (sperm-egg fusion^{57,58} and polyspermy rate⁵⁸); testis function (size^{31,41,77,161}, histology^{36,77,78} and HSP gene expression^{31,33,36,51,63,64,68,162}); sperm development (apoptosis^{34,63}, number^{30,38,40,161} and DNA damage^{34,42,43,64,162,163}); and sperm function (gene expression^{34,61,74}, swimming speed^{31,38,40,164} and viability^{29,40-42,58,161}). For further information, see Supplementary Information.

Caenorhabditis elegans, DNA damage was linked to heat-stress effects on spermatogenesis but notably not on oogenesis⁴³. Reactive oxygen species and heat stress can also affect transposable element activity^{43,49}, which influences sex-specific gene regulation, and can generate novel genetic and phenotypic variation⁵⁰. In *Drosophila subobscura*, heat stress resulted in different transposable element expression patterns between the testes and ovaries⁵¹, although whether this difference is associated with reduced fertility and DNA damage is unknown. Links between reactive oxygen species and sex-specific transposable element responses to heat stress should be assessed for their contribution to taxonomic variation in sex-specific heat-induced declines of reproductive output.

Effects on reproductive fluids

Both sexes produce reproductive fluids that affect fertilization and can mediate how heat stress affects reproductive output. For example, ejaculates contain seminal proteins, which have roles in sperm motility, storage and female post-mating responses (such as increased egg-laying⁵²). In *D. melanogaster*, heat stress during development reduced the size of the adult male accessory glands that produce seminal fluid and decreased males' ability to prevent female remating, suggesting damage to the amount or quality of seminal proteins⁴¹. In the lizard *Tropidurus spinulosus*, female ovarian fluid was found to mitigate negative temperature effects on sperm velocity⁵³. In the fish *Symphodus ocellatus*, warmer water decreases the extent to which ovarian fluid biases fertilization towards high-performing sperm of dominant males⁵⁴. Thus, the potentially high-temperature-buffering roles of sex-specific reproductive fluids vary among taxa.

Sperm storage and fertilization failure

In species in which females store sperm, heat stress might substantially reduce stored sperm viability^{40,55}. Whether this reduction arises from the female's inability to maintain sperm viability under heat stress, or from sensitivity of mature sperm to thermal conditions during storage, is unknown. In *Drosophila virilis*, female fertility recovered following remating after heat stress, suggesting that damage to previously stored sperm rather than to the female reproductive tract was the cause of infertility⁵⁵.

Even if sperm and eggs meet, heat stress can decrease fertilization success^{23,24} (Fig. 2; Supplementary Table 1). In *D. melanogaster*, sperm from developmentally heat-stressed males fertilized fewer eggs and, when fertilization did occur, further embryo development was not supported⁵⁶. In mammals, in vitro heat stress increases sperm-egg fusion failure⁵⁷ and increases lethal polyspermy⁵⁸. Increases in polyspermy were associated with decreased transcript abundance of a gene involved in preventing polyspermy⁵⁸.

Conserved heat-shock response

A central defence against thermal stress is the heat-shock response, an evolutionarily conserved cellular programme that includes changes in gene expression of heat-shock proteins (HSPs), immune genes and other stress-response pathways⁵⁹. HSPs are essential for gametogenesis⁶⁰. Transcriptome analysis of testes of pigs⁶¹, mice³¹, rats³⁶, rabbits⁶², chickens⁶³ and octopus⁶⁴ and ovaries in rabbits⁶⁵ and *Drosophila*⁵¹ reveal temperature-driven changes in expression of HSPs and genes associated with oxidative stress, DNA-damage repair, apoptosis and immunity, and small and micro RNAs (Fig. 2; Supplementary Table 1). Whether sex-specific variation in the magnitude and direction of transcriptional responses in the gonads⁵¹ underlies sex differences in heat-induced

declines of reproductive output is unknown. HSPs in females from monogamous species or from species that store sperm for long periods, such as honeybees, may protect stored sperm. For example, honeybee females increase expression of HSPs in their spermathecae after heat shock⁹. Likewise, anti-oxidative responses in females might protect stored sperm from heat stress. However, no increase in GAPDH (which helps to maintain stored sperm) or in enzymes that function in anti-oxidative stress was found in heat-shocked honeybee females⁹. Peak HSP induction has been linked to differences in CT_{max} between populations of killifish (*Austrofundulus limnaeus*)⁶⁶ and *Drosophila* species⁶⁷, although the relationship with TFLs remains to be investigated. A study examining changes in expression patterns of seven HSP genes of six *Drosophila* species with varying lethal temperatures and TFLs found that species with lower lethal temperatures showed greater upregulation of these genes in somatic tissue following a mild heat shock⁶⁸. Although differential regulation in response to mild heat shock was noted in reproductive tissue, there was no clear pattern in relation to the species' absolute TFL. However, there was some indication that species with a TFL lower than their lethal limit (Fig. 3a) showed less differential regulation in reproductive tissue⁶⁸. Thus, while HSP regulation seems to be consistently associated with measures of somatic maintenance following heat shock, how HSPs function to mediate heat-induced sterility might be species-specific, requiring further study.

The kinetics of the heat-shock response can vary with exposure duration. In female mice, chronic heat exposure resulted in substantial damage to ovarian function and elicited sustained HSP gene-expression changes even after 6 weeks under benign conditions⁶⁹. By contrast, acute exposure elicited transient HSP responses that returned to baseline within hours with reduced ovarian damage⁶⁹. However, the link between gene-expression kinetics and reproductive damage is untested. Generally, upregulation of heat-shock responses for an extended period is likely to be costly⁷⁰, and potentially suggestive of poor adaptation to the thermal environment⁷¹. Exposure regime might also alter HSP gene expression. In killifish liver tissue, small HSPs altered gene expression more strongly in response to fluctuating temperatures, whereas larger HSP chaperones (such as Hsp70 and Hsp90) were more active under chronic heat⁷². Whether reproductive tissue shows a similar pattern and how this affects reproductive output was not assessed.

Epigenetic effects

The heat-induced changes in gonad gene expression discussed above might be the result of heat stress causing various types of epigenetic modification, such as DNA or chromatin methylation or acetylation. These modifications alter gene expression, generate phenotypic plasticity and influence phenotypes across generations^{30,50}. In the European corn borer moth (*Ostrinia nubilalis*), heat stress during pupal and adult life stages increased DNA methylation, with higher rates in females. Although methylation was localized to unique genomic regions in each sex, some methylation shifts were associated with gametogenesis, indicating that both oogenesis and spermatogenesis could be affected epigenetically by heat stress – but whether reproductive output was affected was not examined⁷³. Negative transgenerational effects, in which heat-exposed males sire offspring with reduced fertility, occur in the red flour beetle *Tribolium castaneum*⁴⁰ and the bean weevil *Callosobruchus maculatus*⁴⁴ but not in *D. melanogaster*⁷⁴. In cows, heat stress in the F_0 generation leads to a reduced conception rate in F_1 daughters, an increased interval from calving to first insemination in F_2 granddaughters, and

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increased stillbirths in F_3 great-granddaughters⁷⁵. What epigenetic mechanisms cause these transgenerational effects and why taxa vary in responses is undetermined.

Fertility recovery after heat stress

The extent and timing of fertility recovery following heat stress dictates the duration of an individual's absence from the mating pool, which influences other aspects of fitness, such as sexual selection, and subsequent population dynamics⁷⁶ (Box 2). Partial or complete recovery following heat stress can occur in some species under some conditions^{41,77–79} (Supplementary Table 1). In the insect pest, *Monochamus alternatus*, individuals heat-stressed for 15 days showed full recovery of fecundity, sperm number and sperm viability within 4 weeks, although recovery time varied across these traits⁸⁰. Recovery was mediated in part by Hsp20, which seemed to help to refold reproductive proteins during the recovery phase, suggesting that HSPs might also influence longer-term recovery after heat stress.

Recovery potential might depend on the severity and duration of heat stress, the mechanism behind sterility and the exposed life stage. For instance, exposure duration influenced recovery rates in adult female *D. melanogaster*, although fertility was never restored to control levels³². In this species, the extent of fertility recovery for males was reduced as temperatures increased, but was particularly diminished when heat stress was applied to either the adult or pupal stage compared to earlier developmental stages²⁹. Uncovering the interplay between the extent of reproductive damage, protective responses, and variation in sex and life-history stages that mediate recovery potential will be key to better predict how thermal variability affects reproductive output and subsequently influences population persistence.

Variation in thermal vulnerability

Consequences of thermal stress on reproductive output vary, both within and between taxa, with individuals of some populations or some species being more resistant than others (see section 'Patterns of heat-induced sterility'). Identifying patterns in factors influencing this variation can help to predict which organisms might be more at risk. Here we examine four factors that mediate thermal stress impacts on fertility and fecundity: sex-specific effects, with potential downstream consequences on sexual selection (Box 2), fertilization mode, life stage at exposure and exposure regime. We also consider how different types of phenotypic plasticity influence variation in reproductive output responses.

Sex-specific effects

A key outstanding question is whether fertility and fecundity are more sensitive to warming in males or females⁸¹ and what the ultimate effect of this sex-specific variation is on evolutionary responses and population persistence. Female fertility loss is expected to be more detrimental to population fitness given that females generally have a lower potential reproductive rate than males⁸². However, most research exposed either only one sex or both sexes simultaneously to heat stress, with the latter approach typically not delineating sex-specific effects given that heat-stressed individuals were not mated to control mates¹⁴. This reduces the ability to adequately assess sex-specific contributions to heat-induced fertility loss. Male fertility is thought to be more sensitive to high-temperature stress because sperm are particularly sensitive to heat^{5,6,29,47,83}. However, a meta-analysis spanning 241 aquatic invertebrates and ectothermic vertebrates found that the sexes had similar responses to temperature effects regarding fecundity and gamete

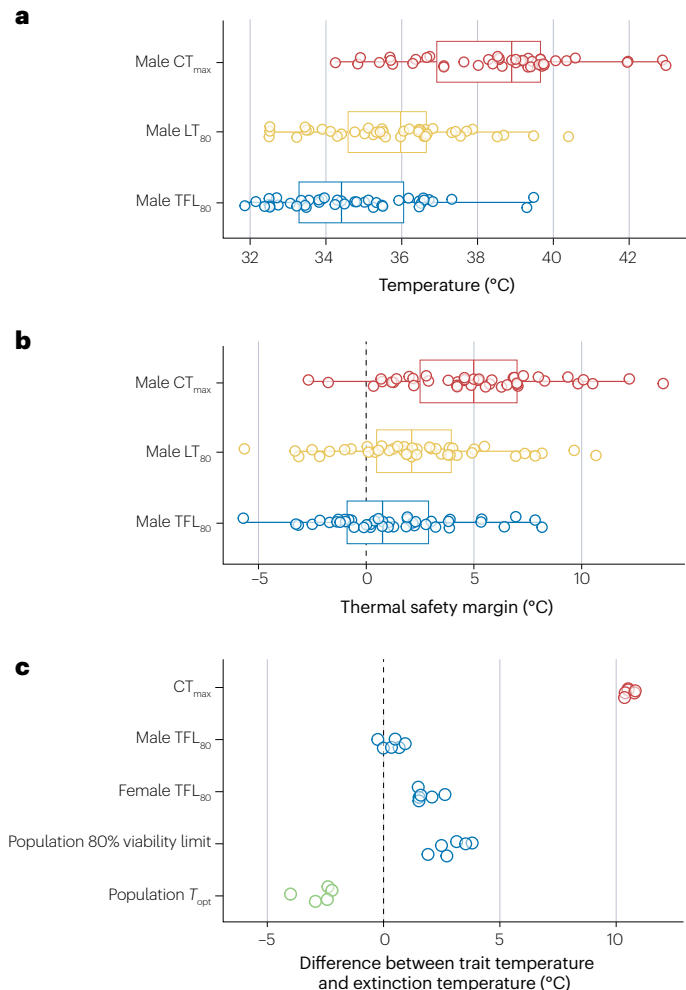


Fig. 3 | The ecological relevance of thermal fertility limits. **a**, Across 43 species of *Drosophila*, the male thermal fertility limit (TFL), at which 80% of males are sterile (TFL₈₀; blue points) tends to occur at a lower temperature than either the male 80% lethal temperature (LT₈₀; yellow points) or the male critical thermal maximum (CT_{max}; red points). **b**, Across 43 species of *Drosophila*, the male TFL₈₀ (blue points) has a narrower thermal safety margin than either the LT₈₀ (yellow points) or the CT_{max} (red points). The box shows the interquartile range and the median. The whiskers extend to the most extreme value no further than 1.5× interquartile range to either side of the box. Data in panels **a** and **b** are adapted from ref. 5. **c**, Across six species of *Drosophila* fruit fly, the temperature at which laboratory populations go extinct is almost identical to the male TFL₈₀. The female TFL₈₀ and other metrics of population viability either overestimated (such as CT_{max}; red points) or underestimated (such as the optimum performance temperature, T_{opt} ; green points) the extinction temperature. The population 80% viability limit is the temperature at which 80% of eggs fail to reach adulthood. T_{opt} is the temperature that maximizes population fitness, here estimated as a composite of egg-to-adult viability, developmental speed and fecundity. Data in **c** adapted from ref. 6.

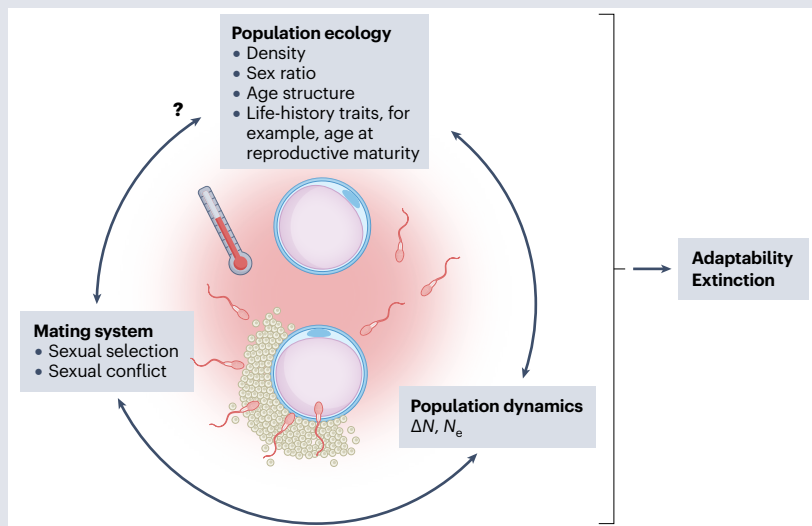
traits⁸⁴. In terrestrial organisms, sex differences for the impact of thermal stress on fecundity seem to be variable⁸⁵, with different studies demonstrating more negative effects either for males^{29,40,47,86} or females^{17,87–89}, or showing no sex differences^{22,90}. Determining the extent of sex-specific variation and what factors contribute to this variation

Box 2 | Sexual selection and heat-induced sterility

Rising temperatures can influence sexual selection via heat-induced reduction in fertility and fecundity⁷⁶. Sexual selection might either promote adaptation by purging deleterious alleles or hinder it by escalating sexual conflict and reducing female fitness^{173,174}. Ecological factors are key influencers of mating systems^{175,176}, shaping both the strength and sex-specific direction of sexual selection, sexual conflict and sexually antagonistic coevolution^{176,177}. Previous reviews have highlighted that the interaction between thermal ecology and sexual selection is of major interest¹⁷⁸, but here we focus on how heat-induced sterility affects sexual selection, an area largely overlooked (see figure).

Heat-stress reductions in fertility and fecundity directly affect population dynamics, including change in population size (ΔN) and effective population size (N_e), influencing genetic variation and evolutionary consequences, while altering population ecology parameters. These population-level factors in turn influence mating systems^{179–183} (including sexual selection and sexual conflict). All of these components interact to determine adaptability and extinction probability, although whether there is a reciprocal effect of sexual selection on ecological parameters is uncertain¹⁸⁴. Some research has begun to investigate whether heat-induced sterility affects ecological components. In *D. virilis*, pupal heat stress delays reproductive maturity, leaving many adults temporarily infertile, whereas adult heat stress causes permanent sterility^{79,179}, which can age-restructure populations. Moreover, if the sexes differ in thermal sensitivity, the operational sex ratio shifts¹⁷⁹, modifying sex-specific variance in reproductive success and the strength and direction of sexual selection.

Heat-induced sterility might also select for different mating behaviours, thus affecting sexual selection. Mating with sterile males often triggers facultative polyandry as a fertility assurance strategy¹⁸⁵, intensifying sexual conflict and changing sex-specific variance in reproductive success. Condition-dependent male signals¹⁸⁶ might be reduced by heat stress, favouring female choice for fertile males¹⁸⁷. Conversely, reduced female fecundity might drive male mate choice towards more fecund partners¹⁸⁷. These behavioural and operational-sex-ratio-mediated effects alter the strength and



sex bias of sexual selection. A meta-analysis suggests that warming generally reduces variance in male fitness while increasing variance in females¹⁷⁸, although data specific to heat-induced sterility are lacking.

Sexual selection might change how the sexes invest in reproductive success, and heat stress might reveal trade-offs in this investment. In two insect species, males evolving under regimes promoting greater sexual selection showed reduced progeny production⁴⁴ or sperm competitiveness¹⁸⁸ after heat stress. This might reflect a trade-off between investment in intensified male–male competition and germline maintenance¹⁸⁹. By contrast, multigenerational experimental sexual selection in other taxa indicates that polyandry can benefit populations: when both sexes experience warming, populations with a history of strong sexual selection have higher fecundity^{190–192}.

Overall, heat-induced declines in fertility and fecundity have the potential to reshape interactions between ecology, demography and sexual selection. However, current data are limited and taxonomically biased. Expanding research across diverse taxa and ecological contexts is critical to determine whether altered mating systems in response to heat-induced sterility buffer populations against climate change or hasten their decline.

is crucial given the effect on operational sex ratio, population growth and subsequent ecological and evolutionary consequences (Box 2; see section ‘Ecological and evolutionary effects’).

Fertilization mode

The mode of fertilization alters the environment experienced by gametes, which could influence the effect of heat stress on fertility and fecundity. External fertilizers release gametes into the environment, directly exposing them to the abiotic environment, whereas internal fertilizers avoid this exposure. Additionally, the sperm of internal fertilizers might be stored inside the female, raising the possibility that the female reproductive tract could protect sperm from heat stress, although this does not seem to be the case in some insects^{9,55}. A meta-analysis examining effects of temperature (but not necessarily heat stress) on fertility and

fecundity in aquatic external- and internal-fertilizing invertebrates and ectothermic vertebrates found weak support for the idea that fertilization mode moderates thermal fertility effects⁸⁴. Stronger effects might be found if using only studies that applied thermal stress, or studies comparing fertilization-mode effects for terrestrial ectotherms (which might experience greater thermal variation than aquatic ectotherms) but this has not yet been tested.

Life stage

TPCs (Box 1) vary across life-cycle stages⁹¹. This ontogenetic variation can generate demographic bottlenecks, in which a vulnerable stage, such as during reproduction, might constrain population growth, having downstream evolutionary consequences (Box 2). Ignoring this variation might lead to overestimates of population resilience

if only more-tolerant life stages are tested⁹². In two insect species, heat stress during the pupal and adult stages reduced subsequent offspring production more than exposure during earlier developmental stages^{29,45,77,93}. In externally fertilizing fish, spawners are more heat sensitive, with lower thermal safety margins than non-reproductives and juveniles⁹². A meta-analysis across aquatic invertebrates and ectothermic vertebrates found that the gamete stage was more thermally susceptible than developing or adult individuals⁸⁴. By contrast, the gamete stage in other taxa is more robust to thermal stress than later stages^{23,94,95}.

Exposure regime

Quantifying the consequences of heat stress on trait performance can be done in different ways that vary the magnitude, duration and frequency of increased temperature (Box 1). Laboratory experiments apply heat stress through acute heat shock (either static or ramping), prolonged warming under constant or fluctuating temperatures (with various amplitudes and temporal patterns), or discrete heatwaves lasting a few days, each potentially producing distinct reproductive outcomes. For example, across laboratory lines of a *D. melanogaster* population, some lines maintained fertility when experiencing a constant mean high temperature throughout development, but fluctuating temperatures with the same mean reduced fertility in all lines⁹⁶. The meta-analysis of aquatic invertebrates and ectothermic vertebrates found that experimental increases in temperature had more negative effects on reproductive traits than natural thermal exposure, but other experimental variables such as exposure duration, magnitude of temperature increase, and whether temperature treatment was constant or fluctuating did not influence temperature effects on fertility⁸⁴. The thermal death time approach (Box 1) has been used to examine effects on both survival and reproductive output in *Drosophila suzukii*⁸⁷. Both traits decreased exponentially with increasing temperature, but reproduction was more sensitive than survival. Using estimated microclimate temperatures from the population's sampling location, models found that damage to reproductive output accumulated faster than for survival, subsequently resulting in more days experienced by the population in which high temperature would have greater cumulative damage on fecundity relative to survival.

Multiple stressors

As well as thermal effects on reproductive output, climate change also modifies other abiotic factors such as ultraviolet radiation (which might increase mutation rates), precipitation, salinity, pH and seasonal patterns. Thus, individuals in the wild are typically exposed to multiple stressors simultaneously. Studies manipulating multiple stressors, such as diet^{97,98}, humidity⁹⁹, pollutants¹⁰⁰ and salinity²³, have shown that stressor interactions could influence fertility and fecundity in complex ways. For example, studies testing the effects of climate change on marine organisms often manipulate the impact of both rising temperature and ocean acidification caused by higher dissolved carbon dioxide levels¹⁰¹, with the combined effect of these two stressors being more negative than each independently, such as effects on fertilization success in oysters¹⁰². By contrast, in the subarctic copepod, *Calanus finmarchicus*, increased acidification partly ameliorates the harmful effects of warming on fecundity¹⁰³, whereas the interaction between temperature and salinity did not affect fertilization success in mussels beyond temperature effects alone²³. Expanding the number of studies that combine heat stress with other stressors will be important to

increase our understanding of climate change impacts on reproductive output.

Phenotypic plasticity

Phenotypic plasticity can modulate the effects of heat stress on fertility and fecundity. We focus on three types of plasticity that have been studied in this context. First, prior exposure of adults to sub-lethal temperatures can induce reversible physiological changes (hardening or acclimation) that improve tolerance to subsequent extreme temperatures¹⁰⁴. This acclimation can mitigate fertility loss in some species, such as *Drosophila*⁷⁰ (but see elsewhere⁷⁹) and sea urchins¹⁰⁵. Second, heat exposure during development often impairs adult reproduction even when adults experience benign conditions^{48,77,90,106}, although such exposure can sometimes enhance adult thermal tolerance^{107,108}. Overall, most of the evidence indicates limited acclimation capacity and few sex-specific differences¹⁰⁹. Third, parents exposed to heat can alter offspring phenotype via non-genetic mechanisms, a form of transgenerational plasticity that might be adaptive when environmental conditions persist across generations¹¹⁰ (see section 'Mechanisms of heat-induced fertility loss'). Such plasticity improves thermal resilience in offspring in some species^{111,112} but in other species the reproductive output of those offspring might be reduced, even when they develop in benign temperatures^{30,40}. Understanding the evolutionary effect of phenotypic plasticity on heat-induced loss of reproductive output requires additional research.

Ecological and evolutionary effects

Although thermal effects on fertility and fecundity are increasingly well documented, the broader ecological and evolutionary effects remain underappreciated. Here we explore how heat-induced reductions in fertility and fecundity can shape species distributions, extinction risk and sexual selection (Box 2). Range shifts caused by heat-suppressed reproductive output might influence other evolutionary and ecological responses.

Species distributions

Understanding how climate change affects where species can live is a crucially important research question. Species distribution models predict where species currently live and incorporate thermal tolerance data in different ways: mechanistic species distribution models use physiological limits directly; statistical or envelope species distribution models infer limits from matching species' occurrence data with climate variables that are assumed to correlate with physiological limits; and hybrid models combine the two approaches¹¹³. This information can then be used to map areas of predicted suitable habitat under future climate change scenarios.

If fertility and fecundity limits (TFLs) are better predictors of current species distributions than survival limits (either the lethal temperature or the temperature at which performance fails, CT_{max} ; Box 1, Fig. 3), then TFLs would provide better resolved data for forecasting climate change responses. This has been shown to be the case in two separate studies, both using multiple *Drosophila* species^{5,6}. In the first study⁵ the authors compared TFL_{80} (by applying a 4-hour static heat shock) with LT_{80} (by applying a 4-hour static heat shock) and CT_{max} (by ramping the temperature). Across 43 species, >25% of species had TFLs lower than survival limits the day after heat shock, rising to 44% 7 days later (Fig. 3a). The gap between the temperature causing sterility in 80% of males (TFL_{80}) versus the temperature causing 80% to die (LT_{80}) was species-specific. This species-specific gap between TFL and lethal limit ranged between

0 °C and 4.3 °C, changing the species ranking of thermal tolerance and producing narrower estimates of each species' thermal safety margin (Fig. 3b). Despite being measured under laboratory conditions, TFL₈₀ values improved predictions of current species distributions by >35% compared to using only CT_{max}, thus emphasizing the ecological relevance of this trait. A second study, using chronic and lower heat conditions, found that TFL₅₀ values also improve species distribution predictions⁶. Thus, regardless of how they are measured, TFLs improve our understanding of species distribution limits. These findings emphasize that many species live closer to their reproductive threshold than previously appreciated, making some species even more vulnerable to climate change than when considering survival data alone. Although TFL₅₀, CT_{max} and LT₈₀ were positively correlated across species, there was a weaker association between fertility and survival limits, suggesting distinct underlying processes⁵. However, a thermal death time (Box 1) study on *D. sukuzii* found that fertility and survival limits were strongly correlated⁸⁷. Both the TFL and the thermal death time approaches show that reproduction tends to be more thermally sensitive than survival in many species, although the mechanistic basis for this divergence between reproduction and survival, and why some species show more of a thermal gap between these two measurements, is unresolved.

Population persistence and extinction

Heat-mediated decreases in reproductive output suggest negative consequences for population persistence, assuming that evolutionary potential is limited. Whether TFLs predict population extinction risk has only been directly tested in *Drosophila*⁶. Experimental evolution of six *Drosophila* species under gradual warming (0.2 °C every 2 weeks) found that all species went extinct within 46 weeks, with tropical species going extinct sooner and thus at relatively cooler temperatures than temperate species. Male TFLs explained extinction probability better than did female TFLs, lethal limits or CT_{max} (Fig. 3c), demonstrating that heat-induced sterility can drive rapid population collapse with little scope for short-term adaptive responses. In *D. melanogaster*, extinction occurred at 29.2 °C (ref. 6), consistent with another study showing that either constant or fluctuating regimes at this temperature severely reduces both male fertility and fecundity with low heritability for resistance⁹⁶. However, other *D. melanogaster* populations persist in regions that regularly exceed 29 °C (ref. 114), suggesting local adaptation via mechanisms that mitigate heat damage to reproductive output⁷⁸ (see section 'Evolutionary potential and constraints').

Further insight comes from the sterile insect technique, in which mass release of sterile males, either through ionizing radiation or through genetic manipulation, is used to suppress pest populations¹¹⁵. Population crashes can be rapid, with guidelines suggesting the release of between 90% and 99% sterile males¹¹⁵. Although natural heat extremes are currently unlikely to cause sterility on this scale, population declines do not need to be immediate to have ecological consequences. At least two factors are expected to influence the proportion of infertile males needed to reduce the population growth rate, and by extension, the vulnerability of populations to warming-induced infertility. One is related to mating behaviour and sexual selection (Box 2). If sterile males are less competitive – for instance, through reduced mating success, production of fewer sperm or reduced ability to suppress female remating – then they are less effective at slowing population growth, thereby reducing the demographic cost of male sterility¹¹⁶. Moreover, as the sterile insect technique eliminates reproduction for most males, this will produce extreme variance in male reproductive success, and could generate selection for increased

female discrimination against sterile males¹¹⁷ (Box 2). Whether this evolutionary response could emerge under natural heat-induced male sterility is unexplored. The second factor is fertility recovery after heat stress¹¹⁶ (see the section 'Fertility recovery after heat stress'). Demography will be negatively affected when sterility is either long-lasting relative to the organism's lifespan or is irreversible.

Sympatric species and adaptive introgression

Species are shifting polewards in response to rising temperatures¹¹⁸, potentially bringing closely related species into secondary contact. If reproductive isolation barriers are incomplete, then such shifts into sympatry might result in increased gene flow and recombination arising from hybridization between taxa¹¹⁹. Hybridization could have negative, positive or a mix of fitness consequences.

Negative hybridization consequences can occur when sympatry generates reproductive interference¹²⁰, which could be exacerbated under climate change if these species differ in thermal fertility sensitivity. For example, if females of a more thermally resilient species mate with males from a less thermally resilient species, which may be sterile, this could negatively affect female reproductive success. On the island of São Tomé, two sister species, *Drosophila santomea* and *Drosophila yakuba*, exhibit different thermal fertility sensitivities and meet in a contact zone, generating some hybrids¹²¹. Because matings between *D. yakuba* females and the more thermally sensitive *D. santomea* males are more likely than the reciprocal¹²², high temperatures could exacerbate reproductive interference, resulting in increased hybridization, which is costly in this species pair because hybrid males are genetically sterile¹¹⁹. Increased hybridization could either result in reduced species barriers or select for reinforcement of stronger prezygotic barriers¹²³. Experimental evolution with these two species showed reinforcement of prezygotic gamete barriers that reduced hybridization cost¹²⁴, but temperature was not used to change these costs, leaving the role of differential thermal fertility between sympatric species in reproductive interference and its consequences an open question. In the marine tubeworm, *Galeolaria*, reproductive isolation is strong at cooler temperatures between two sympatric species but higher temperatures increase cross-fertilization success¹²⁵. Whether this hybridization is costly or beneficial is unknown.

Hybridization may be beneficial by allowing the acquisition of thermal fertility resilience alleles from a species adapted better to heat. Such adaptive introgression might provide a route for rapid evolutionary change (although this could also blur species boundaries)¹²⁶. In the copepod *Tigriopus californicus*, an evolve and re-sequence experimental hybridization approach between two populations with divergent heat tolerance found increased survival heat tolerance in hybrid lines associated with introgression from the more heat-tolerant southern population¹²⁷. A similar study has extended this to fertility tolerance genes between the more heat-tolerant *Drosophila montana* and the less tolerant *Drosophila flavomontana*. Using experimental hybridization under heat stress, heat-evolved lines had more introgression than control lines, and hybrid males had improved fertility when heat-stressed¹²⁸. Thus, adaptive genetic material mitigating heat-induced sterility can cross strong genetic barriers between species, increasing reproductive resilience to heat stress.

Invasive species

Invasive species have multiple ecological and evolutionary consequences on communities and ecosystems¹²⁹. They harm native species through ecological competition and can exhibit superior thermal

performance and phenotypic plasticity¹³⁰. However, no direct comparison of thermal sensitivity in fertility either between invasive and native species or between native and invasive populations of the same species have been made. Interestingly, the invasive round goby has adapted to local salinity by improving sperm velocity, enabling population spread¹³¹. This example highlights both the potential for local adaptation and the threat of invasive species, and focuses knowledge acquisition on understanding the extent of genetic variation within and between populations and species in how temperature influences reproductive output.

Evolutionary potential and constraints

TFLs place an upper bound on the ability of populations to reproduce under climate warming. Niche tracking to higher altitudes or latitudes might buy time, but is limited by geography. Sustained persistence requires in situ adaptive evolution of greater heat tolerance for fertility and fecundity. Below, we synthesize factors influencing the evolution of fertility tolerance, including heritability and genetic architecture, mutational input and epigenetic change, and geographic variation and local adaptation in thermal fertility resilience.

Adaptive potential

The evolutionary potential to increase thermal fertility tolerance requires heritable genetic variation in relevant physiological processes (see section on 'Mechanisms of heat-induced fertility loss'). Empirical estimates of this variation are scarce and reflect broad-sense heritability. These are point estimates of evolutionary potential that do not separate additive genetic variation from other sources of variation and are subject to several different factors that affect the heritability estimate (including methodological, life-stage and population factors) that need to be considered when predicting potential adaptive responses¹³². A study in heat-stressed male mice, *Mus musculus*, found genetic variation in fertility, with resistant males having higher performance in several traits related to offspring production relative to susceptible males, with heritability per trait ranging from 0.09 to 0.13 after heat stress¹³³. In *D. melanogaster*, developmental exposure to chronically high temperatures revealed low heritability for fertility tolerance, but broad-sense heritability rose as stress increased, revealing cryptic genetic variation⁹⁶. The release of cryptic genetic variation is a common response under genetic or environmental stress, resulting in increased trait variance caused by DNA-repair mechanisms losing fidelity and subsequently generating phenotypic plasticity and extreme phenotypes that could facilitate adaptation to novel environments¹³⁴.

Despite evidence for cryptic genetic variation in the heat tolerance of reproductive traits, multi-generational selection experiments in several *Drosophila* species failed to elicit evolutionary shifts in male fertility limits⁶ or female fecundity¹³⁵ under steadily increasing temperatures. The lack of heat tolerance response for fertility echoes patterns for non-fertility traits¹³⁶, in which upper thermal tolerances are typically more constrained than lower tolerances¹³⁷. However, *T. castaneum* males undergoing experimental evolution under constant high temperature were found to have improved fertility under subsequent heatwave conditions at even higher temperatures¹³⁸, suggesting that sustained selection can sometimes yield increased fertility tolerance.

Genetic architecture

The genetic architecture of adaptive traits can also affect evolutionary response. With sufficient genetic variation, evolution can be facilitated or constrained by selection on pleiotropically or physically linked traits.

Constraints occur when selection acts non-orthogonally to genetic variance among genetically linked traits, indicating antagonistic pleiotropy¹³⁹. For example, a negative genetic correlation between heat and cold reproductive tolerance in ostriches, *Struthio camelus*, might constrain adaptation to increasing fluctuating temperatures¹⁷. In *C. maculatus*, experimental lines evolving under warming had increased longevity but reduced reproduction, indicating a trade-off in life-history investment¹⁴⁰. However, in isogenic *D. melanogaster* lines, genetic variation associated with thermal fertility effects was uncorrelated with many other stress responses, including CTLs, suggesting that they have evolved independently^{47,96}. The typically positive relationship between body size and fecundity¹⁴¹ but the negative relationship between high developmental temperature and body size in ectotherms¹⁰⁴ might affect the ability of reproductive output to respond to climate warming if genetically correlated. However, populations can vary in the extent of the relationship between developmental temperature and body size¹⁴². Moreover, fecundity under high thermal stress can be reduced independently of body size²⁶ and experimental warming studies show conflicting results on evolution of body size, including its relationship to reproductive output^{143,144}. So whether changes in body size, which has been suggested to represent a universal response to climate warming¹⁴⁵, genetically influences the evolution of reproductive output as temperatures continue to rise requires substantial experimental dissection.

Sex-specific genetic architecture might also influence responses. In ostriches, individual variation in thermal fecundity resilience was found for both males and females. However, some females were able to increase both egg number and mass at high temperatures, without a trade-off with egg mass at other temperatures, indicating adaptive genetic variation in females that perform well at multiple temperatures¹⁷. In *C. maculatus*, selection for male sexual competitiveness resulted in increased thermal sensitivity of fertility in both sexes, suggesting that sexually selected traits carry hidden costs for fertility under heat stress and that the heat sensitivity of female reproductive output is genetically correlated with sexually selected reproductive traits in males⁴⁴ (Box 2). Conversely, *D. melanogaster* lines revealed a weak correlation between thermal sensitivity of male and female fertility⁴⁷, suggesting potential for sex-specific evolution in some systems.

Thermal induction of mutations

Although standing genetic variation is critical to rapid evolutionary responses, mutations are the ultimate source of genetic variation. Environmental stress can trigger mutagenesis via mechanisms such as increased DNA damage or decreased DNA repair – processes related to compromised fertility under heat stress (see section on 'Mechanisms of heat-induced fertility loss'). Increased mutational variance under high temperatures can enhance selection efficiency and accelerate adaptation¹⁴⁶. Heat stress can also trigger increased transposable element activity, which can generate mutations⁵⁰. Additionally, heat stress might interact with other mutation-inducing stressors, such as in *C. maculatus*, in which lines evolving under warming had enhanced germline repair after exposure to mutation-inducing radiation¹⁴⁰. This response hints that selection to warming can affect mutational robustness. However, whether elevated mutation rates in response to increasing temperature can provide evolutionary rescue of fertility in animal populations is untested, although there is evidence that increased mutation rate can result in thermal adaptation in *Escherichia coli*¹⁴⁷.

Latitudinal variation of fertility resilience

Tropical and temperate species experience distinct thermal regimes. Tropical species tend to be adapted to relatively stable and warm temperatures and live closer to their CTLs (that is, have a narrower thermal safety margin), such that increases in both the mean temperature and its variability are predicted to make survival of tropical species vulnerable to even modest warming^{2,145}. Although limited in scope, a study demonstrated that across six *Drosophila* species, TFL₅₀ occurred at lower temperatures in tropical species, indicating lower fertility resilience and faster extinction compared to temperate species⁶. Generally, tropical species also exhibit narrower thermal fertility safety margins⁶ that are lower than CT_{max}-based thermal safety margins⁶. However, whether tropical species are more vulnerable to temperature increases induced by climate change is debated¹⁴⁸. An analysis of 38 ectotherm species suggested higher extinction risk in temperate species owing to greater thermal variance and longer extreme events (that is, longer periods of heat stress in the summer) compared to tropical species¹⁴⁹. However, this study could not disentangle temperature effects on fertility from survival. By contrast, a study of 17 species of hemipterous insects that could disentangle these effects found that low-latitude species suffered reduced per capita birth rates, whereas high-latitude species suffered from increased mortality¹⁵⁰. Thus, which traits determine the vulnerability of low-latitude species versus high-latitude species, and to what extent, remains an outstanding question.

Local adaptation

Thermal tolerance studies often use one population to represent a species, which might suffice if thermal limits show limited evolutionary potential. However, trait values can vary across a species' distribution range in response to variation in local ecology, enabling local adaptation. Locally adapted populations, by definition, harbour locally beneficial alleles. Local adaptation for fertility tolerance occurs in *D. subobscura*^{89,106} and *D. melanogaster*⁷⁸, in which low-latitude populations exhibit higher fertility resilience in response to heat stress than higher-latitude populations. In the ostrich, females with higher egg-laying rates under hotter temperatures regulated their head temperature better¹⁵¹. This response was both heritable and exhibited a signature of local adaptation given that females from more variable and unpredictable areas had greater thermoregulatory head capacity. Determining whether there is local adaptation of fertility and fecundity in response to heat stress is important, because these alleles could be transferred to other populations via gene flow. Moreover, incorporating local adaptation into predictions of how species will respond to climate change could improve the quality of these forecasts.

Summary and future perspectives

Reproduction underpins population persistence, yet fertility and fecundity remain under-represented in forecasts of species' responses to climate warming^{3,83}. Reproductive failure can occur at temperatures well below lethal temperatures, as has been demonstrated across diverse animal taxa. Research has focused on model organisms or livestock, but across multiple species heat stress consistently reduces gamete quantity and quality and fertilization success, often via DNA damage and oxidative stress. Genes for HSPs and other stress-related genes might have central roles in mediating fertility responses. Male fertility is thought to be more sensitive to heat stress than female fertility but this might be species-specific. External fertilizers might be more vulnerable than internal fertilizers, although the data are sparse. Life-history stages vary in both heat sensitivity and potential for recovery, with

important implications for population dynamics. Within and between generations, plastic responses of reproductive output to rising temperatures may be limited, resulting in more rapid exposure to selection with potential negative demographic consequences.

The ecological and evolutionary effects of heat-induced sterility are little studied and taxonomically limited. Research in *Drosophila* found that TFLs and fertility-based thermal safety margins can be lower than survival-based estimates, resulting in increased population vulnerability, including extinction risk. Indeed, fertility-based measures predicted both *Drosophila* extinction risk and current species distributions better than death temperatures. However, similar studies in other taxa are lacking, the link between fertility loss and range limits remains correlative in *Drosophila*, and the effect of other factors such as behaviour, acclimation and the use of microclimates¹¹³ in buffering fertility impacts is unknown. Although heritability of fertility resilience is low, the role of genetic architecture, mutations or hybridization in shaping its adaptive potential are underexplored. Adaptive potential is also contingent on the demographic and ecological context, with small populations, species with narrow ranges, and geography (for example, tropical versus temperate habitats) all affecting vulnerability. Such taxa also face other anthropogenic stressors, reinforcing the need for multi-stressor experiments. Invasive species might prove more resilient but this assertion requires further study. The effects of heat-induced sterility on sexual selection remain strikingly sparsely studied.

Thus, as a burgeoning field, important knowledge gaps remain. Below we highlight six research priorities aimed at providing better predictions of future fertility and fecundity costs and responses to climate change.

- (1) The taxonomic breadth of research studies should be expanded. Many of the mechanistic and ecological insights into reproductive output loss in response to warming come from a narrow set of model and agricultural organisms. Broader phylogenetic sampling is essential to identify generality and outliers, and to uncover the factors causing some taxa to be more vulnerable. Surveying multiple populations is critical to quantify the genetic variation that could respond to thermal selection for fertility and fecundity resilience.
- (2) Functional and ecological endpoints need to be integrated. Understanding what magnitude and duration of thermal exposure is most relevant for each taxon, and integrating these data into models of population persistence, such as species ranges and extinction risk, is needed. Modelling efforts are essential to simulate diverse scenarios predicting male and female responses across life-history stages, and to identify the conditions that promote population persistence versus those leading to extinction. Experimental studies could validate these models, deepening understanding of species-specific vulnerabilities and informing conservation strategies.
- (3) Multi-stressor scenarios should be performed. For example, interactions between heat and other stressors could result in a situation in which lower temperatures with milder fertility and fecundity effects (for example, TFL₃₀) generate a larger effective reproductive loss (such as TFL₅₀) at the same temperature. Multi-stressor experiments are necessary to understand the consequences of these interactions to better predict future responses.
- (4) Broader community and ecosystem consequences need evaluation. Range shifts are a common climate change response¹¹⁸, and male TFLs might partly underlie these observed patterns. Whether variability in thermal fertility tolerance (including invasive species

Glossary

Acclimation

Physiological adjustment to an increase in temperature, in particular when longer-term exposure to a mildly stressful temperature increases tolerance of a subsequent higher temperature.

Adaptive introgression

Where genes from outcrossing to other populations or species improve fitness under novel conditions.

Antagonistic pleiotropy

When a gene/allele has beneficial effects in one/some traits but negative effects for other traits.

Critical thermal limit

The upper (CT_{max}) or lower (CT_{min}) temperature at which critical biological function (often measured as motor control or coordinated movement) is lost, or death occurs.

Critical thermal maximum

The highest temperature at which a physiological function is lost (often measured as motor control or coordinated movement), or death occurs.

Cryptic genetic variation

Genetic variation that is normally masked, having little to no effect on the phenotype, but that is exposed under stressful conditions, thereby putatively available to fuel adaptation.

Evolutionary rescue

Selection for evolutionary change enabling individuals to survive under stressful or novel conditions, sufficiently rapidly to save a population from extinction.

Fecundity

Number of eggs or viable offspring produced by an individual. (We note that in the medical literature the definitions of fertility and fecundity can be switched in meaning compared to those used in most non-human contexts. When fecundity is measured as the number of eggs produced, then the effect of heat treatment on offspring

survival is not conflated. However, in many taxa, only offspring number is reported. In cases in which a paper reports offspring number, we included it in our review only when the parents, and not offspring, were placed under heat stress. This limits the potential confounding effect of heat stress on survival of offspring that is separate from the effect on the parents).

Fertility

Ability to produce viable offspring. (We note that in the medical literature the definitions of fertility and fecundity can be switched in meaning compared to those used in most non-human contexts).

Hardening

A period of elevated temperature that improves performance under subsequent higher (extreme) temperatures.

Heat-shock proteins

A family of proteins found in virtually all living organisms, produced by cells in response to exposure to high or low temperatures, as part of the heat-shock response; many heat-shock proteins function as chaperone proteins, stabilizing the structure of other proteins that are sensitive to heat stress.

Heatwave (or heat wave)

A meteorological term for a sustained period of abnormally warm weather that lasts for multiple days, measured relative to the normal climate for a given location and time of year. (We note that there is no universally agreed definition for the duration and severity of a heatwave, but as a rough estimate, most definitions require three to five consecutive days of temperatures 5 °C or more above the seasonal average. Many studies attempt to mimic heatwaves to make their temperature manipulations more ecologically realistic).

Infertile

Inability to produce viable offspring; can specifically mean inability to produce gametes.

Lethal temperature

The temperature at which there is, for example, 80% (LT_{80}) or 50% (LT_{50}) mortality in a population or set of experimental organisms, often measured similarly to a lethal dose response.

Local adaptation

Divergent selection leading to local populations having higher relative fitness under local environmental conditions.

Microclimate

A local set of atmospheric conditions that differ from those in the surrounding areas, of interest to thermal biologists because of the potential to provide protection from temperature extremes; for example, animals may be able to shelter from high temperatures in burrows or shaded areas that remain cool.

Operational sex ratio

The ratio of males to females within the mating pool (those animals fertile and ready to mate).

Phenotypic plasticity

The ability of a genome to produce multiple phenotypes depending on the environment.

Polyspermy

Entrance of more than one sperm into the egg (typically lethal).

Reinforcement

Natural selection for reduced reproductive costs for hybrid reproduction increases reproductive isolation.

Reproductive interference

When reproductive isolation is incomplete and heterospecific individuals still engage in mating/reproductive activities, resulting in a fitness reduction for at least one of the interacting partners.

Reproductive output

A holistic term incorporating both fertility and fecundity.

Secondary contact

Populations diverge during a period of geographic isolation but then come back into contact, increasing the opportunity for gene flow.

Sterile

Inability to produce viable offspring; can specifically mean inability to produce gametes.

Thermal death time

The integration of stress intensity (temperature) and exposure time. (We note that through cumulative effects, organisms may suffer more from a mildly stressful high temperature experienced for a long time than from an extreme temperature experienced for a short time. Because the relationship is exponential, a small increase in temperature may lead to a large decrease in tolerance time).

Thermal fertility limit

The temperature at which individuals become (at least temporarily) sterile (scored as a binary 0/1 outcome); as for lethal temperature, it is measured as TFL_{80} or TFL_{50} , the temperature at which either 80% or 50% of individuals in a population or set of experimental organisms are infertile.

Thermal performance curve

Graphical representation of the relationship between temperature and the value of a biological trait (for example, how activity, climbing ability or offspring production change over a range of temperatures).

Thermal safety margin

The difference between the thermal limit and the maximum ambient temperature experienced in the environment.

Thermal sensitivity of fertility

The relationship between temperature and reproductive output; the number of offspring produced or proxies thereof (such as sperm velocity, follicle number) are a measure of fertility rather than a binary score (0/1) of fertility.

and hybridization potential) has community effects is unknown. Studies assessing species-specific CT_{max} effects on community interactions have found that range shifts and variability in critical thermal tolerance across trophic levels alter species composition and community interactions, such as changing food webs¹⁵² and modifying density-dependent competition processes¹⁵³. Similar studies could illuminate whether species-specific TFLs also have broad community and ecosystem consequences.

(5) Interactions with other evolutionary processes and the effect of genetic architecture need defining. Both natural selection (for example, differences across life-history stages in how heat stress influences trait expression and fitness consequences) and sexual selection, along with genetic correlations between traits within and between the sexes, influence evolutionary response. More sophisticated experiments across taxa quantifying how these factors affect the evolution of reproductive output tolerance under a warming world are necessary. A variety of experimental approaches may be envisioned but could, for example, expose multiple populations to multiple stressors, and measure survival and reproductive output traits under a quantitative genetic framework that estimates genetic correlations and evolvability. Such data would enable estimation of population-specific vulnerability and adaptive constraints and, if combined with genomic sequencing, could identify genes contributing to population-level variation in stress response. Any evidence of local adaptation could be included in analyses investigating how species may respond to future anthropogenic scenarios, including stress factors other than temperature.

(6) Mechanistic insights must be translated into applied solutions. Shared pathways of thermal reproductive damage across species offer targets for conservation, agriculture and aquaculture interventions. Agricultural practices have incorporated cooling techniques and selective breeding to prevent fertility loss¹⁵⁴, but this has not translated to considerations for wild organisms. However, despite shared pathways, related taxa clearly vary in thermal fertility resilience, which might reflect local adaptations. Genomic screening for such resilience along with selective breeding or adaptive introgression could provide, all else being equal, management solutions for vulnerable or economically important taxa.

In summary, safeguarding biodiversity, and therefore its ecosystem services, in a warming world requires not only keeping organisms alive but also keeping them reproducing. By examining the consequences of warming on reproductive output we can deliver more realistic projections of organismal responses to climate change and improve the design of effective conservation and management interventions. Expanded research in this area will provide fundamental insights into the future of biodiversity under rapid environmental change.

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